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Gingival Lichen Planus: A Case Report

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Abstract:
Lichen planus is an autoimmune mucocutaneous disorder. Orally the most commonly involved sites are buccal mucosa, tongue and gingiva. About 10% of the patients with oral lichen planus have lesions confined to the gingiva .A 48 years old post menopausal female patient complained of redness and burning sensation in the gums since 1 year. Intraoral examination revealed bilateral erythematous areas showing diffused generalised gingival involvement. Plaque like reticular lesions were present in the area of upper and lower molars. However, no cutaneous lesions were seen. The case was diagnosed as Gingival Lichen Planus based on the clinical and histopathological examination. The patient was treated with topical corticosteroid that resolved the burning sensation though the lesion persisted.In spite of correct diagnosis and treatment plan, management of these cases remains dilemma for the clinician.This paper aims to establish the importance of diagnosing oral manifestations of systemic conditions and the holistic management of these conditions.

Keywords: *Lichen planus, autoimmune, mucocutaneous.*

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I. Introduction

Gingival diseases are classified as plaque induced and non plaque induced gingival conditions.

Several mucocutaneous disorders such as lichen planus, pemphigoid, pemphigus vulgaris (PV), erythema multiforme (EM), lupus erythematosus (LE), drug-induced lesions, and others, are listed in the subgroup of non plaque induced gingival conditions together with allergic reactions to dental materials, foods, and other substances for topical application. Besides their heterogeneous nature, all of these disorders share two features: an immunomediated pathogenesis and common clinical appearance of ‘‘desquamative gingivitis’’ (DG).

Lichen planus (LP) is a common chronic mucocutaneous disorder, which involves skin and mucosa. It was first described clinically by *Wilson* in 1869 and histologically by *Dubdreuilh* in 1906.[1]Lichen planus derives its name from the term lichens, which means lace-like pattern produced by symbiotic algae and fungi colonies found on surface of rocks.

Oral lichen planus (OLP) is a chronic, inflammatory mucocutaneous disease of unknown etiology, with prevalence rate of 0.2 - 4% in the population.[2] The usual age of presentation is between 30-60 years, and it is more frequently seen in women, with male to female ratio of 2:3.[3,4] Intraorally, the buccal mucosa is the most commonly affected site (64.3%), however, the gingiva may be involved with a similar frequency (59.8%). In approximately 10% of the patients the oral lesions are confined to the gingiva.

An important complication of OLP is the development of oral squamous cell carcinoma, which led world health organization (WHO) to classify it as potentially malignant disorder.[5] Therefore there is a need for a mandatory long term follow-up regimen for all cases of OLP

II. Aetiology And Pathogenesis

OLP is a T cell-mediated autoimmune disease but its cause is unknown in most cases.[6]

The increased production of TH1 cytokines is an important determinant in LP. It is genetically induced, and genetic polymorphism of cytokines seems to govern whether lesions develop in the mouth alone (interferon-gamma (IFN-γ) associated) or in the mouth and skin (tumour necrosis factor-alpha (TNF-α) associated).[7] Migration of activated T cells towards the oral epithelium occurs due to expression of intercellular adhesion molecules (ICAM-1 and VCAM), upregulation of epithelial basement membrane extracellular matrix proteins, including collagen types IV and VII, laminin and integrins, and possibly by CXCR3 and CCR5 signalling pathways. Cytokines secreted by keratinocytes such as TNF-α and interleukins (IL)-1,IL-8, IL-10, and IL-12 are also chemotactic for lymphocytes.

The T cells then bind to keratinocytes and IFN-γ, that subsequently causes upregulation of matrix metalloproteinase 1 (MMP1) and MMP3. This leads to programmed cell death (apoptosis) and destroys the epithelial basal cells.

The chronic course of OLP may result from the activation of the inflammatory mediator nuclear factor kappa B (NFk-B), and the inhibition of the transforming growth factor control pathway (TGF-beta/smad) that causes keratinocytes hyperproliferation leads to the development of white lesions.[8]

III. Case Report

Recently we encountered a case of lichen planus. A48 years old post menopausal female patient complained of redness and burning sensation in the gums since 1 year. She gave the history of periodic exacerbation of the burning sensation with no known aggravating or relieving factors.

Patient was unable to perform oral hygiene due to the burning sensation since 6 months.

Patient gave history of visit to gastroenterologist and oncologist 6 months ago. Patient was prescribed multi vitamin tablets. Tablets were taken regularly for 2 months but there was no relief so she discontinued the medicine.

Patient was also taking homeopathic medicine for 4 months.

Extraoral examination:

No abnormalities were detected on extra oral examination. (Figure 1)

Figure 1: No cutaneous lesions on extensor and palmar surfaces.

Intraoral examination:

Labial and buccal mucosa, tongue and floor of the mouth were clinically normal.

Intraoral examination revealed generalized diffused erythematous gingival involvement. (Figure 2) Plaque like lesions were present in the region of 45, 46 and 35, 36. (Figure 3) Lesions were bilateral in distribution with more or less symmetric pattern consisting of lacelike network and white slightly keratotic lines (Wickham striae)..

Based on clinical appearance, a provisional diagnosis of atrophic lichen planus of gingiva was established.

Figure 2: Erythematous areas showing generalized and diffused gingival involvement

Figure 3

Investigations:

Hameatologic investigation and biopsy was advised.

An incisional biopsy was taken from mandibular molar region of the attached gingiva irt 45, 46 after thorough oral prophylaxis. (Figure 4)

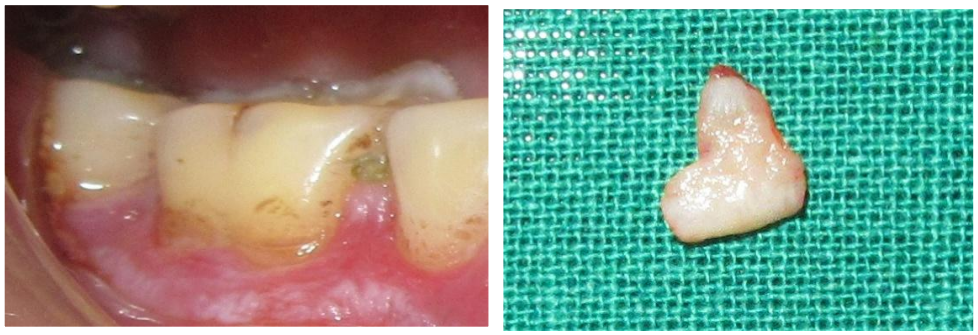

Figure 4: Site for biopsy and Biopsy specimen

Histopathology: (Figure 5)

- .Section shows covering of parakeratinized and orthokeratinized stratified squamous epithelium with atrophic changes at places.
- In some areas the rete ridges exhibit saw tooth appearance with liquefaction degeneration of basal cells.
- The sub epithelial connective tissue is loose with dense chronic inflammatory cell infiltration and many blood vessels.

Deeper region is relatively free of inflammation

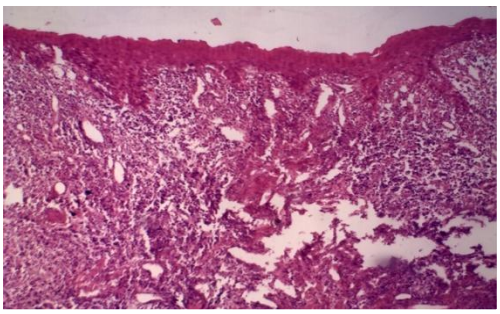

Figure 5: Histopathology

Diagnosis:

Based on clinical and histopathological features the case was diagnosed as oral lichen planus.

Treatment:

Patient was counseled about the causes, nature and course of the condition and response to different treatment modalities which was followed by oral prophylaxis.

Patient was advised to use topical Kenacort orabase 0.1% ointment (triamcinolone acetonide) twice daily for 2 weeks after that the dose was tapered. Patient was also prescribed multivitamins and anti oxidants as supportive therapy. Patient reported a month after and was free of burning sensation in the oral cavity but oral lesions were persistent.

1 month post treatment

IV. Discussion

Lichen planus is a benign condition that affects skin and mucus membrane.

Among the various forms of OLP (reticular, atrophic, erosive, and bullous), atrophic lichen planus although not as common as the reticular form, but it is of more significance for the patient as such lesions are usually symptomatic.

In the present case, patient showed atrophic, erythematous areas on the gingiva. The periphery of the atrophic regions was bordered by fine, white radiating striae and plaques.

A clinical and a histopathological definition of OLP was formulated by the WHO in 1978[9] . Later, in 2003, van der Meij and van der Waal[10], proposed a modification in the WHO criteria , stating OLP diagnosis should be clinico-pathological.

According to this the clinical criteria includes presence of bilateral, more or less symmetrical lesions along with Presence of a lacelike network of slightly raised gray-white lines (reticular pattern). Hisopathologic criteria is presence of a well-defined band like zone of cellular infiltration that is confined to the superficial part of the connective tissue, consisting mainly of lymphocytes ,signs of liquefaction degeneration in the basal cell layer and absence of epithelial dysplasia. The present case is in accordance with this criteria.

V. Conclusion

Diagnosis of Lichen Planus requires a succession of clinical,and histopathologic procedures. In spite of correct diagnosis and treatment plan, management of these cases remains challenging for the clinician. The potentially malignant nature of oral lichen planus necessitates a long term follow-up regimen for all the cases.

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