Oral Reticular Lichen Planus: Diagnosis & Management.

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Abstract

Lichen planus is a chronic systemic disease that commonly involves mucosa and skin. Thus the objective of paper is to report a case of lichen planus in male patient and discuss the main aspects of this disease in relation to etiopathogenesis and treatment.

Keyword -Oral lichen planus (OLP),Systemic corticosteroids

Introduction

Lichen in greek means tree moss and planus means flat. Lichen planus was first reported by Erasmus Wilson in 1869.[1]Oral lichen planus (OLP) is a chronic inflammatory mucocutaneous disorder, affects skin and mucosa and its stratified squamous epithelia, with a prevalence of 0.02% - 1.2%morecommonlyseen in females.[2] This disease is most often reported in middle-aged patients 30-60 years of age with a female to male ratio 1.4:1. Rarely, OLP is seen in children.[3]It is believed that the disease is caused by an abnormal cellmediatedimmune response of both T4 and T8 lymphocytes in the basal epithelial cells. Autocytotoxic CD^{8+} T-cells activate apoptosis of oral epithelial cells. The CD^{8+} cytotoxic cells trigger the keratinocyte apoptosis through activation of the cells by an antigen associated with major histocompatibility Class I on basal keratinocytes.The chronic course of OLP may result from the activation of the inflammatory mediator nuclear factor kappa B, and the transforming growth factor control pathway may cause keratinocyte hyper proliferation that leads to the white lesion.[4]

OLP(also termed oral mucosal lichen planus), is a form of mucosal lichen planus, where lichen planus involves the oral mucosa, the lining of the mouth. Six clinical forms of oral lichen planus are recognized which are a) Reticular b) Plaque c) Papular d) Bullous e) Erosive / Ulcerative.[5-6]

Here we report a case on reticularlichen planus, which involveboth sides of buccal mucosa in its clinical presentation and its effective treatment using synthetic corticosteroids and topical corticosteroids.

Case report

A 52 year old medically fit male patient reported to our departmentwith a chief complaint of burning sensation while havingfood, since duration of approximately one year. The patient gave history of episodes of intermittent burning sensation onboth sides of cheek, which mainly aggravated on having food, which relieved on its own. Patient's medical and family histories were non-contributory. Patient had smoking habit since last 25 years. The patient had visited many dentists before for the same chief complaint and dentist prescribed local anesthetic ointment for relieving burning sensation.

On clinical examination, interlacing white keratotic striaewith erythematous borders giving a web-like appearance were seen on right buccal mucosa and left buccal mucosaand vestibular region [Figure 2 to 3]. On palpation the lesionwas non-scrapable. Other clinical findings included missing teeth in relation to maxillary right first pre molar to left maxillary first molar. Based on the clinical presentation a provisional diagnosis of reticular lichen planus of right and left buccal mucosa was given. The patient was advised for incisional biopsy. But as the patient was not willing for biopsythen, he was advised a regimen of anti-oxidants (Cap.A Charge®), Systemic corticosteroids (Tab.Wysolone 10mg® contains prednisolone 10mg) once a dayand Topical corticosteroids (Kenacort 0.1% ® contains Triamcinolone 0.1%). Patient was asked to report for periodic recall after everytwo weeks. The lesion regressed almost within 2 weeks and chief complaint of burning sensation resolved completely [Figure 4]. Then, we shifted the dose of Tab Wysolone 10mg to 5mg once a day. The prognosis was excellent and after a period ofjust one and half months the lesion had regressed completely and the patient's oral mucosa was back to normal[Figure 5].Patient was further recalled after one, three and six months; noincidence of recurrence was seen.

Discussion

Lichen planus is a mucocutaneous disease characterized by a cellular inflammatory infiltrate enriched by CD4+cells. These cells with the presence of acidophilic bodies causes the apoptosis and vacuolation of epithelial cells, causing degeneration of the basal epithelial layer of affected mucosa.[7]Globally, OLP affects about 1-2% of population and prevalence in India ranges from 0.1-1.5%. OLP can develop on any mucosal surface including larynx and oesophagus but lesions are most commonlynoted on the posterior buccal mucosa which is similar to present case. The specific

etiology of oral lichen planus is unknown. Itis believed to result from an abnormal cell mediated immune response with infiltrating cell population composed of both T4 and T8 lymphocyte in the basal epithelial cells. They are recognized as foreign bodies because of changes in the antigenicity of their cells surface.[8]

The classic skin lesions of the cutaneous form of lichen planuscan be described as purplish, polygonal, planar, pruritic papules and plaques.These skin lesions commonly involvethe flexor surfaces of the legs and arms, especially the wrists.

The nail beds may also be affected, with resultant ridging, thinning and subungual hyperkeratosis. Scalp involvement, if untreated,can lead to scarring and permanent hair loss. Since 30% to 50% of patients with oral lesions also have cutaneous lesions, the presence of these characteristic cutaneous lesions can aid in the diagnosis of OLP.[8]

Although lichen planus can present with a variety of lesions, the most common presentation is as a well-defined area of purple-colored, itchy, flat-topped papules with interspersed lacy white lines (Wickham's striae). This description is known as the characteristic "6 Ps" of lichen planus: planar (flat-topped), purple, polygonal, pruritic, papules, and plaques. This rash, after regressing, is likely to leave an area of hyperpigmentation that slowly fades.[10]Reticular, the most common presentation of oral lichen planus,which is seen in present case as symptomaticand ischaracterized by the net-like or spider web like appearance of lacy white lines, oral variants of Wickham's striae. Erosive/ulcerative, the second most common type of oral lichen planus, is identified by oral ulcers presenting with persistent, irregular areas of redness, ulcerations and erosions covered with a unique yellow slough. This can occur in single or multiple areas of the mouth. In 25% of people with erosive oral lichen planus, the gingivais involved, described commonly as desquamative gingivitis (a condition not unique to lichen planus). Following may be the initial or only signs of the condition.

• Papular, with white papules.

• Plaque-like appearing as a white patch which may resembleleukoplakia.

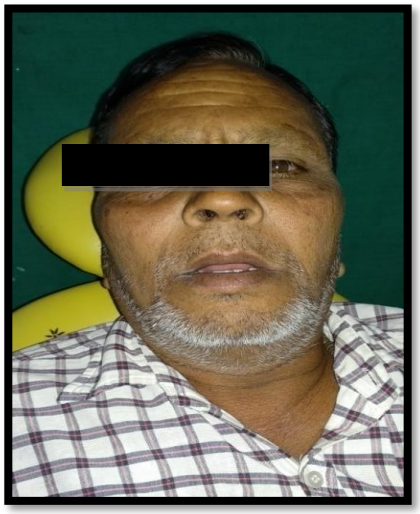
• Atrophic, appearing areas. Atrophic oral lichen planus may also manifest as desquamative gingivitis.

• Bullous, appearing as fluid filled vesicles which projectfrom the surface.The cause of OLP is unknown. It is said some certainfactorsmention below may trigger an inflammatorydisorder.[12-13-14] Hepatitis C infection and other types of liverdisease, Allergy-causing agents (allergens), such as foods,

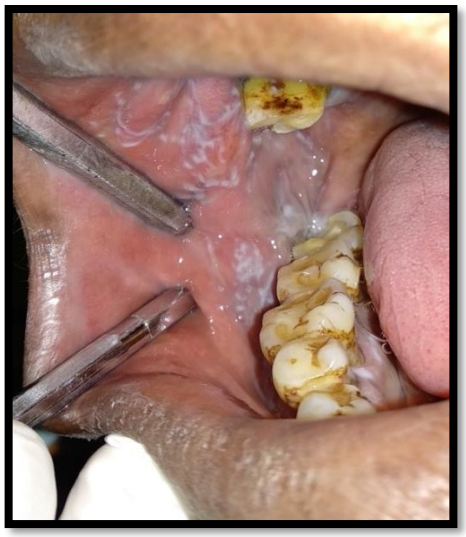
dental materials or other substances, genetic background, Immunodeficiency disorder, Some bacterial and viral diseases, Certain medications for heart disease, High blood pressure or arthritis, Certain drugs like ibuprofen and naproxen, Stress, Graft versus host disease.Many controversies exist about the pathogenesis of OLP. A large body of evidence supports a role of immune dysregulationin the pathogenesis. The various mechanisms hypothesized to be involved in the immunopathogenesis are:[15] Antigen-specific mechanism, Non-specific mechanisms, Autoimmune response, Humoral immunity. The diagnosis of OLP is based on a combination of characteristicclinical findings, history and histopathological examination. The hyperkeratotic (white) variant of OLP is often symptomless. The atrophic or the erythematous (red) variant and the erosive or the ulcerative (yellow) variants of OLP generally have persistent symptoms. Treatment of symptomatic OLP is challenging. Several drugs have been used with varying efficacy. Specific treatment includes corticosteroids (topical, intralesional or systemic), retinoids, cyclosporine, griseofulvin, hydroxychloroquine and dapsone.[16] Non-pharmacological modalities include PUVA therapy, photodynamic therapy andLASER therapy.The most commonly accepted treatment for lesions of OLP involves topical or systemic corticosteroids to change the patient's immune response. Topical corticosteroids are the commonly used in treating mild to moderately symptomatic lesions. Options (presented in terms of decreasing potency) include 0.05% clobetasol propionate gel, 0.1% or 0.05% betamethasonevalerate gel, 0.05% fluocinonide gel, 0.05% clobetasol butyrate ointment or cream, and 0.1% triamcinolone acetonideointment.[17]The goal of treatment for symptomatic patients is palliation. The following flow-chart illustrates an easyprotocol which will help in effective treatment. The most important issue concerning OLP is its potential for malignant transformation into OSCC. Although the WHO has categorized OLP as a potentially malignant epithelial lesion, the risk of malignant transformation of OLP remains a subject of debate in the literature. Some researchers accept the possible malignant potential of OLP, while others oppose this belief. Based on recent reports, the overall malignant transformationrate of OLP is estimated to be extremely low.[19]A variety of systemic conditions may be associated withlesions of OLP and at times oral manifestations are the only signs and symptoms present in practice for the underlying conditionas seen in our patient. And

since there is tendency for malignant transformation, it is elementary and fundamental as oral clinicians to make an accurate and timely diagnosis andrender the appropriate treatment plan. As they rightly say "timely effort will prevent more work"

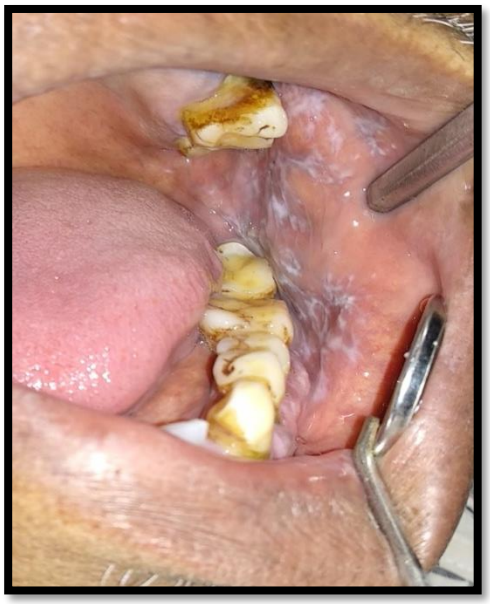
- **Pretreatment**



[Figure 1].

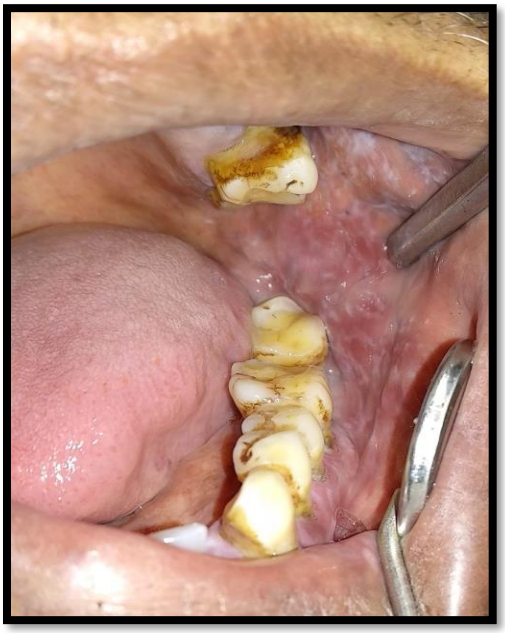


[Figure 2].



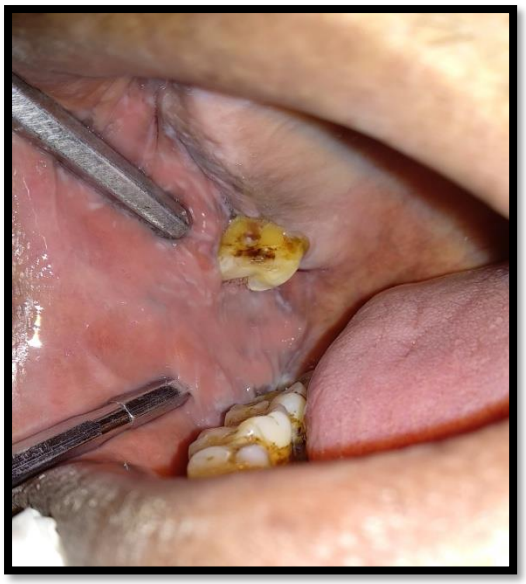
[Figure 3],

- **Post-treatment after 2 week.**



[Figure 4].

- **Post-treatment after 1 Month.**



[Figure 5].

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