



## Erosive Lichen Planus: A Mucocutaneous Presentation

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**Vikrant Rai**

Department of Oral Pathology and Microbiology, Surendera Dental College and Research Institute  
Sriganganagar, Rajasthan, **India**

**Karthikeyan Ramalingam**

Department of Oral Pathology and Microbiology, Surendera Dental College and Research Institute  
Sriganganagar, Rajasthan, **India**

**Sandeep Goyal**

Department of Oral Pathology and Microbiology, Surendera Dental College and Research Institute  
Sriganganagar, Rajasthan, **India**

**Syed Wali Peeran**

Department of Periodontics, Faculty of Dentistry, Sebha University, Sebha, **Libya**

### Abstract:

Lichen planus is an autoimmune chronic mucocutaneous disease of indefinite etiology and variable clinical manifestations. It is common among the middle-aged and elderly people. The erosive form (ELP) is the second most common form. It is more symptomatic than non-erosive type. It is characterized by multifocal white lines with erosive ulcerated areas. This case report reviews the clinical approach to an erosive variant of OLP involving the oral mucosa and skin of a 54-year-old female patient.

**Keywords:** *Lichen Planus, Erosive, Autoimmune, Mucocutaneous, Indefinite Etiology*

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### 1. Introduction

Lichen planus(LP) is a common dermatosis that affects the skin, mucosae, and nails. The typical rash of LP is well described by the “6 Ps” well defined pruritic, polygonal, purplish, papules and plaques <sup>[1]</sup>. Many variants in morphology and location also exist, including oral, nail, linear, annular, erosive, atrophic, hypertrophic, inverse, eruptive, bullous, ulcerative, LP pigmentosus, lichen planopilaris, vulvovaginal, actinic, LP-lupus erythematosus overlap syndrome, and LP pemphigoids <sup>[2]</sup>. The erosive form is the second most common type of OLP. Variants of the erosive form include atrophic and

bullous forms. It clinically manifests as a mixture of erythematous and ulcerated areas bounded by finely radiating keratotic striae. Unlike the keratotic variants such as reticular and plaque type lesion, erosive OLP presents with symptoms ranging from intermittent mild pain to severe discomfort and carries an increased risk for malignant transformation. The microscopic appearance of LP is pathognomonic and shows hyperkeratosis with thickening of the granular cell layer, degeneration of basal cell layer and a band like lymphocytic inflammation in the subepidermal layer with civatte body (CBs) formation <sup>[3]</sup>. The clinical diagnosis of LP can be confirmed by classical histopathological findings.



However, direct immunofluorescence (CDIF) studies may be helpful in patients with no specific clinical or histological features, or with overlapping features of other diseases like DLE.

In this case presentation, we discuss Erosive LP with a mucocutaneous presentation involving the skin and oral cavity.

## **2. Case Report:**

A 55-year-old female patient reported to the department of oral and maxillofacial pathology, Surendra dental college and research institute, Sriganganagar, Rajasthan, India. Her chief complaint was burning sensation in the oral cavity for 6-8 months. History revealed that she had skin lesion 10 years back. Oral lesions started 6-8 months back because of some dental procedure is done intraorally. She visited a dentist, the dentist gave her a topical application but lesion has not healed.

Her past medical history revealed that she had lesions on the skin, abdomen and back region associated with severe itching which subsided on medication. But the lesions recurred after some time.

Past drug history revealed that she was on systemic steroids and vitamin supplements. On general examination, she had cutaneous lesions on the dorsum of both feet (Figure 1).

Intraoral examination revealed a reddish white erythematous lesion with variable brownish pigmentations. The lesion was extending 2-3cm behind the commissure to the 2<sup>nd</sup> molar region in the left buccal mucosa. The lesion was approximately 3cm anterior posteriorly and 2cm superior inferiorly. (figure 2). A white lesion with brownish areas which also showed radiating white striae was noted in the right buccal mucosa. Erythematous

patches were also noted on maxillary labial gingiva (figure3).

Routine blood investigations were performed before biopsy and it did not reveal any abnormalities. Allergy test revealed that she had hypersensitivity to paracetamol and etoricoxib. An incisional biopsy was performed and sent to the department of oral and maxillofacial pathology for further analysis.

Histopathology revealed varying degrees of hyper orthokeratosis on the surface of the stratified squamous epithelium. The thickness of the spinous layer was also variable with the absence of rete ridges in many areas. Evidence of destruction of the basal layer of epithelium accompanied by a dense, band-like infiltrate of lymphocytes immediately subjacent to the epithelium.

Correlating clinically, the histopathological features were suggestive of Erosive Lichen planus.

## **3. Discussion:**

LP (Greek “lichen= tree moss, Latin “planus” =Flat, even <sup>[4]</sup> was explained in 1869 by Dr. Wilson as an inflammatory disorder of the stratified squamous epithelia with unknown etiology. It was originally named “lichen ruber planus” and lichen psoriasis” <sup>[5]</sup>. Weyl initially described the characteristic surface markings on LP papules, known as Wickham striae with an increase in the thickness of granular cell layer <sup>[6]</sup>.

An abnormal T cell-mediated immune response is the main underlying factor which results in basal epithelial cells to be recognized as foreign bodies due to change in the antigenicity of their cell surface. However, the cause of this immune-mediated damage of basal cell layer is still not known. <sup>[7]</sup>.



OLP is a chronic autoimmune mucocutaneous disease primarily middle-aged women. The exact prevalence of LP is unknown. Nevertheless, the estimated prevalence of LP is in the range of 0.22 to 5% worldwide [8]. The epidemiological studies lack clear diagnostic criteria or a uniform methodology, furthermore, the disease classical presentation and the asymptomatic nature of the most common subtypes of OLP make the disease an undiagnosed health issue. [9]. OLP typically affects middle-aged adults of both genders. No sexual predilection is evident but some reports indicate a slight predominance in women up to a ratio of 2:1 [10]. Sharma et al [11] found the male to female ratio as 1:2. The lesion is typically bilateral and relatively symmetric. Our presentation is also on a 55-year-old female with bilateral lesions in the buccal mucosa.

Oral LP (OLP) can be the sole clinical presentation of the disease or accompanied by cutaneous or other mucosal manifestations including the genital area, gastrointestinal tract and eyes [12]. Although there are several clinical forms of OLP (reticular, patch, erosive, and bullous), the most common are the reticular and erosive forms. Oral lesions are more common than skin lesions, and in few cases, the former precedes the latter [13]. In our case, lesions were noted in the buccal mucosa, gingiva, skin on the dorsum of the foot, abdomen and back region.

The etiology of OLP appears to be multifactorial and complicated. Ismail et al [14] Reported a list of exacerbating factor for OLP and OLP reactions such as stress, drugs (anti-malarial, diuretics, gold salts, antiretroviral, beta blockers, and penicillamine), certain dental materials, and metals), chronic liver disease and hepatitis C virus, genetics and tobacco chewing.

Systemic diseases seen associated with OLP include diabetes mellitus, hypertension, ulcerative colitis, myasthenia gravis, lupus erythematosus, etc. In the present case, medication and dental procedure seems to be a possible aggravating factor.

In OLP, reticular and atrophic lesions usually tend to develop within all erosive lesions unlike other vascular-erosive diseases such as pemphigus and pemphigoid. The latter occurs as solitary erythematous lesions which are not usually associated with any white striae and pathognomonic feature being Nikolsky, s positive. This can aid in clinical differential diagnosis since erosive and atrophic forms of OLP usually show concomitant reticular forms. [10].

Erosive form of OLP has the highest malignant transformation rate when compared to the other variants [15].

Shi Q et al [16] performed a meta-analysis on the association between polymorphism of interleukins and OLP. But, they failed to show a statistical association between genetic polymorphism of IL-6, 174 G/C, IL-10 819 C/T and IL-10.1082, G/A and OLP susceptibility in any genetic models.

Gheorghe C et al [17] found an association of OLP with chronic C hepatitis infection in a meta-analysis of retrospective studies conducted since 1990 till now. They found a prevalence of chronic hepatitis C virus 3-9 times higher in patients with oral lichen planus compared to controls.

Xao et al published the only global gene expression analysis of the tissue from OLP. Adani RG et al [18] performed gene expression analysis of innate immune response activation in the epithelium with OLP. They concluded that the changes in expression at least for CXCL1, TLR1, and



CD14 could represent changes in the keratinocytes.

Topical or systemic corticosteroid is the mainstay of treatment for mild to moderate symptomatic lesions of OLP, and it functions by modulating patients' immune response. Other treatment modalities available include pharmacotherapy by immunomodulators chloroquine, immunosuppressants such as tacrolimus, newer drugs such as azathioprine, mycophenolate mofetil, psoralen and ultraviolet A therapy, and CO<sub>2</sub> LASER.<sup>[19]</sup>

#### 4. Conclusion:

Oral lichen planus is an autoimmune disease of unknown etiology and varying clinical manifestations. Erosive lichen planus (ELP) is more painful and debilitating than the non-erosive types and is characterized by multifocal white lines with erosive or ulcerated areas. This classical manifestation is rarer than other types and causes diagnostic difficulty. In addition, ELP requires topical or systemic treatment for healing and pain control.

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### Legends:



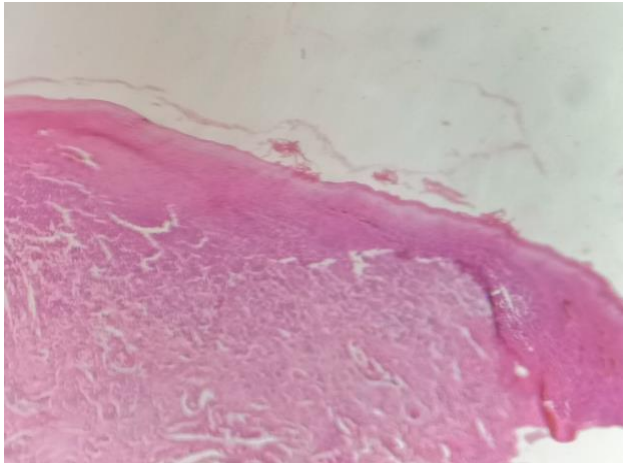
*Figure 1: Clinical picture showing the white patches on dorsum of the feet*



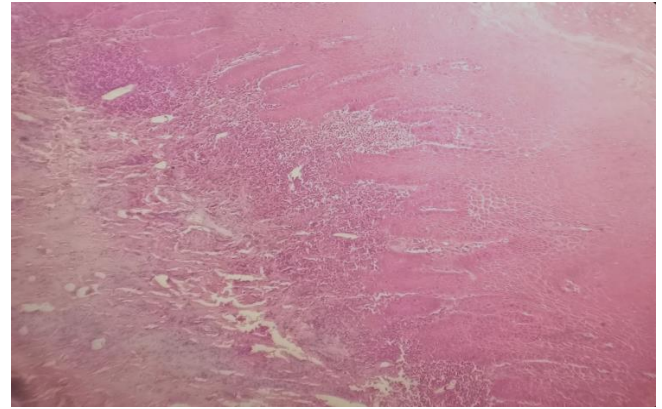
*Figure 2: Intra-oral clinical picture showing whitish lacy striae arranged in the buccal mucosa*



*Figure 3: Intra oral clinical picture showing the erythematous patches on maxillary gingiva*



*Figure 4: photomicrograph showing surface epithelium and subepithelial inflammation (4x, H & E).*



*Figure 5: Photomicrograph showing hyperkeratosis and basal cell degeneration of surface epithelium along with sub-epithelial band of chronic inflammatory cell infiltrate. (10x H&E).*