

## CLINICAL EVALUATION OF 150 SAUDI PATIENTS WITH LICHEN PLANUS

Abdullah M. AlDosari, BDS, MSD, PhD\*; Maysara al Shawaf, BDS, MS\*\*;  
Nii Otu Nartey, BDS, MSc, MRCD\*\*\*; Asma'a Faden, BDS\*\*\*\*

مائة وخمسون مريضاً سعودياً مصابون بالحزاز المنبسط تمت دراسة حالتهم خلال اثني عشر عاماً. فقد تم تقييم حالاتهم فيما يخص العمر والجنس ومواقع الإصابة بجانب حشوات الأسنان والتعويضات السنية من حيث وجودها من عدمه. وقد أشارت نتائج هذه الدراسة إلى ارتفاع نسبي بشكل عام في إصابة النساء (53%) مقارنة بالرجال (47%) ومعظم حالات الإصابة كانت في الفترة الزمنية بين الأعمار 41 و 50 سنة. من الدراسة الإكلينيكية للمرضى تبين أن النوع الشبكي هو الأكثر شيوعاً (56%) يتبعه كل من المتخدش (26%) الطبقي (11%) والناعم (6%) والفقاعي (4%) وكانت أكثر المواقع إصابة في الفم كان الحد (83%) وأقلها اللسان (5%). كما قد تم تحليل حالات المرضى من حيث المتغيرات القمية والجسمية بجانب دراسة طبيعة الحزاز المنبسط المرضية وقابليته للتسرب.

One hundred and fifty patients with oral lichen planus (LP) seen over a period of about 12 years were retrospectively studied and evaluated. Age, gender, site of lesion, presence of dental restorations and/or appliances were recorded and analyzed. Findings showed a slight preponderance of females (53%) to males (47%). The highest prevalence was found to be between 41-50 years of age. Among the different clinical forms of oral LP, the reticular type was the most common (56%) followed by erosive (26%), plaque (11%) atrophic (6%) and bullous (0.4%) types. The buccal mucosa was the most commonly affected site (83%) while the least affected was the ventrum of the tongue (5%). Beside pathogenesis and premalignant potential of lichen planus, local and systemic findings are discussed.

### Introduction

Lichen planus (LP) is one of the important mucocutaneous lesions that should be of concern to dental clinicians. Its prevalence among the general population was reported to vary between 0.02% and 2%.<sup>1,3</sup> A study of 981 patients with oral lesions referred to an oral medicine private practice clinic showed that 20% of patients had oral LP.<sup>4</sup> The different clinical and histological features of oral LP had been extensively discussed in several review reports.<sup>5,9</sup> The literature show that only two studies were reported on oral LP among the Saudi populations with a prevalence of 0.6% and 1.7%, respectively.<sup>10,11</sup>

The purpose of this study was to present our clinical experience with 150 Saudi patients who had oral LP and were seen over a period of about 12 years.

### Materials and Methods

One hundred and fifty patients with oral LP were examined in the Oral Medicine Clinic at the College of Dentistry, King Saud University during the period 1981-1993. Some of these patients were referred by general practitioners in the city of Riyadh, where the College is considered to be one of the main referral centers for oral diseases. Other patients were referred from different parts of the Kingdom of Saudi Arabia. Another group of patients was referred from oral diagnosis clinic in the same College where their lesions were discovered during a routine dental examination.

Age, gender, site of lesion, presence of dental restorations and/or appliances were recorded and analyzed. Classification of cases was based on the scheme of Scully and El-Kom.<sup>6</sup> The diagnosis, especially for the reticular or plaque type of LP, was based on the history and clinical examination. Cases presenting atrophic, erosive or bullous type were biopsied whenever the diagnosis was questionable [Figs. 1-2].

Received 12/12/96; revised 26/02/97, accepted 01/03/97

\* Assistant Professor and Head, Division of Oral Medicine & Diagnosis, Department of Maxillofacial Surgery & Diagnostic Sciences, King Saud University College of Dentistry.

\*\* Assistant Professor, Department of Maxillofacial Surgery & Diagnostic Sciences, King Saud University College of Dentistry.

\*\*\* Former Lecturer, Department of Maxillofacial Surgery and Diagnostic Sciences, King Saud University College of Dentistry.

\*\*\*\* Postgraduate Student, Department of Maxillofacial Surgery & Diagnostic Sciences, King Saud University College of Dentistry, P.O.Box 60169, Riyadh 11545, Saudi Arabia. Address reprint requests to : Dr. A. M. AlDosari



Figure 1. Areas of reticular plaque and atrophic lichen planus affecting the hard palate.



Figure 2. Erosive lichen planus affecting the buccal mucosa.

**Results**

Age-range of patients was 16 to 80 years, with the highest prevalence among the 41-50 years old. There was a slight preponderance of females (53%) in comparison to the males (47%). Females were affected almost equally between the ages of 31-40 (16%) and 41-50 (17%) years. Males showed a significantly higher prevalence at the age of 41-50 years (19%) compared to other age ranges. Distribution of age and gender is presented in Table 1.

Table 1. Age and sex distribution of studied patients.

Age Group (Years)	Male		Female		Total	
	No.	(%)	No.	(%)	No.	(%)
< 20	1	0.7	3	2	4	3
21-30	12	8	11	7	23	15
31-40	15	10	24	16	39	26
41-50	29	19	25	17	54	36
51-60	11	7	14	9	25	17
61-70	2	1	1	0.7	3	2
71-80	1	0.7	1	0.7	2	1
<b>Total</b>	<b>71</b>	<b>47</b>	<b>79</b>	<b>53</b>	<b>150</b>	<b>100</b>

Two-hundred and forty clinical lesions were seen in 150 patients (Table 2). In both males and females, the reticular form predominated followed by the erosives, the plaque and the atrophic forms, respectively. Bullous type was rare and seen in only one female patient.

Table 2. Clinical forms of lichen planus in 150 patients.

Clinical Types	No. of Lesions	Percentage of Patients Affected		
		Male	Female	Total
Reticular	135	27	29	56
Erosive	63	11	15	26
Plaque	27	7	4	11
Atrophic	14	3	3	6
Bullous	1	0	0.4	0.4
<b>Total*</b>	<b>240</b>	<b>48</b>	<b>52</b>	<b>100</b>

Some patients exhibited more than one clinical form.

The distribution of lesions in the oral cavity is shown in Table 3. The right and left buccal mucosa were affected almost equally with 123 and 124 lesions, respectively.

Similarly, the upper and lower gingiva were almost equal with 31 and 29 patients affected, respectively. The least commonly affected site was the dorsum of the tongue (5%).

Table 3. Site distribution of lichen planus lesions in the oral cavity.

Site*	Male		Female		Total	
	No.	(%)	No.	(%)	No.	(%)
Buccal Mucosa - Right	56	37	67	45	123	82
Buccal Mucosa - Left	57	38	67	45	124	83
Gingiva - Upper Jaw	20	13	11	7	31	21
Gingiva - Lower Jaw	18	12	11	7	29	19
Tongue (dorsum & border)	28	19	31	21	59	39
Tongue (ventrum)	3	2	5	3	8	5
Palate	7	5	6	4	13	9

\* Involvements of more than one site were present.

The presence of dental restorations and/or appliances is summarized in Table 4. One hundred and two patients (68%) had amalgam restorations, 21(14%) had fixed metal prostheses, while those wearing partial or complete dentures were 27(18%) and 19 (13%), respectively.

Table 4. Associated restorations/prostheses among the studied patients.

Item	No. of Patients*	%
Amalgam	102	68
Partial Denture	27	18
Fixed Bridge	21	14
Complete Denture	19	13
Composite	2	1

\* Some patients have more than one restoration/prosthesis.

Table 5 presents the relationship of local factors to the site of lesions. Amalgam restorations were found to be associated with a significantly high percentage of oral LP lesions on the right buccal mucosa (55%), left buccal mucosa (56%), lateral border and

**Table 5.** Relationship between presence of restoration/prosthesis to the sites of lichen planus lesions

Site	Amalgam (%)	Partial Denture (%)	Complete Denture (%)	Fixed Bridge (%)	Composite (%)
Buccal Mucosa - Right	55	17	12	14	2
Buccal Mucosa - Left	58	17	10	13	2
Gingiva-Upper Jaw	0	22	0	78	0
Gingiva-Lower Jaw	5	21	0	73	0
Tongue (dorsum & border)	63	4	19	14	0
Tongue (ventrum)	41	0	0	40	20
Palate	65	12	0	23	0

\* Involvements of more than one site were present

dorsum of tongue (63%), ventrum of tongue (41%) and palate (65%). On the other hand, lesions of the upper and lower gingivae were associated with fixed metal prostheses, 78% and 73%, respectively.

Systemic findings showed that diabetes mellitus was the most common condition among the studied cases (19%) followed by skin lesions (16%), allergy (13%), hypertension (9%), and history of stress (9%). In fifty one patients (34%) no medical condition were detected or reported (Table 6).

**Table 6.** Systemic findings among patients with oral lichen planus.

Status*	Total No.	Male (*)	Female (%)	Total (%)
Diabetes Mellitus	29	7	12	19
Skin Lesion	24	9	7	16
Allergy	20	7	7	13
Hypertension	14	2	7	9
History of Stress	14	3	7	9
Others**	16	6	5	11
No detected medical conditions	51	14	20	34

\* More than one condition found in some patients.

\*\* e.g. headache, peptic ulcer, kidney disorders, etc.

## Discussion

The current study of 150 cases of oral LP is the largest report to date from a single center in Saudi Arabia. In 1985, Mani<sup>10</sup> reported four cases among 674 dental patients. Salem<sup>11</sup> reported 72 cases in 1989 among a Saudi population of 4,277 individuals.

Findings of the present study in regard to age and gender of patients and the site distribution of oral LP lesions are in agreement with previously reported studies from different parts of the world.<sup>12-15</sup> Among the clinical forms of oral LP in this study, the reticular type was the most common, followed by the erosive, plaque and atrophic types, respectively. One study, however, reported that the erosive type was the most common.<sup>16</sup> This could be explained by the fact that the erosive type presents with associated clinical symptoms, thus, patients are more likely to report for treatment. In our institution the initial examination of all

patients seeking any type of dental treatment is usually carried out by a trained oral diagnosis specialist, thus, the reticular type of oral LP is recognized and referred to the oral medicine clinic.

Several reports linked LP with some systemic conditions, like diabetes, hypertension, stress, liver diseases and others.<sup>17-20</sup> In this study, the general percentage of patients with diabetes mellitus (19%) was found to be higher than the reported incidence of 6.5% and 4.3% among normal individuals.<sup>11</sup> The association of LP in this regard could not be determined due to shortage of information on the prevalence of other systemic conditions among the general population of Saudi Arabia.

The pathogenesis of LP is not fully understood, but recent investigations at both cellular and molecular levels indicate that cell mediated immunity plays a major role in the histopathological changes of LP lesions. Jontell and co-workers reported an increase in mast cells in the subepithelial infiltrate similar to that seen in classic reactions of delayed hypersensitivity. Walsh et al<sup>24</sup> noted changes in the number of Langerhans and mast cells and suggested that oral LP may be initiated by degranulation of mast cells, which induces adhesion of molecules on the endothelium and, subsequently, facilitates lymphocytic infiltrate to the involved tissues. Hypersensitivity reactions to dental materials, such as mercury, ammonium chloride, and nickel sulfate, were noted in a significant number of patients with lichenoid oral mucosal lesions.<sup>25,29</sup>

In this study, the distribution of local factors in relation to the site of LP lesions (Table 5) showed that 78% of the gingival lesions were associated with fixed bridges whereas no cases of palatal lesions were seen in patients with complete dentures. This might give some indication about the importance of local factors in the development of LP lesions, an observation for further investigation.

The malignant potential of lichen planus is still controversial. In an extensive study involving 722 Indian patients, Murti et al<sup>30</sup> observed malignant change in only 0.4% after a follow-up period ranging between three and ten years. This observation was not significantly different from the estimated number in the general Indian population. Holmstrup<sup>31</sup> and co-workers reported a 50-fold increase in malignant development in oral lichen planus cases compared to the general Danish population. Krutchkoff and Eisenberg<sup>32</sup> coined the term lichenoid dysplasia to describe conditions diagnosed clinically as lichen planus but which, histologically, showed some features of epithelial dysplasia. The lichenoid dysplasia concept was supported by Lovas et al who suggested that the apparent malignant transformation of oral lichen planus might likely represent erythroplakia or leukoplakia, which were dysplastic *ab initio*. In the present study, there was no malignant transformation in oral lichen planus in contrast to the study conducted in Gizan region of Saudi Arabia by Salem<sup>11</sup> who reported such malignant transformation in 4 cases of 72 patients after a follow-up period of 3.2 years. Moreover, Gizan region is noted for a relatively high prevalence of oral cancer due to 'Shama' usage. This explains the significant difference between our observation and those reported by Salem.

## References

1. Pindborg JJ, Mehta FS, Daftary DK, Gupta PC, Bhonsle RB. Prevalence of oral lichen planus among 7639 Indian villagers in Kerala, South India. *Acta Derm Venereol (Stockh)* 1972;52:216-20.
2. T. Rundquist L. Oral lichen planus - a demographic study. *Community Dent Oral Epidemiol* 1987;15:52-6.
3. T. A prevalence study of oral mucosal lesions in an adult Swedish population. *Odontol Revy* 1976;27:101-03.
4. Bottomley WK, Brown RS, Lavigne GJ. A retrospective survey of the oral conditions of 981 patients referred to an oral medicine private practice. *J Am Dent Assoc* 1990;120:529-33.
5. 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;61:373-81.
6. Scully C, El-Kom M. Lichen planus: Review and update on pathogenesis. *J Oral Pathol* 1985;14:431-58.
7. Silverman S Jr. Lichen planus. *Curr Opin Dent* 1991;1:769-72.
8. Bricker SL. Oral lichen planus: A review. *Semin Dermatol* 1994;13:87-90.
9. Jungell P. Oral lichen planus. A review. *Int J Oral Maxillofac Surg* 1991;20:129<sup>TM</sup>35.
10. Mani NJ. Preliminary report on prevalence of oral cancer and precancerous lesions among dental patients in Saudi Arabia. *Community Dent Oral Epidemiol* 1985; 13: 247-48.
11. Salem G. Prevalence of oral lichen planus in Gizan, Saudi Arabia. *Community Dent Oral Epidemiol* 1989;17:322-24.
12. Brown RS, Bottomley WK, Puerte E, Lavigne GL. A retrospective evaluation of 193 patients with oral lichen planus. *J Oral Pathol Med* 1993;22:69-72..
13. Thorn JJ, Holmstrup P, Rindum J, Pindborg JJ. Course of various clinical forms of oral lichen planus: a prospective follow-up study of 611 patients. *J Oral Pathol* 1988;17:213-18.
14. Silverman S Jr, Gorsky M, Lozada-Nur F, Giannotti K. A prospective study of findings and management in 214 patients with oral lichen planus. *Oral Surg Oral Med Oral Pathol* 1991;72:665-70.
15. Bagan-Sebastian JV, Milian-Masanet MA, Pe\_arrocha-Diago M, Jimenez Y. A clinical study of 205 patients with oral lichen planus. *J Oral Maxillofac Surg* 1992;50:116-18
16. Tyldesley WR. Oral lichen planus. *Br J Oral Surg* 1974;11:187-89.
17. Bagan JV, Aguirre JM, del Olmo JA, et al. Oral lichen planus and chronic liver disease: A clinical and morphometric study of the oral lesions in relation to transaminase elevation. *Oral Surg Oral Med Oral Pathol* 1994;78:337-42.
18. Strauss RA, Fattore L, Soltani K. The association of mucocutaneous lichen planus and chronic liver disease. *Oral Surg Oral Med Oral Pathol* 1989;68:406-10.
19. Bagan JV, Donat JS, Pearrocha M, Milian MA, Sanchis JM. Oral lichen planus and diabetes mellitus. A clinico-pathological study. *Bull Group Int Rech Sci Stomatol Odontol* 1993;36:3-6.
20. Albrecht M, Banoczy J, Dinya E et al. Occurrence of oral leukoplakia and lichen planus in diabetes mellitus. *J Oral Pathol Med* 1992;21:364-66.
21. Bacchus RA, Bell JL, Madkour M, Kilshaw B. The prevalence of diabetes mellitus in male Saudi Arabs. *Diabetologia* 1982;23:330-32.
22. Fatani HH, Mira SA, El-Zubier AG. Prevalence of diabetes mellitus in rural Saudi Arabia. *Diabetes Care* 1987;10:180-83.
23. Jontell M, Hansson HA, Nygren H. Mast cells in oral lichen planus. *J Oral Pathol* 1986;15:273-75.
24. Walsh LJ, Savage NW, Ishii T, Seymour GJ. Immunopathogenesis of oral lichen planus. *J Oral Pathol Med* 1990;19:389-96.
25. Laine J, Kalimo K, Forsell H, Happonen RP. Resolution of oral lichenoid lesions after replacement of amalgam restorations in patients allergic to mercury compounds. *Br J Dermatol* 1992;126:10-5.
26. Skoglund A, Egelrud T. Hypersensitivity reactions to dental materials in patients with lichenoid oral mucosal lesions and in patients with burning mouth syndrome. *Scand J Dent Res* 1991;99:320-28.
27. Jameson MW, Kardus TB, Kirk EE, Ferguson MM. Mucosal reactions to amalgam restorations. *J Oral Rehabil* 1990;17:293-301.
28. Bolewska J, Hansen HJ, Holmstrup P, Pindborg JJ, Stangerup M. Oral mucosal lesions related to silver amalgam restorations. *Oral Surg Oral Med Oral Path* 1990;70:55-8.
29. Ostman P, Anneroth G, Skoglund A. Amalgam-associated oral lichenoid reactions. Clinical and histologic changes after removal of amalgam fillings. *Oral Surg Oral Med Oral Pathol* 1996;81:459-65.
30. Murti PR, Daftary DK, Bhonsle RB, Gupta PC, Mehta FS, Pindborg JJ. Malignant potential of oral lichen planus: observations in 722 patients from India. *J Oral Pathol* 1986;15:71-7.
31. Holmstrup P, Thorn JJ, Rindum J, Pindborg JJ. Malignant development of lichen planus affected oral mucosa. *J Oral Pathol* 1988;17:219-25.
32. Krutchkoff DJ, Eisenberg E. Lichenoid dysplasia: a distinct histopathologic entity. *Oral Surg Oral Med Oral Pathol* 1985;60:308-15.
33. Lovas JG, Harsanyi BB, ElGeneidy AK. Oral lichenoid dysplasia: a clinico-pathologic analysis. *Oral Surg Oral Med Oral Pathol* 1989;68:57-63