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*Letter to Editor*

# Clinicohistopathological study of oral lichen planus

Naseema Rashid[1], Najeeba Riyaz[2], Mampully Geetha[3]

Departments of [1]Dermatology and [3]Pathology, Government Medical College, [2]Department of Dermatology, KMCT Medical College, Kozhikode, Kerala, India.

***Corresponding author:**
Naseema Rashid,
Department of Dermatology,
Government Medical College,
Kozhikode, Kerala, India.

seema_rashu@yahoo.com

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Sir,

Lichen planus (LP) is a common dermatosis affecting 1–2% of population.[1] It affects skin, nails, mucosae, and hair. Oral lesions are seen in 60–70% of patients.[2] The prevalence of oral LP without any skin involvement is 0.4%.[2] The most common clinical form of oral LP is reticular pattern, though other morphologies are also described.[3]

The World Health Organization has included oral LP in the group of potentially malignant disorders. Squamous cell carcinoma remains the most serious complication of oral LP[4,5]. Reports suggest that 1.1% of oral LP progress to squamous cell carcinoma, and the risk is higher in individuals who smoke or consume alcohol. Coexisting hepatitis C infection is another risk factor for malignant transformation.[6] Erosive LP is the common clinical type associated with malignant transformation. It has been suggested that proliferation of basal layer cells induced by various inflammatory mediators promotes tumor development.[7]

This manuscript is based on the data collected for a cross-sectional study on white lesions in oral cavity that was carried out after getting clearance from the Institutional Ethics Committee and Written Informed Consent from individual study subject. Patients aged 20 years or above and who attended the outpatient clinics of dermatology and dental medicine departments of Government Medical College, Kozhikode during the 1 year period with white lesions in oral cavity and who were willing for biopsy were included in the mentioned study. Patients not willing to participate in the study were excluded from the study.

The current manuscript describes the clinicohistopathological features of oral LP as documented in the study.

The presence of keratotic white slightly elevated papules, lace-like network of slightly raised gray-white lesions or plaque-like configuration in oral cavity, gingiva or tongue was clinically diagnosed as oral LP. A detailed history regarding the evolution of disease, symptoms, duration of disease, history of similar illness, exposure to chemicals or drugs known to induce LP, and individual's habits such as smoking, betel nut chewing, and alcohol intake was enquired and documented using a preset pro forma. Family history of similar illness was noted.

Thorough examination of the oral cavity in good daylight was done. The site, number, location, and morphology of the lesions were recorded. Regional lymphadenopathy, when present, was noted. Tongue blade was used to find out whether the lesions were scrapable or not.

Routine blood and urine analysis, random blood sugar estimation, liver function tests, serology for anti-hepatitis C antibody, and potassium hydroxide smear to detect *Candida albicans* infection were carried out in all patients. A biopsy was performed in all cases.

Single biopsy was taken from each patient. In patients with multiple lesions, biopsy was taken from the most representative lesion.

The presence of well-defined band-like zone of cellular infiltration confined to the superficial part of connective tissue, consisting mainly of lymphocytes along with liquefaction degeneration of basal cell layer, was considered as histological evidence of oral LP.[8]

Diagnosis and grading of dysplastic changes were done according to the WHO classification. Dysplasia when limited to basilar and parabasilar portions of the epithelium was graded as mild to moderate and when entire thickness of epithelium was affected, it was considered as severe dysplasia. Enlarged and hyperchromatic nuclei, enlarged cells, large and prominent nucleoli, increased nuclear-to-cytoplasmic ratio, loss of polarity and loss of typical epithelial cell cohesiveness were the parameters used to diagnose dysplasia.[9]

The data were entered in Microsoft Excel. Clinical patterns and histology findings of oral LP were studied.

The study group comprised 53 cases of clinically diagnosed oral LP [Table 1].

Of the 53 patients, 36 (67.9%) were female. The age of the affected ranged from 21 to 70 years. 40/53 (75.5%) patients were 50 years or below. Duration of disease varied from 3 to 18 months in the study group.

At the time of presentation, 18/53 cases (34%) had no symptoms and 35 (66%) were symptomatic. All symptomatic patients complained of burning sensation in the oral cavity on taking spicy food. Among the symptomatic group, 14 (40%) had erosive form of LP. Thirty-eight of 53 cases (71.7%) had already received treatment with modern medicine at the time of recruitment to the study. The treatment received was clotrimazole mouth paint in 18 patients (34% of total), topical steroids in 12 patients (22.6% of total), and Vitamin B complex tablets in the remaining eight patients (15.1% of total).

Three (5.7%) and four (7.5%) patients in the study group suffered from diabetes mellitus and hypertension, respectively. Glibenclamide (2) and metformin (1) were the drugs received by diabetic patients. The duration of treatment with oral hypoglycemic agents preceded oral lesions by 2–3 years. Among those with hypertension, three received beta-blockers (5.7%) and one was on calcium channel blocker (1.9%). Oral lesion preceded drug intake in one of the three patients on beta-blockers and in the lone patient receiving calcium channel blocker. Time interval between onset of drug intake and appearance of oral LP, varied from 5 to 8 years in the remaining two patients on beta-blockers.

Two others (3.8%) had amalgam filling of teeth that were performed 10 years and 15 years ago, respectively. Among 17 male patients with LP, 4 (23.5%) had the habit of cigarette smoking. None of the female patients had the habit of smoking or any substance abuse.

Cutaneous LP was present in only 17% of the study subjects (nine patients). In most patients, LP lesions were located in the buccal mucosa (45 patients, 84.9%). This was followed by dorsum of tongue (22 cases, 41.5%). Other sites affected were labial (10 patients, 18.9%) and alveolar mucosae (3 patients, 5.7%) and palate (2 cases, 3.7%). Forty-one (77.4%) patients had more than one site affected. Thirty-five patients (66%) had lesions on buccal mucosa bilaterally.

Most common clinical type of LP documented in the study was the reticular form [Figure 1] (25, 47.2%) followed by plaque [Figure 2] (20, 37.7%), papular (19, 35.8%), and erosive forms (14, 26.4%). All four smokers had plaque type lesions.

Cervical lymphadenopathy was not documented in any of the cases.

None of the patients manifested abnormal liver function tests. Serology for anti-hepatitis C virus antibody was negative in all. Eight patients (15.1%) manifested colonization by

**Table 1:** Age and sex distribution of the study group.

| Age group in years | Lichen planus (53) | | |
|---|---|---|---|
| | Male | Female | Total (%) |
| 21–30 | 1 | 5 | 6 (11.3) |
| 31–40 | 5 | 16 | 21 (39.6) |
| 41–50 | 6 | 7 | 13 (24.5) |
| 51–60 | 2 | 5 | 7 (13.2) |
| 61–70 | 3 | 3 | 6 (11.3) |
| 71–80 | 0 | 0 | 0 (0) |
| 81–90 | 0 | 0 | 0 (0) |
| Total | 17 | 36 | 53 (100) |

*Percentage of total in each category is given in brackets

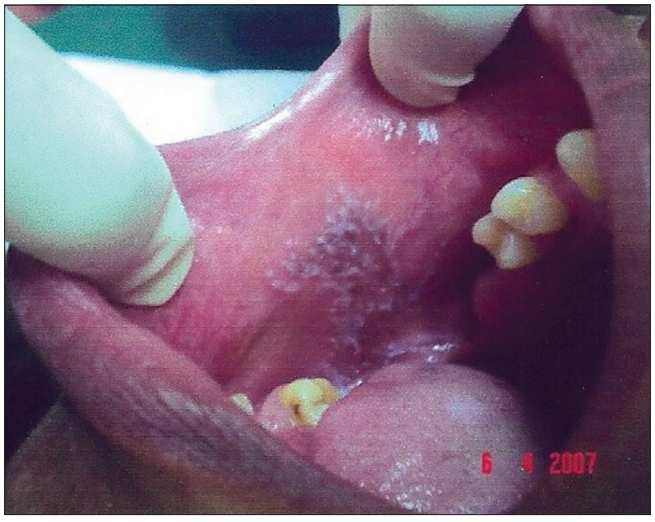


**Figure 1:** Reticular oral lichen planus.

candida (all erosive type) and two of them gave history of treatment with topical steroids previously.

Clinical and histopathological correlation was present in 51 cases (96.2%). Nonspecific features were noted in 2 patients (3.8%); no evidence of dysplasia or malignant transformation was noted in any of the 53 patients [Figure 3].

Predilection of oral LP for female sex documented by us was consistent with the previous studies.[1] Higher percentage of affected being young or middle-aged was also concordant to previous literature.[1] Hormonal influences have been cited as the reason for the female predilection.[9] However, Chitturi *et al.* in their study documented no sex predilection in oral LP.[10]

The majority of oral LP patients manifesting symptomatic lesions in the study was as reported earlier and was expected among patients seeking treatment in a tertiary referral center.[9]

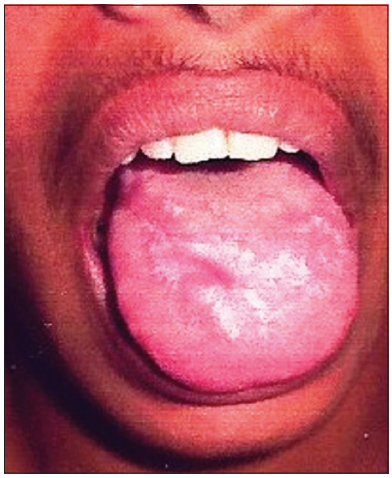

**Figure 2:** Plaque type oral lichen planus on dorsum of tongue.

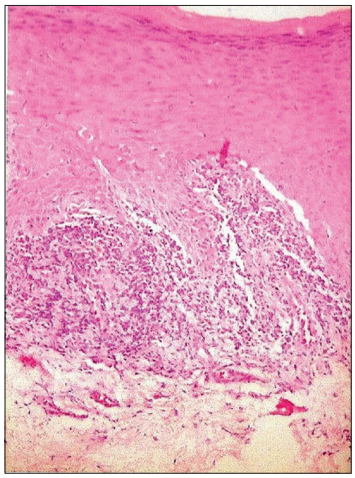

**Figure 3:** Biopsy from oral lichen planus showing basal cell degeneration without any dysplastic changes (hematoxylin and eosin, ×100).

Exposure to medications such as glibenclamide and beta-blockers and mercury amalgams in dental fillings as noted in some of our patients are documented as precipitating factors for LP.[3] Whether the drugs played a causative role in our patients remains unclear since we do not have information on the effect of withdrawal and substitution of the mentioned drugs. The coexistence of hypertension or diabetes mellitus with LP in seven of our study participants was consistent with literature.[10]

Only 17% of the study group manifesting cutaneous LP could be attributed to the selection bias of including only those who were willing for oral biopsy in the study. Patients who had lesions limited to oral mucosae are more likely to be concerned about the oral manifestation and hence agreed for oral biopsy.

The common type of oral LP identified, the areas of oral mucosa affected, and the involvement of multiple sites in nearly 80% of the affected were concordant to the previous studies.[10,11]

Candidiasis in 15.1% of the study group noted by us was consistent with previous data. It is considered to be a secondary colonizer in oral LP. Whether candida infection has a direct etiological role in oral LP remains unclear.[3] Treatment with topical steroids might have acted as a predisposing cause in two of our cases.

Serology for hepatitis C (the infection which is known to be associated with LP) was negative in all the study subjects. This was comparable to another Indian study.[12]

The absence of dysplastic changes observed in the study group was contrary to the findings of Werneck *et al.* who reported mild or moderate dysplasia in 50% patients.[11] Most of their study subjects having the habit of smoking or alcohol intake which are known risk factors for dysplastic changes in oral mucosa might have contributed to this finding.[11] Another study recorded dysplastic changes in 1.1% of oral LP cases and one of them (8.3%) went on to develop squamous cell carcinoma in 3-year time.[13] The absence of dysplastic changes in our study participants cannot be taken as an evidence of oral LP showing less risk for neoplastic transformation in our population since the median time interval documented for progression of oral LP to squamous cell carcinoma is 5 years whereas the longest duration of disease documented in the current study was 18 months.[4]

Small sample size and lack of follow-up with serial biopsies were the major study limitations. Moreover, since the manuscript is based on the data collected for the study on white lesions in oral cavity, some of the lichen planus cases (especially erosive form which is at greatest risk for malignant transformation) might have been excluded.

Oral LP is not an uncommon disease, especially in patients seeking care in a tertiary referral center. The prospective studies with a large sample size designed to analyze serial

biopsies may help to document the risk of malignant transformation associated with oral LP.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms

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Nil.

### Conflicts of interest

There are no conflicts of interest.

## REFERENCES

1. Parmar D, Thacker K, Shah J. Assessment of griseofulvin in the management of lichen planus. Int J Res Dermatol 2017;3:523-6.
2. Omal PM, Jacob V, Prathap A, Thomas NG. Prevalence of oral, skin, and oral and skin lesions of lichen planus in patients visiting a dental school in Southern India. Indian J Dermatol 2012;57:107-9.
3. Gupta S, Jawanda MK. Oral lichen planus: An update on etiology, pathogenesis, clinical presentation, diagnosis and management. Indian J Dermatol 2015;60:222-9.
4. Shen ZY, Liu W, Feng JQ, Zhou HW, Zhou ZT. Squamous cell carcinoma development in previously diagnosed oral lichen planus: *De novo* or transformation? Oral Surg Oral Med Oral Pathol Oral Radiol Endodontol 2011;112:592-6.
5. Peng Q, Zhang J, Ye X, Zhou G. Tumor-like microenvironment in oral lichen planus: Evidence of malignant transformation? Expert Rev Clin Immunol 2017;13:635-43.
6. Aghbari SM, Abushouk AI, Attia A, Elmaraezy A, Menshawy A, Ahmed MS, *et al*. Malignant transformation of oral lichen planus and oral lichenoid lesions: A meta-analysis of 20095 patient data. Oral Oncol 2017;68:92-102.
7. Tampa M, Caruntu C, Mitran M, Mitran C, Sarbu I, Rusu LC, *et al*. Markers of oral lichen planus malignant transformation. Dis Markers 2018;2018:1959506.
8. Zhang L, Michelsen C, Cheng X, Zeng T, Priddy R, Rosin MP. Molecular analysis of oral lichen planus. A premalignant lesion? Am J Pathol 1997;151:323-7.
9. Speight PM. Update on oral epithelial dysplasia and progression to cancer. Head Neck Pathol 2007;1:61-6.
10. Chitturi RT, Sindhuja P, Parameswar RA, Nirmal RM, Reddy BV, Dineshshankar J, *et al*. A clinical study on oral lichen planus with special emphasis on oral hyperpigmentation. J Pharm Bioallied Sci 2015;7 Suppl 2:S495-8.
11. Werneck JT, Costa JO, Stibich CA, Leite CA, Dias EP, Junior AS. Oral lichen planus: Study of 21 cases. An Bras Dermatol 2015;90:321-6.
12. Jayavelu P, Sambandan T. Prevalence of hepatitis C and hepatitis B virus infection(s) in patients with oral lichen planus. J Pharm Bioallied Sci 2012;4 Suppl 2:S397-405.
13. Irani S, Esfahani A, Ghorbani A. Dysplastic change rate in cases of oral lichen planus: A retrospective study of 112 cases in an Iranian population. J Oral Maxillofac Pathol 2016;20:395-9.

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