

Reticular oral lichen planus: The intra-oral web – A case report

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SUMMARY

Oral lichen planus (OLP) is a chronic mucosal condition commonly encountered in clinical dental practice. Lichen planus is believed to represent an abnormal immune response in which epithelial cells are recognized as foreign, secondary to changes in the antigenicity of the cell surface. OLP is a chronic inflammatory disease characterized by relapses and remissions. Treatment is aimed primarily at reducing the length and severity of symptomatic outbreaks. Topical steroids are the first-choice agent for the treatment of symptomatic, active OLP.

Key words: Lichen Planus, Reticular, Intra-oral, Corticosteroids.

Introduction

Lichen planus in greek means tree moss and planus means flat. Lichen planus was first described by Erasmus Wilson in 1869. [1]Oral lichen planus (OLP) is a chronic inflammatory mucocutaneous disorder, affecting stratified squamous epithelia, with a prevalence of 0.02% - 1.2% among the various with a more female preponderance.[2] This disease has most often reported in middle-aged patients 30-60 years of age with a females to males ratio (1.4:1). Rarely, OLP is seen in children. [3]It is believed that the disease is caused by an abnormal cell-mediated immune response of both T4 and T8 lymphocytes in the basal epithelial cells. Autocytotoxic CD8 + T-cells activate apoptosis of oral epithelial cells. The CD8 + 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.[5][6]Here we report a case on reticular lichen planus, its clinical presentation and its effective treatment using synthetic corticosteroids.

Case report

A 37 year old medically fit female patient came to our department with a chief complaint of burning sensation whilst having food since a duration of approximately one year. The patient gave history of episodes of intermittent burning sensations on both sides of cheek which mainly aggravated on having food, while relieved on its own. The Visual Analog Scale(VAS) score obtained was six. Patient's medical and family history were non-contributory. The patient had visited dentists before for various treatments.

On clinical examination, an interlacing white keratotic striae with erythematous borders giving a web-like appearance were seen on right buccal mucosa and left buccal mucosa and vestibular region [Figure 1- A and B]. On palpation the lesion was non-scrapable. Other clinical findings included prosthetic crown in relation to maxillary right first molar, restorations in relation to maxillary right second molar, maxillary left second premolar, all mandibular left molars and mandibular right second molar, decay in relation to maxillary right and left third molars and missing teeth in relation to maxillary left first molar and mandibular right first molar. Based on the clinical presentation a provisional diagnosis of reticular lichen planus

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of right and left buccal mucosa was given. The patient was advised for a biopsy. But as the patient was not willing for biopsy then, she was advised a regimen of anti-oxidant (Cap.Oxirtard®) and topical cortico-steroids(Tess ointment®) for one month. Patient was asked to report for periodic recalls every two weeks. The prognosis was excellent and after a period of just two months the lesion had regressed completely and the patient's oral mucosa was back to normal [Figure 1 – C and D]. Patient was further recalled after one, three and six months; no incidence of recurrence was seen.

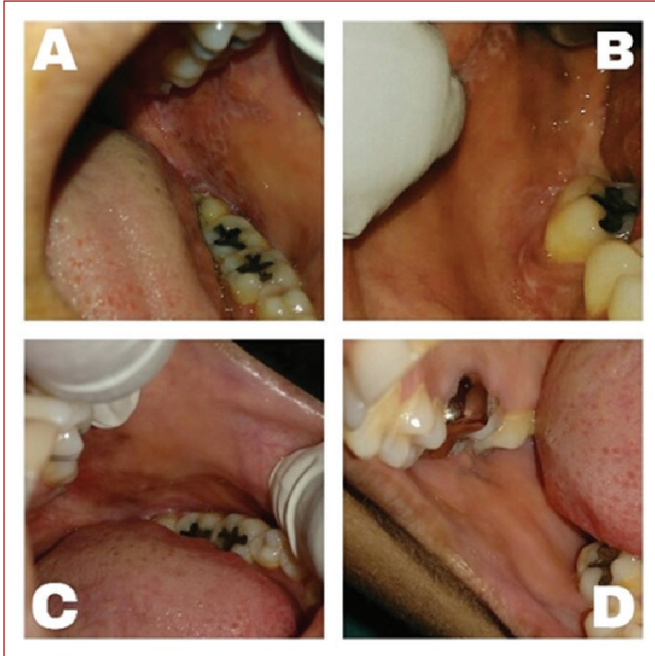


Figure 1: A& B - reticular lichen planus of left buccal mucosa and right buccal mucosa and gingiva. C&d – regression of the lesion on left and right buccal mucosa after a period of two months.

Discussion

Lichen planus is a mucocutaneous disease characterized by a cellular inflammatory infiltrate enriched in CD4+ cells, by the presence of acidophilic bodies that may represent apoptotic epithelial cells, and by vacuolating degeneration of the basal epithelial layer.[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 have predilection for the posterior buccal mucosa. The specific etiology of oral lichen planus is unknown. It is 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 because of changes in the antigenicity of their cells surface.[8]

The classic skin lesions of the cutaneous form of lichen planus can be described as purplish, polygonal, planar, pruritic papules and plaques.[9] These skin lesions commonly involve the 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-coloured, 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]

Six clinical forms of oral lichen planus are recognized:[11]

- Reticular, the most common presentation of oral lichen planus, is characterised by the net-like or spider web-like appearance of lacy white lines, oral variants of Wickham's striae. This is usually asymptomatic.
- Erosive/ulcerative, the second most common form of oral lichen planus, is characterised by oral ulcers presenting with persistent, irregular areas of redness, ulcerations and erosions covered with a yellow slough. This can occur in one or more areas of the mouth. In 25% of people with erosive oral lichen planus, the gums are involved, described as desquamative gingivitis (a condition not unique to lichen planus). This may be the initial or only sign of the condition.
- Papular, with white papules.
- Plaque-like appearing as a white patch which may resemble leukoplakia.
- Atrophic, appearing as areas. Atrophic oral lichen planus may also manifest as desquamative gingivitis.
- Bullous, appearing as fluid-filled vesicles which project from the surface.

The cause of OLP is unknown. It is said some certain factors mention below may trigger an inflammatory disorder.[12] [13][14]

- Hepatitis C infection and other types of liver disease.
- 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 dysregulation in 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 characteristic clinical findings, history and histopathological examination. The hyperkeratotic (white) variant of OLP is often symp-

tomless. 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 and LASER therapy.

The most widely accepted treatment for lesions of OLP involves topical or systemic corticosteroids to modulate the patient's immune response. Topical corticosteroids are the mainstay 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% betamethasone valerate gel, 0.05% fluocinonide gel, 0.05% clobetasol butyrate ointment or cream, and 0.1% triamcinolone acetonide ointment. [17]The goal of treatment for symptomatic patients is palliation. The following flow-chart illustrates a simple systematic protocol which will aid in effective treatment [Figure 2]. [18]

One of the most important issues concerning OLP is its potential for malignant transformation into OSCC. Although the WHO has categorized OLP as a precancerous condition, the risk of malignant transformation of OLP remains a subject of debate in the literature. Some authors accept the possible malignant potential of OLP, while others oppose this suggestion. Based on recent reports, the overall malignant transformation rate of OLP is estimated to be very low. [19]

Conclusion

A variety of systemic conditions may be associated with lesions of OLP and at times oral manifestations are the only signs and symptoms present in lieu for the underlying condition as 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 and render the appropriate treatment plan, because we all know that – “A stitch in time saves nine”.

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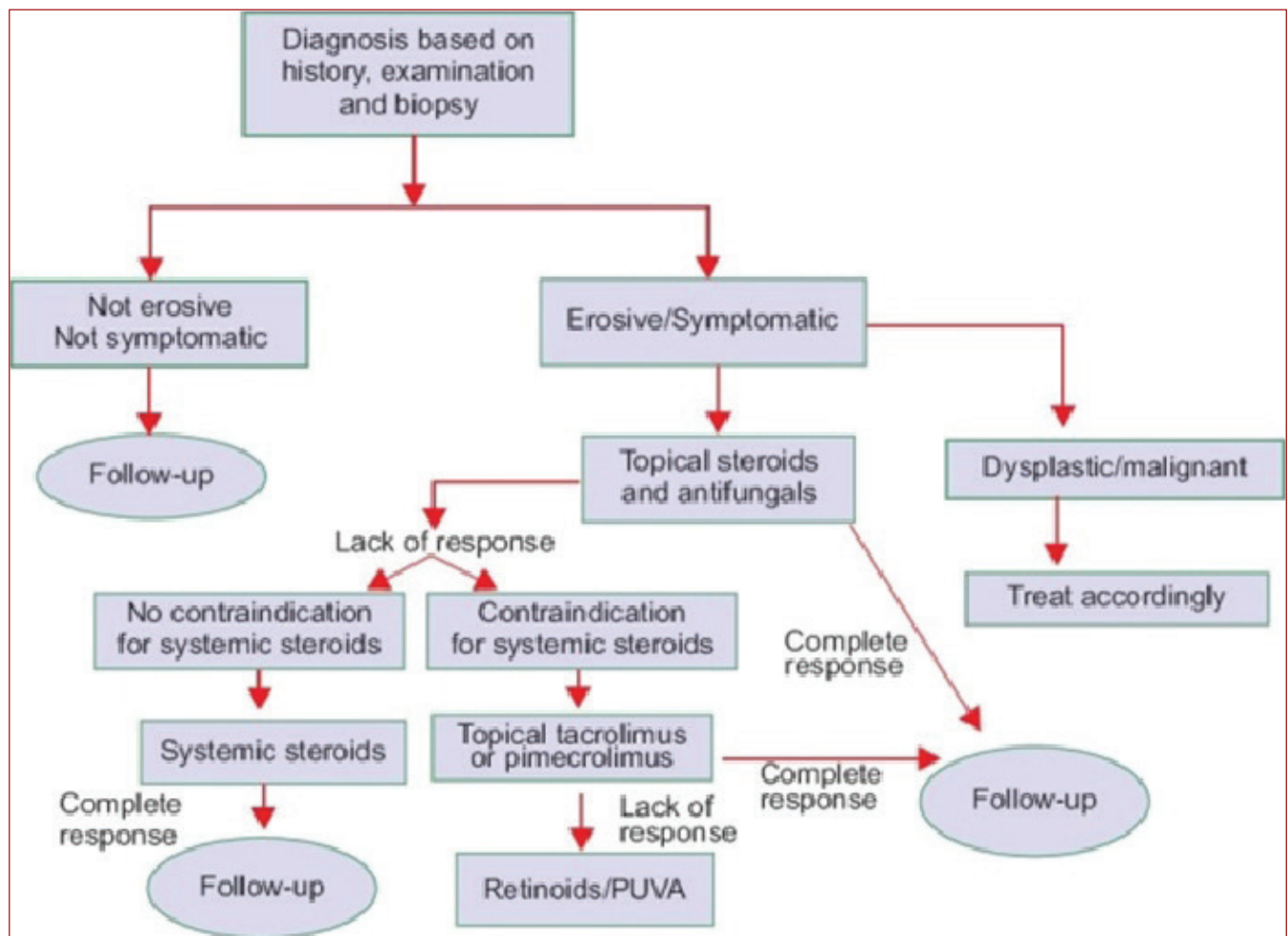


Figure 2: - A simple systematic flow-chart protocol for effective treatment planning.

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