

## CASE REPORT

# A DIAGNOSTIC DILEMMA: ORAL LICHEN PLANUS OR LICHENOID REACTION - A SERIES OF CASE REPORTS

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### ABSTRACT:

Oral lichen planus and oral lichenoid reactions/lesions (OLRs/OLLs) are clinically and histopathologically similar kind of lesions but with a distinct etiology. Diagnosing clinically or histopathologically alone the lesion is a dilemma. A clinicopathologic correlation is necessary to justify the patient's treatment as both OLP and OLL have different line of treatment to be followed. A series of case reports are presented here, for the better evaluation of the clinical and histopathologic features which are helpful in diagnosis or differentiation of OLP from OLR/ OLL.

Key words: Oral hypoglycemics, NSAIDs, Indirect immunofluorescence, Patch test.

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### INTRODUCTION:

Lichen planus was first described by Wilson in 1869, which in literal sense means algae over the rock. The word Lichenoid means Lichen like (suffix oid – like). Lichen planus (LP) is an autoimmune, inflammatory disease that affects oral and cutaneous tissues or both.<sup>1</sup> It has been reported in middle aged patients and is more prevalent in females than males. Oral lichen planus is also sometimes seen in children and young adults.<sup>2</sup> The terms oral lichenoid reactions (OLRs) or oral lichenoid lesions (OLLs) refer to lesions histologically and clinically similar to oral lichen planus (OLP) usually with identifiable underlying cause. They may be considered as disease by itself or as an exacerbation of existing OLP, by the presence of dental materials or medications.<sup>3</sup>

Lichen planus is a cell-mediated chronic condition in which there is damage to the basal keratinocytes in the oral mucosa. Classical cases affect approximately 2% of the population and presents itself as bilateral keratotic lesions of the buccal

mucosa, gingiva and tongue.<sup>4</sup> OLRs has been widely reported with the use of dental materials like amalgam, composites, dental acrylic and numerous drugs like beta blockers, dapsones, oral hypoglycemics, non-steroidal anti-inflammatory drugs, penicillamine, phenothiazines, sulfonyleureas and gold salts.<sup>5-9</sup>

Microscopically, lichen planus is characterized by hyperkeratosis, “saw-tooth” rete ridge formation, basal cell degeneration and presence of lymphocytic infiltration juxtaepithelially.<sup>10</sup> It is mandatory for all clinicians to be aware of its clinical presentation and management since it is one of the most common chronic conditions affecting the oral mucosa. Uptil now, the most widely accepted treatment for OLP is topical corticosteroids. Apart from that, some of the other adjunct treatments include retinoids, cyclosporine, tacrolimus, surgery and carbon dioxide laser.<sup>1</sup> Resolution of majority of OLR cases have been seen following replacement of causative restorations in some studies.<sup>7</sup> In the case of drug-

induced OLRs, due evaluation of the risk / benefit ratio of suspending the medication is required. As has been commented, even if the causal medication can be suspended, the lesions may take several months in improving. In addition, the pharmacological treatment of OLRs is often not feasible, because the long list of agents capable of causing such lesions includes many substances used to inhibit autoimmune T lymphocytes responses.<sup>11</sup> In most cases it is difficult to differentiate OLRs from OLP on the clinical or histopathologic basis alone. Indirect immunofluorescence study and patch test is useful in diagnosis.

#### **CASE REPORTS:**

##### **I CASE: (LICHEN PLANUS I)**

A 60 year old lady reported with the chief complaint of burning sensation of the mouth since 4-5 years while taking hot and spicy foods. Intraoral examination revealed white raised non scrapable lesion circumscribed by fine keratotic interlacing striae involving both sides of the buccal and alveolar mucosa posteriorly. Lesion was firm in consistency and grainy in texture. No skin lesion was visible. Medical and dental histories were non-contributory. Histopathological features revealed the presence of hyperplastic orthokeratinized stratified squamous epithelium along with areas of basal cell degeneration while connective tissue stroma consisted of chronic inflammatory cells predominantly lymphocytes subepithelially, bundles of loose collagen fibres, blood vessels, adipose tissue and muscle.

##### **II CASE: (LICHEN PLANUS II)**

A 25 year old male reported with the chief complaint of white spots and burning sensation in the mouth since 2 months. When patient was examined intraorally, diffuse multiple white popular lesions having erythematous component were seen on left and right buccal mucosa with radiating white striae from it. The lesions were soft in consistency, grainy in texture with ill defined margins. No associated skin lesion was seen. Medical and dental histories were non-contributory. Histopathological features confirmed the presence of hyperparakeratinized stratified squamous epithelium with areas of liquefactive degeneration of basal layer along with thick band of chronic inflammatory infiltrate is seen juxtaepithelially predominantly lymphocytes, Underlying fibrovascular connective tissue was composed of thick collagen bundles, adipose tissue and muscle bundles.

##### **III CASE: (LICHEN PLANUS III)**

A 40 year old female patient reported with the chief complaint of burning sensation of the mouth since 6 months. Intraoral examination revealed the presence of diffuse white lesion circumscribed by fine keratotic interlacing striae involving left side of the buccal mucosa posteriorly. Lesion was firm in consistency, rough in texture with irregular margins. No associated skin lesion was visible. Medical and dental histories were non-contributory. Histopathological findings included orthokeratinized stratified squamous epithelium having a dense band of chronic inflammatory infiltrate predominantly lymphocytes subepithelially along with focal areas of basal cell degeneration. Connective tissue stroma comprised of loosely arranged collagen fibres, blood vessels and muscle bundles in longitudinal and cross sections.

##### **IV CASE: (LICHENOID REACTION 1)**

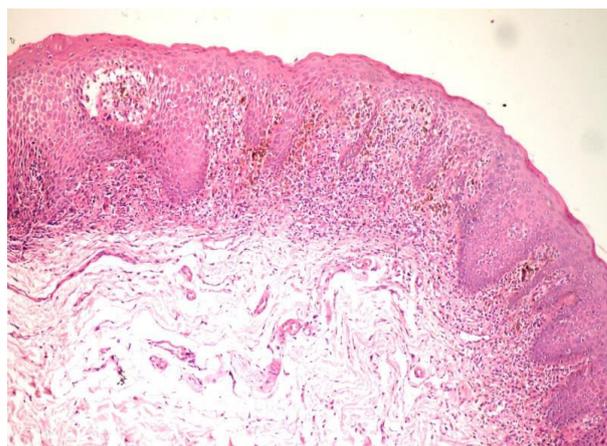
A 35 year old male patient reported with a chief complaint of burning sensation in left posterior buccal mucosa since 6 months. Past dental history of multiple amalgam restoration since 7 months in relation to 48 was also reported. On clinical examination macular lesion with peripheral striae was revealed. Extra oral examination revealed no relevant findings. Past medical history was not significant. Histopathologic examination revealed the presence of parakeratinized stratified squamous epithelium with irregular rete ridges. Subepithelial region shows chronic inflammatory infiltrate predominantly lymphocytes and plasma cells extending into submucosa close to muscles and adipocytes along with perivascular inflammatory cell infiltration.

##### **V CASE: (LICHENOID REACTION II)**

A 64 year old male patient reported with a chief complaint of burning sensation and pain on eating spicy food since 7-8 months. On clinical examination diffuse, white ulcerated lesion, non scrapable was observed on right upper and left lower marginal gingiva in relation to 14 and 34 respectively. Past medical history of diabetes under control with the usage of hypoglycaemic drug (Glimepiride 2 mg). Histopathologic examination revealed the presence of atrophic stratified squamous epithelium with basal cell degeneration in focal areas. Diffuse chronic inflammatory cells predominantly lymphocytes and plasma cells in vascular and edematous connective tissue stroma are also evident.



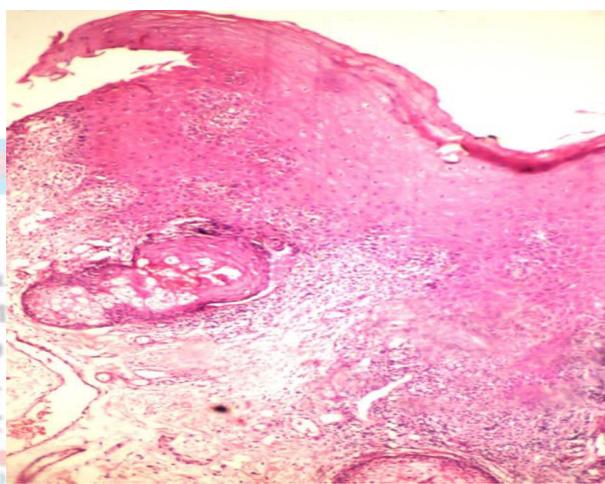
**Figure 1:** Clinical photograph of Oral Lichen Planus



**Figure 2:** Histopathological photomicrograph of Oral Lichen Planus



**Figure 3:** Clinical photograph of Oral Lichenoid Reaction



**Figure 4:** Histopathological photomicrograph of Oral Lichenoid Reaction

### VI CASE: (LICHENOID REACTION III)

A 40 year old female patient complains of burning sensation in left posterior buccal mucosa since 1 year. On clinical examination white reticular striae at the periphery of non scrapable patch was observed in relation to 26, 27. Past medical history revealed the use of hypoglycemic drugs since 2 years. Histopathologic examination revealed the presence of stratified squamous epithelium with irregular rete ridges. Underlying connective tissue shows diffuse band of mixed inflammatory cell infiltrate predominantly of lymphocytes and eosinophils. Blood vessels of varying shape and size along with muscle bundles are also evident.

### DISCUSSION:

Although lichen planus and lichenoid reactions are clinically similar due to the presence of erosive and reticular components in both lesions but a distinguishing feature between them could be the

presence of ulcerative component, atypical location as well as absence of bilateral manifestations. Despite recent advances in understanding the etiopathogenesis of oral lichen planus, the initial questions about its formation and the pathways are still unanswered. The pathogenesis of lichen planus is believed to involve both antigen-specific and non-specific mechanisms. Antigen-specific mechanisms include antigen presentation by basal keratinocytes and destruction of keratinocytes by CD8+ T-cells whereas non specific mechanisms include dual activities of degranulation of mast cells and matrix metalloproteinase activation in such lesions. The keratinocytes are recognized by the immune system as foreign substances, triggering the release of inflammatory mediators like cytokines, chemokines and chronic inflammatory infiltrate comprising mainly of T lymphocytes resulting in destruction of basal keratinocytes.<sup>11</sup>

The etiopathogenic mechanism by which these lesions are produced is not known. The literature has identified a series of triggering factors, such as dental restoration materials, graft-versus-host disease, and a broad range of drugs.<sup>12</sup>

It is well accepted, that on the basis of histopathologic aspects alone it is difficult to discriminate between various types of OLRs and with regard to OLP.<sup>13-16</sup> Clinically OLRs are very similar to OLP except very few differences like unusual site like palate and commonly unilateral occurrence.<sup>17</sup> Four types of oral lichenoid lesions (OLLs) can be distinguished, being 1) amalgam restoration, topographically associated lesions 2) drug related lichenoid lesions, 3) lichenoid lesions in chronic graft versus host disease (cGVHD) and 4) lesions that have a lichen planus like aspect, but that lack one or more characteristic clinical aspects.<sup>18</sup> histopathologically it has been suggested that the presence of a mixed subepithelial infiltrate, in contrast to the strict lymphohistiocytic infiltrate that defines OLP and a deeper more diffuse distribution within the lamina propria and superficial submucosa is a marker of drug related lichenoid oral lesion<sup>19</sup>. However, the sensitivity and specificity of histological diagnosis are very low. In addition, no definitive molecular diagnostic markers have been established to date. Van der Meij gave a modified diagnostic criteria of WHO (1978) according to which certain clinical aspects along with histopathological features could be suggestive of both oral lichen planus and lichenoid reactions as stated below.<sup>20</sup>

#### Clinical criteria

1. Presence of bilateral, more or less symmetrical lesions
2. Presence of a lacelike network of slightly raised gray-white lines (reticular pattern)
3. Erosive, atrophic, bullous and plaque-type lesions are accepted only as a subtype in the presence of reticular lesions elsewhere in the oral mucosa

In all other lesions that resemble OLP but do not complete the aforementioned criteria, the term “clinically compatible with” should be used

#### Histopathological criteria

1. 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.

2. Signs of liquefaction degeneration in the basal cell layer
3. Absence of epithelial dysplasia

When the histopathologic features are less obvious, the term “histopathologically compatible with” should be used.

#### Final diagnosis OLP or OLL/OLR

To achieve a final diagnosis, clinical as well as histopathologic criteria should be included

- A. OLP- A diagnosis of OLP requires fulfillment of both clinical and histopathologic criteria
- B. OLL-The term OLL will be used under the following conditions:

1. Clinically typical of OLP but histopathologically only compatible with OLP
2. Histopathologically typical of OLP but clinically only compatible with OLP
3. Clinically compatible with OLP and histopathologically compatible with OLP.

#### CONCLUSION:

Although the oral cavity may present lichen planus lesions with a distinct clinical configuration and distribution, the clinical presentation sometimes may simulate other muco cutaneous disorders thus making it a difficult disease to be identified. Therefore, for achieving a clear cut final diagnosis the clinical findings should always be co-related with histopathologic or immunologic diagnosis.

#### REFERENCES:

1. Sharma S, Saimbi CS, Koirala B. Erosive Oral Lichen Planus and its Management: A Case Series. J Nepal Med Assoc 2008;47(170):86-90.
2. Ismail SB, Kumar SK, Zain RB. Oral lichen planus and lichenoid reactions: etiopathogenesis, diagnosis, management and malignant transformation. J Oral Sci. 2007;49:89-106.
3. Scully C, Carrozzo M. Oral mucosal disease: Lichen planus. Br J Oral Maxillofac Surg.2008;46:15-21.
4. Thornhill MH, Sankar V, Xu XJ , Barrett AW, High AS, Odell EW, Speight PM, Farthing PM. The role of histopathological characteristics in distinguishing amalgam-associated oral lichenoid reactions and oral lichen planus. J Oral Pathol Med 2006;35:233–40.

5. Rice PJ, Hamburger J. Oral lichenoid drug eruptions: their recognition and management. *Dent Update* 2002;49:442-7.
6. Lind PO, Hurlen B, Lyberg T, Aas E. Amalgam-related oral lichenoid reaction. *Scand J Dent Res* 1986;94: 448-51.
7. Thornhill MH, Pemberton MN, Simmons RK, Theaker ED. Amalgam-contact hypersensitivity lesions and oral lichen planus, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; 95:291-9.
8. Lind PO. Oral lichenoid reactions related to composite restorations. Preliminary report. *Acta Odontol Scand* 1988;46:63-5.
9. Van Loon LA, Bos JD, Davidson CL. Clinical evaluation of fifty-six patients referred with symptoms tentatively related to allergic contact stomatitis. *Oral Surg Oral Med Oral Pathol* 1992;74:572-5.
10. Juneja M, Mahajan S, Rao NN, George T, Boaz K. Histochemical analysis of pathological alterations in oral lichen planus and oral lichenoid reactions. *Journal of Oral Science* 2006;48(4):185-93.
11. Sanchez SP, Bagan JV, Soriano J, Sarrion G. Drug-induced oral lichenoid reactions. A literature review. *J Clin Exp Dent*. 2010;2(2):e71-5.
12. Sugerma PB, Savage NW, Walsh LJ, Zhao ZZ, Zhou XJ, Khan A, Seymour GJ, Bigby M. The Pathogenesis of Oral Lichen Planus. *Crit Rev Oral Biol Med* 2002;13(4):350-65.
13. McCartan BE, McCreary CE. Oral lichenoid drug eruptions. *Oral Dis* 1997;3:58-63.
14. McCartan BE, Lamey P. Lichen planus--specific antigen in oral lichen planus and oral lichenoid drug eruptions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2000;89:585-7.
15. Rice PJ, Hamburger J. Oral lichenoid drug eruptions: their recognition and management. *Dent Update*. 2002;29:442-7.
16. Larsson A, Warfvinge G. The histopathology of oral mucosal lesions associated with amalgam or porcelain-fused-to-metal restorations. *Oral Dis*. 1995;1:152-8.
17. Lamey PJ, McCartan BE, Macdonald BE, MacKie RM. Basal cell cytoplasmic autoantibodies in oral lichenoid reactions. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1995;79:44-9.
18. Van der Waal. Oral lichen planus and oral lichenoid lesions; a critical appraisal with emphasis on the diagnostic aspects. *Med Oral Patol Oral Cir Bucal* 2009;14(7):E310-4.
19. Savage NW. Oral lichenoid drug eruption. *Oral Dis* 1997;3:55-7.
20. Van der Meij EH, van der Waal I. Lack of clinicopathologic correlation in the diagnosis of oral lichen planus based on the presently available diagnostic criteria and suggestions for modifications. *J Oral Pathol Med* 2003;32:507-12.

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