# Clinical Characteristics of 539 Patients with Oral Lichen Planus

Xiao Song LIU[1], Jian Qiu JIN[2], Yang LIU[1], Jie CAO[1], Hong Wei LIU[1]

**Objective:** *To assess the clinical characteristics of oral lichen planus (OLP) in a Chinese cohort.*
**Methods:** *A total of 539 OLP patients were enrolled in this study. Clinical and histopathological examinations were used for diagnosis. All data were analyzed statistically by* t *test, chi-square test, and Spearman correlation analysis with SPSS.*
**Results:** *The mean age of OLP patients was 50.2 years. The ratio of males to females was 1:1.44. Reticular type occurred in 56.40% of the patients, followed by erosive type (24.86%) and erythematous type (18.74%). Patients aged less than 40 years were susceptible to reticular lesions ($P = 0.001$) whereas patients more than 60 years were susceptible to erosive lesions ($P < 0.05$). In total, 25.34% of the OLP male patients were smokers, higher than that of the control group (16.76%; $P < 0.05$). With the increase of duration and quantity of tobacco consumption, the erosion type became less frequent ($P = 0.009$ and 0.007 respectively). However, no difference in smoking was observed between the women with and without OLP.*
**Conclusion:** *OLP occurs more commonly in middle-aged women. In patients younger than 40 years, the reticular type occurs more often, whereas erosion type is more common in patients older than 60 years. Erosion was less frequent in the OLP patients who smoked.*
**Key words:** *lichen planus, oral disease, mucosa oral lichen planus, smoking*

Lichen planus (LP) is a common, mucocutaneous, chronic inflammatory disorder affecting stratified squamous epithelium, including oral and genital mucosa, skin and nails. The frequency of oral LP (OLP) is 0.4 to 2%[1,2]. About 15% of OLP patients also had cutaneous lesions[3] and 20% of female patients had genital mucosa involvement[4]. Buccal mucosa is the most common site affected in the oral cavity, followed by tongue, gingiva, labial mucosa and vermilion of the lower lip[5–7]. OLP is classified into three clinical types: reticular, erythematous (atrophic) and erosive (ulcerated, bullous)[8].

1 Department of Traditional Chinese Medicine and Oral Medicine, Peking University School and Hospital of Stomatology, Beijing, P.R. China
2 Department of of Dentistry, Beijing Hospital, Beijing, P.R. China

**Corresponding author:** Dr Hong Wei LIU, Department of Traditional Chinese Medicine and Oral Medicine, Peking University School and Hospital of Stomatology, #22 Zhongguancun Nandajie, Haidian District, Beijing, 100081, P.R. China. Tel: 86-10-62179977 ext 2571; Fax: 86-10-62183939. E-mail: hongweiL2569@gmail.com

## Materials and Methods

From 1988 to 2007, 539 patients were diagnosed with OLP clinically and histopathologically at the Department of Oral Medicine, Peking University Hospital of Stomatology, China. Detailed inquiries were taken, including the age of onset, gender, skin and genital involvement, general health condition, family history, and tobacco consumption habits.

Biopsy specimens from the oral and cutaneous lesions were fixed in formalin and processed according to routine histopathological procedures. Hematoxylin and eosin (H&E)-stained tissue sections were evaluated under a light microscope.

To evaluate the influence of tobacco on the occurrence of OLP, 437 individuals with normal oral mucosa, who came for health examinations at the First Hospital, Peking University, were enrolled as the control.

SPSS was used to analyze the data. Measurement and enumeration data were compared by *t* test and chi-square test respectively. The correlation was analyzed by Spearman correlation analysis. Statistical significance for all analyses was set at $P < 0.05$.

## Results

Among the 539 OLP patients, the mean age of onset was 50.2 years (range 7 to 86 years). The ratio of males to females was 1:1.44. In the male patients, the mean age of onset was 49.6 years (range 16 to 83 years), while in the female patients it was 50.5 years (range 7 to 86 years). There was no statistically significant difference between men and women for the age of OLP occurrence ($P > 0.05$). In the control group, the mean age was 52 years (range 22 to 82 years) and the ratio of males to females was 1:1.44.

Seven patients were younger than 20 years and five were older than 80 years, as shown in Table 1. The youngest was a 7-year old girl with a congenital heart disorder. Based on the thin plate, longitudinal ridging and fissuring, and pterygium on her nails, she was diagnosed with nail LP by the dermatologist.

Reticular type was predominant (56.40%), followed by erosive (24.86%) and then erythmatous types (18.74%). According to the Spearman correlation analysis, 21 to 40-year-old patients were susceptible to reticular lesions ($P = 0.001$). By contrast, erosion type was often seen in patients of 61 to 80 years ($P < 0.05$), as shown in Table 2. Eight patients (1.48%) also had extra-oral lesions of LP, seven patients with lesions on the skin, and one on the nails. No genital lesions were observed.

Among the male patients with OLP, the proportion of smokers was 25.34%, which is higher than that in the control group (16.76%; $P < 0.05$). However, no statistically significant difference in smoking was observed between the women with and without OLP, as shown in Table 3. Smokers were not susceptible to erosive OLP ($P = 0.008$). Owing to the statistically negative correlation of erosive OLP and smoking, it was concluded that OLP became less erosive with the increase of duration and quantity of tobacco consumption ($P = 0.009$ and 0.007 respectively), as shown in Table 4.

In total, 197 patients had additional systemic disorders, including 16 (2.97%) with diabetes mellitus, 34 (6.31%) with gastrointestinal disorders, 7 (1.30%) with thyroid gland disorders and 9 (1.67%) with gynecological diseases. In addition, the frequency of hepatitis in OLP patients was 3.40%: 11 (2.04%) infected with hepatitis A, 6 (1.11%) with hepatitis B and 1 (0.19%) with hepatitis C. The site and types of lesion were not associated with diabetes and hepatitis significantly (data not shown). Moreover, one female patient and her sister both had OLP.

**Table 1** General status of OLP patients

| | Number | Percentage (%) |
|---|---|---|
| Gender | | |
| male | 221 | 41.00 |
| female | 318 | 59.00 |
| Age (years) | | |
| ≤20 | 7 | 1.30 |
| 21–40 | 123 | 22.82 |
| 41–60 | 283 | 52.50 |
| 61–80 | 121 | 22.45 |
| ≥81 | 5 | 0.93 |
| Site | | |
| labial mucosa | 112 | 20.78 |
| gingival mucosa | 85 | 15.77 |
| buccal mucosa | 421 | 78.11 |
| lingual dorsum | 133 | 24.68 |
| ventral tongue | 183 | 33.95 |
| floor of mouth | 7 | 1.30 |
| palate mucosa | 8 | 1.48 |
| Type | | |
| reticular | 304 | 56.40 |
| erythematous | 101 | 18.74 |
| erosive | 134 | 24.86 |
| Accompanied with extra-oral LP | 8 | 1.48 |
| skin LP | 7 | 87.50 |
| nail LP | 1 | 12.50 |
| genital LP | 0 | 0 |
| Smoking | 62 | 11.50 |
| Systemic disorders | 197 | 36.55 |
| diabetes mellitus | 16 | 2.97 |
| hepatitis | 18 | 3.40 |
| thyroid gland disorders | 7 | 1.30 |
| gynecological disorders | 9 | 1.67 |
| gastrointestinal disorders | 34 | 6.31 |

**Table 2** Correlation of type of lesions and age

| Age (years) | Reticular | | Erosive | |
|---|---|---|---|---|
| | r | P | r | P |
| 21–40 | 0.148 | 0.001** | –0.140 | 0.001** |
| 61–80 | –0.092 | 0.033* | 0.093 | 0.030* |

**P < 0.05; **P < 0.01*

**Table 3** The relationship of OLP and smoking

| | Patients with OLP *(n = 539)* | | People with normal mucosa *(n = 437)* | |
|---|---|---|---|---|
| | Male | Female | Male | Female |
| Smoking | 56 (25.34%)* | 6 (1.89%) | 30 (16.76%) | 3 (1.16%) |
| Nonsmoking | 165 (74.66%) | 312 (98.11%) | 149 (83.24%) | 255 (98.84%) |
| Total | 221 (100%) | 318 (100%) | 179 (100%) | 258 (100%) |

**P < 0.05 versus control group*

**Table 4** Correlation of type of lesions and smoking

| Type | Smoking | | History | | Quantity | |
|---|---|---|---|---|---|---|
| | r | P | r | P | r | P |
| Reticular *(n = 304)* | 0.070 | 0.105 | 0.065 | 0.132 | 0.072 | 0.094 |
| Erythematous *(n = 101)* | 0.035 | 0.415 | 0.042 | 0.336 | 0.036 | 0.410 |
| Erosive *(n = 134)* | –0.114 | 0.008* | –0.113 | 0.009* | –0.116 | 0.007* |

**P < 0.01*

## Discussion

Similar to previous reports[6,7,9,10], the present results showed that OLP occurred most commonly in middle age and more frequently in women than in men. However, the age of onset between men and women was not significantly different.

OLP is rare in children. Laeijendecker et al[11] showed that the frequency of OLP in child dermatology patients was 0.3%. No children younger than 16 years old develop OLP, and only 1% of patients under 20 years old develop OLP[10]. Although Asian people are thought to be predisposed to develop LP, only four children out of 674 (0.59%) Chinese aged from 10 to 13 years developed OLP and one case was combined with cutaneous lesions[12]. Similarly, the present data showed that seven patients (1.30%) were under 20 years, and two of them wer aged 7 and 13 years.

Generally, OLP patients manifest multiple clinical features, including reticular, plaque-like, bullous, erythematous and ulcerative lesions, which are classified into three types: reticular (including classical reticular and plaque-like), erythematous (atrophic) and erosive (bullous and ulcerated)[8]. In the present investigation, reticular lesions were predominant and more common in patients 21 to 40 years old. However, erosive lesions were more frequent in 61 to 80-year-old patients, consistent with previous studies by Xue et al[12]. Cutaneous and genital LP can precede or erupt concurrently with or after oral lesions[7]. In contrast to previous reports in which 15% of OLP patients developed cutaneous lesions[3] and 20% of females with OLP erupted genital lesions[4], the present data showed that only 1.48% of the patients had extra-oral lesions, among which cutaneous lesions were predominant and no genital lesions were observed.

OLP was uncommonly accompanied with nail lesions[3]. And in children with OLP, skin was thought to be the major extra-oral site affected[13]. However, in the present data, a 7-year-old patient was also diagnosed with nail LP in addition to the oral lesions, which presented with thin plates with longitudinal ridging, fissuring and ptyergium healing from a scar. The little girl had an untreated congenital heart disorder. It was not known whether the congenital heart

disorder might have played a role in the development of LP.

Most previous reports have not supported the relationship between OLP and smoking[12,14–16]. It was also difficult in the present study to predict the role of tobacco in the occurrence of OLP due to the inconsistent results among men and women patients. However, smoking might influence the progression of this disease. The reticular form was predominant in smokers, as reported by Machado et al[17]. In the present investigation, the OLP patients that smoked had less erosive lesions; with an increase in duration and amount of tobacco consumption, the probability of erosive OLP decreased. Tobacco might promote epithelial keratosis and inhibit the occurrence of erosive OLP.

For the etiology and pathogenesis of OLP, the role of systemic disorders is always considered. It is generally agreed that the frequency of erosive OLP increased in patients with diabetes mellitus[18]. However, no such correlation was noticed in this study in the OLP patients with diabetes. Similarly, the association with hepatitis was also negative.

The genetic contribution to LP is still uncertain. In some reports, the frequency of familial LP ranged from 1% to 11%[19–25]. And the genetic predisposition was supported by Carrozzo et al[26]. The gene polymorphism of the first intron of the promoter gene of IFN-gamma was thought to be a risk in the development of OLP, and the increased frequency of the -308A TNF-alpha allele was thought to contribute to the skin lesions[26]. While in a recent study in a Chinese cohort with Han ethnicity, the frequency of the TNF-alpha-308A allele was thought to be associated with oral erosive lesions, and the polymorphism of IL-10 might influence the susceptibility to OLP[27]. In contrast, other common factors that family members have were thought to be involved in the occurrence of the disease[10], such as environmental and psychological factors.

In conclusion, OLP occurs more commonly in middle-aged women. The reticular form is predominant in patients younger than 40 years old, whereas erosive OLP is predominant in patients older than 60 years of age. Erosive lesions were less frequent in the OLP patients that smoked.

## References


1. Bouquot JE, Gorlin RJ. Leukoplakia, lichen planus, and other oral keratoses in 23,616 white Americans over the age of 35 years. Oral Surg Oral Med Oral Pathol 1986;61:373–381.
2. Scully C, Beyli M, Ferreiro MC, Ficarra G, Gill Y, Griffiths M et al. Update on oral lichen planus: etiopathogenesis and management. Crit Rev Oral Biol Med 1998;9:86–122.
3. Eisen D. The evaluation of cutaneous, genital, scalp, nail, esophageal, and ocular involvement in patients with oral lichen planus. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;88:431–436.
4. Rogers RS 3rd, Eisen D. Erosive oral lichen planus with genital lesions: the vulvovaginal–gingival syndrome and the peno-gingival syndrome. Dermatol Clin 2003;21:91–98, vi–vii.
5. Silverman S Jr, Gorsky M, Lozada-Nur F. A prospective follow-up study of 570 patients with oral lichen planus: persistence, remission, and malignant association. Oral Surg Oral Med Oral Pathol 1985;60:30–34.
6. Bagan-Sebastian JV, Milian-Masanet MA, Penarrocha-Diago M, Jimenez Y. A clinical study of 205 patients with oral lichen planus. J Oral Maxillofac Surg 1992;50:116–118.
7. Eisen D. The clinical features, malignant potential, and systemic associations of oral lichen planus: a study of 723 patients. J Am Acad Dermatol 2002;46:207–214.
8. Eisen D, Carrozzo M, Bagan Sebastian JV, Thongprasom K. Oral lichen planus: clinical features and management. Oral Dis 2005;11:338–349.
9. Carrozzo M, Gandolfo S. The management of oral lichen planus. Oral Dis 1999;5:196–205.
10. Ingafou M, Leaol JC, Porter SR, Scully C. Oral lichen planus: a retrospective study of 690 British patients. Oral Dis 2006;12:463–468.
11. Laeijendecker R, Van Joost T, Tank B, Oranje AP, Neumann HA. Oral lichen planus in childhood. Pediatr Dermatol 2005;22:299–304.
12. Xue JL, Fan MW, Wang SZ, Chen XM, Li Y, Wang L. A clinical study of 674 patients with oral lichen planus in China. J Oral Pathol Med 2005;34:467–472.
13. Sharma R, Maheshwari V. Childhood lichen planus: a report of fifty cases. Pediatr Dermatol 1999;16:345–348.
14. Pentenero M, Broccoletti R, Carbone M, Conrotto D, Gandolfo S. The prevalence of oral mucosal lesions in adults from the Turin area. Oral Dis 2008;14:356–366.
15. Campisi G, Di Fede O, Craxi A, Di Stefano R, Margiotta V. Oral lichen planus, hepatitis C virus, and HIV: no association in a cohort study from an area of high hepatitis C virus endemicity. J Am Acad Dermatol 2004;51:364–370.
16. Gupta PC, Murti PR, Bhonsle RB, Mehta FS, Pindborg JJ. Effect of cessation of tobacco use on the incidence of oral mucosal lesions in a 10-yr follow-up study of 12,212 users. Oral Dis 1995;1:54–58.
17. Machado AC, Sugaya NN, Migliari DA, Matthews RW. Oral lichen planus. Clinical aspects and management in fifty-two Brazilian patients. West Indian Med J 2004;53:113–117.
18. Bagan JV, Donat JS, Penarrocha M, Milian MA, Sanchis JM. Oral lichen planus and diabetes mellitus. A clinicopathological study. Bull Group Int Rech Sci Stomatol Odontol 1993;36:3–6.
19. Gibstine CF, Esterly NB. Lichen planus in monozygotic twins. Arch Dermatol 1984;120:580.
20. Grunnet N, Schmidt H. Occurrence of lichen planus in a family. Genetic susceptibility or coincidence? Clin Exp Dermatol 1983;8:397–400.
21. Katzenelson V, Lotem M, Sandbank M. Familial lichen planus. Dermatologica 1990;180:166–168.
22. Kofoed ML, Wantzin GL. Familial lichen planus. More frequent than previously suggested? J Am Acad Dermatol 1985;3:50–54.
23. Luis-Montoya P, Domínguez-Soto L, Vega-Memije E. Lichen planus in 24 children with review of the literature. Pediatr Dermatol 2005;22:295–298.
24. Lin AN, Srolovitz H, Billrick RC. Familial lichen planus. Cutis 1986;37:135–136.
25. Bermejo-Fenoll A, López-Jornet P. Familial oral lichen planus: presentation of six families. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2006;102:e12–15.
26. Carrozzo M, Uboldi de Capei M, Dametta E, Fasano ME, Arduino P, Broccoletti R et al. Tumor necrosis factor-alpha and interferon-gamma polymorphisms contribute to susceptibility to oral lichen planus. J Invest Dermatol 2004;122:87–94.
27. Bai J, Jiang L, Lin M, Zeng X, Wang Z, Chen Q. Association of polymorphisms in the tumor necrosis factor-α and interleukin-10 genes with oral lichen planus: a study in a Chinese cohort with Han ethnicity. J Interferon Cytokine Res 2009;29:381–388.