Section: Dentistry

Original Research Article

DENTAL AND PERIODONTAL HEALTH STATUS IN PATIENTS WITH ORAL LICHEN PLANUS

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Abstract

Background: The aim and objective is to assess dental and periodontal health status in patients with oral lichen planus (OLP). **Materials and Methods:** 110 cases of clinically and histopathologically diagnosed OLP patients were included in the study. 110 dental patients without OLP or any other oral mucosal lesions served as controls. All subjects were assessed for dental and periodontal health status by assessing stains, calculus, gingivitis, periodontitis and number of missing teeth due to periodontal pathology. Statistical analysis was done by using; Pearson Chi-square test for stains and calculus, Fisher's exact test and Odds ratio for gingivitis and periodontitis, T test for number of missing teeth. **Result:** There was significant difference in values between two groups in periodontitis and number of missing teeth with P value of 0.010 and0.002 respectively. The difference in values of other parameters were insignificant. **Conclusion:** There was strong association between OLP and, periodontitis andnumber of missing teeth when compared with that of control group.

INTRODUCTION

Lichen planus is a chronic autoimmune, mucocutaneous disease which can affect the oral mucosa, skin, genital mucosa, scalp and nails.[1] Oral lichen planus (OLP) affects 1% to 2% of the general adult population and is more common in women than in men.[2-4] The prevalence of OLP in Indian population is about 2.6%.[5]

OLP presents as asymptomatic or symptomatic lesions. White striations (Wickham's striae, reticular type), white papules (papular), white plaques (plaque-like) varieties are usually asymptomatic and erythema (atrophic), erosions (erosive), blisters (bullous) varieties are usually symptomatic. OLP affects the buccal mucosa more commonly although tongue, gingiva, palate, and lips may be affected.[6,7] Symptomatic types cause discomfort and soreness and may make eating and practicing good oral hygiene difficult for affected patients.[6-8]

Although classic white lesions of OLP may be diagnosed correctly, an oral biopsy with histopathological examination is recommended to confirm the clinical diagnosis and which also helps to exclude dysplasia and malignancy.

When there is poor clinicopathological correlation it may be helpful to use direct immunofluorescence, which shows a linear pattern of fibrin and shaggy fibrinogen deposits at the epithelial basement membrane or cytoid bodies (Russell bodies), or both, in the absence of deposition of fibrinogen.[9]

It has been suggested that gingival lesions of OLP could play a role in increasing the long risk for periodontal tissue breakdown at specific sites.[10]

A study demonstrated significant differences in bacterial colonization patterns between sites with confirmed OLP and healthy control sites within the same subject.[11] Hence in long standing periodontal condition, OLP may worsen in these patients.

Aim of this study was to assess dental and periodontal health status in patients with oral lichen planus and compare it with that of healthy controls. The association between periodontitis and oral lichen planus was also assessed.

MATERIALS AND METHODS

Study Group: 110 cases of clinically and histopathologically diagnosed OLP patients, who had visited the Department of Oral Medicine and Radiology, S.D.M.College of Dental Sciences And Hospital, Dharwad were included in the study.

Control Group: Dental patients without OLP or any other oral mucosal lesions served as controls.

Inclusion Criteria

Subjects who had no periodontal management in the six preceding months,had no previous treatment for

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OLP and having minimum of 10 teeth were included in the study.

Exclusion Criteria

Subjects who are on medications which are likely to cause or exacerbate OLP, like penicillin, sulfonamide, NSAIDs, oral contraceptives etc were excluded and Edentulous patients were excluded from the study.

Dental and periodontal health status was assessed in terms of stains, calculus, gingivitis, periodontitis and number of missing teeth due to periodontal etiology in both the groups.

Stains and Calculus: were assessed and scored from score1 to 3. Stains and calculus covering upto 1/3rd of crown as score 1, upto 2/3rd of crown as score 2 and > 2/3rd of crown as score 3.

Gingivitis: was assessed with the signs of gingival inflammation including redness, swelling and/or bleeding on probing without any sign of attachment loss, such as pockets, gingival recessionand bone loss12 and was recorded as absence or presence of gingivitis.

Periodontitis: Presence of Clinical attachment loss > 2 mm on more than one tooth, or when mobility of more than one tooth12 was considered as periodontitis and was recorded as absence or presence of periodontitis.

Number of missing teeth in each subject with periodontitis was scored.

Statistical analysis was done by using: Pearson Chi-square test for stains and calculus, Fisher's exact test and Odds ratio for gingivitis and periodontitis, T test for number of missing teeth.

RESULTS

Demographic details; Study and control both the groups had 110 subjects each. Study group subjects had the age range of 17 to 73years with mean of 46.92 years and control subjects had the age range of 20 to 70 years with mean of 42.20 years [Table 1].

60 subjects were females and 50 subjects were males in the study group, 54 subjects were females and 56 subjects were males in the control group [Table 2]. No significant difference was observed between groups in relation to age and gender (p>0.05)

In frequency distribution of scores of stains and calculus difference was insignificant on comparison of scores in both the groups with p value 0.667 and 0.162 respectively [Table 3 and Table 4].

Comparison of gingivitis and periodontitis; When we compared gingivitis, 95 subjects in study group and 91 subjects in control group had gingivitis. But difference was not significant with P value 0.456. Whereas, 63 of study group and 44 of control group subjects had periodontitis. There was significant difference in values between two groups, with P value of 0.010. That means there was 49% more chance of getting OLP in patients with periodontitis [Table 5].

Comparison of number of missing teeth: When we compared number of missing teeth, mean score was 3.34 in study group, 1.63 in control group. There was significant difference in values between two groups, with P value of 0.002 [Table 6].

Table 1: Demographic details.

	Groups	N	Age range	Mean	Std. Deviation
Age	Study group	110	17 to 73yrs	46.92	14.644
	Control Group	110	20 to 70 yrs	42.20	13.088

Table 2: Demographic details

		Study Group N (%)	Control Group N (%)
Sex	Female	60(54.5%)	54(49.1%)
	Male	50(45.5%)	56(50.9%)
Total		110	110

Table 3: Comparison of scores of stains in both the groups P = 0.667

		Study group		Control group	
Stains		**Frequency**	**Percent**	**Frequency**	**Percent**
	Score 1	57	51.8	55	50.0
	Score 2	44	40.0	42	38.2
	Score 3	9	8.2	13	11.8
	Total	110	100	110	100

Table 4: Comparison of scores of calculus in both the groups, P = 0.162

		Study group		Control group	
Calculus		**Frequency**	**Percent**	**Frequency**	**Percent**
	Score 1	45	40.9	62	56.4
	Score 2	58	52.7	43	39.1
	Score 3	7	6.4	5	4.5
	Total	110	100	110	100

Table 5: Comparison of gingivitis and periodontitis

		Gingivitis		Periodontitis	
		No	**Yes**	**No**	**Yes**
Groups	Study	15(13.6%)	95(86.4%)	47(42%)	63(57%)
	Control	19(17%)	91(82%)	66(60%)	44(40%)

P=0.456P=0.010

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Odds Ratio = 0.49

Table 6: Comparison of number of missing teeth P = 0.002

	Study group		Control group	
No.of missing teeth	**Minimum**	**Maximum**	**Minimum**	**Maximum**
	0	17	0	12
Mean	3.34		1.63	
Std Deviation	4.66		3.09	

DISCUSSION

Ramon Fluixa et al assessed periodontal disease indices in 90 OLP patients and 52 subjects of control group and found significant association between increased plaque and calculus deposites and atrophic-erosive lesions of OLP and also observed increase in the lesion extent. But there was no significant difference between OLP patients and control group in relation to periodontal indices.[13]

Ergun et al. studied 22 patients with OLP and 20 healthy subjects, and revealed mild or moderate periodontal disease. None of the subjects had severe periodontitis.[14]

Pia Lopez-Jornet et al in another study, assessed Gingival Index, Plaque Index and CPITN Index in patients with oral lichen planus and compared with that of control group. They concluded that periodontal condition of the oral lichen planus patients was significantly worse than in the control group.[15]

Michael Bornstein et al demonstrated significant differences in bacterial colonization patterns between healthy site and site with confirmed OLP within the same subject and also sites in healthy subject of the control group without OLP. Microbiologic differences were found between sites with OLP and sites in subjects without a diagnosis of OLP. Specifically, higher counts of staphylococci and S. agalactiae were found in OLP lesions.[16]

In another study the amounts of periodontopathogens in OLP patients were found to be higher in comparison to non-OLP patients with periodontits.[17]

In our study, the periodontal health of OLP patients was significantly worse than in the control group. This may be attributed to the fact that pain or burning sensation in OLP patients prevents maintainance of good oral hygiene which leads to plaque and calculus accumulation increasing the long term risk of periodontal disease which in turn exacerbate OLP lesions.

Different microbial species in plaque and calculus like Bacteroides, Dialister, Neisseria, staphylococci, and beta-hemolytical streptococci species which are linked to skin and soft tissue lesions at locations other than the oral cavity, may also play a role in the complex pathogenesis of OLP.[16]

Further pathologic analysis of the lesions (e.g.,electron microscopy), can give additional information about invasion of bacteria into the tissue.

It has been mentioned in a review article that Infections and autoimmune diseases have multidirectional and multifaceted relationships.[18]

Pathogens can initiate or perpetuate autoimmunity through several mechanisms. Antigen-specific mechanisms include molecular mimicry, expression of modified, cryptic, or new antigenic determinants, and superantigens. Other nonspecific mechanisms collectively known as “bystander activation” include enhanced processing and presentation of self-antigens, immune cell activation, cytokine release, and cell apoptosis/necrosis.[19] So, long standing periodontal infection can alter mucosal immunity towards autoimmunity, thus making mucosa more prone to develop OLP. In our study, significant difference was observed between study and control group in relation to periodontitis. But as periodontal condition and OLP both were assessed at the same time, we can’t say whether both initiated at the same time or one lesion initiated the other. Hence we have to think whether periodontitis can exacerbate OLP or it can initiate OLP or vice versa.

CONCLUSION

There was strong association between OLP and, periodontitis andnumber of missing teeth when compared with that of control group.

Oral lichen planus and periodontitis both were evaluated at the same time. So it was not possible to determine which condition started first.

But higher counts of periodontopathogens can have role in the progression of OLP.

Studies should be conducted to assess the role of severe periodontitis in the development and/or progression of oral lichen planus and also OLP with a longer duration to assess the effects of lesion on periodontal health.

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