

Oral Leukoplakia in Non-Smoking, Non-Chewing, Non-Alcoholic patients: A retrospective study

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Abstract

Aim: to find the prevalence and characteristic features of oral leukoplakia in patients without any habits such as smoking, chewing or alcoholism

Materials & method: the records in the department of Oral Medicine & Radiology, Government Dental College, Kozhikode, was searched for cases of leukoplakia, during a period May 2015 to April 2016. Details such as gender, age, history of habits, type and site of leukoplakia, degree of dysplasia were recorded in a poforma.

Results: among 62 leukoplakia patients, 10 cases were in patients without any habits. The no-habit group had equal gender distribution. Most of the no-habit group patients were below 50 years whereas most of the habit group were above 50 years ($P < 0.05$). The common site among the habit group was buccal mucosa, followed by dorsal tongue, whereas the most common site among no-habit group was lateral and ventral tongue ($P < 0.05$). Though all lesions in no-habit group were of homogenous type, histologically most lesions were having moderate to severe dysplasia ($P > 0.05$)

Summary: leukoplakia in patients without any tobacco or alcohol use, are having unique features. Such lesions require aggressive management and strict follow up, as these cases are having higher risk of malignancy.

Key words: Leukoplakia, tobacco, smoking, alcohol

Date of Submission: 23-05-2018

Date of acceptance: 05-06-2018

I. Introduction

Oral leukoplakia has been defined as "A predominantly white patch or plaque that cannot be characterized clinically or pathologically as any other disorder; oral leukoplakia carries an increased risk of cancer development either in or close to the area of leukoplakia or elsewhere in the oral cavity or the head and neck region".¹ The prevalence of leukoplakia was found to be 4.11% in a recent meta analysis by Mello et al.² Though the most common etiologic agent in leukoplakia is tobacco, followed by alcohol³, there are published reports of leukoplakia and malignancy arising in individuals without any history of tobacco or alcohol use.⁴ Hence a study was planned to assess oral leukoplakia in individuals without any history of tobacco or alcohol use.

II. Aim Of The Study

To find out the prevalence and characteristic features of oral leukoplakia in patients without any history of habits such as smoking, chewing or alcoholism.

III. Materials And Method

Study design

Retrospective descriptive study

Study duration

1 year, from May 2015 to April 2016

Study population

Patients reported to department of Oral Medicine & Radiology, Government Dental College, Kozhikode during the above period and diagnosed as having oral leukoplakia

Study setting

Government Dental College, Kozhikode, a tertiary referral centre in Kerala State, India

Inclusion criteria

Clinically and histologically diagnosed cases of Oral Leukoplakia

Exclusion criteria

1. Incomplete details
 2. Genetic disorders with high incidence of oral precancers such as dyskeratosis congenita
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Study procedure

The records were searched for new cases of oral leukoplakia reported between May 2015 and April 2016. Total number of new patients above 12 years, reported to OP during the same period was also noted. Details of patients such as age, gender, details about use of tobacco, areca nut and alcohol, history or clinical evidence of chronic trauma, site and type of leukoplakia and degree of dysplasia were recorded.

Data collection

The data collected from the records were entered into a proforma made for the study

Data analysis

Patients were divided into two major groups based on the presence or absence of habits. Statistical analysis was done using SPSS for Microsoft Windows. For comparison of qualitative data between two groups, the chi-square test was used. P value of less than 0.05 was considered significant and less than 0.001 was considered highly significant.

IV. Results

Among 46,710 new patients reported to the institution during the above period, there were 62 cases of oral leukoplakia (Prevalence 0.13%). Out of these 62 leukoplakia patients, 10 patients (prevalence of 0.021%) did not have any history of use of tobacco, areca nut or alcohol.

Comparison of groups with and without habits

1. Gender

Table 1 shows the comparison of both groups on the basis of gender. There was a definite male predilection for the habit group, but there was equal gender distribution in the no-habit group. The difference was not statistically significant (P 0.17)

Table 1: Comparison of both groups based on gender

	Male N (%)	Female N (%)	Chi-square value	P value
With habit	38 (71.7)	15 (28.3)	1.89	0.17
Without habits	5 (50.0)	5 (50.0)		

2. Age

Table 2 shows the comparison of distribution of age between the habit group and no-habit group. Majority of the patients in the habit group were above 50 years (mean age: 57.09 ±14 yrs) whereas most of the patients in the no-habit group were below 50 years (mean age: 46.1 ±14.06 yrs). The difference in the age distribution was found to be statistically significant (P < 0.05)

Table 2: Comparison based on Age

Age group (years)	With habit N (%)	Without habits N (%)	Chi-square value	P value
<30	0	1 (10)	11.21	0.01
30-50	18 (34)	7 (70)		
51-70	25 (47.2)	1 (10)		
>70	10 (18.9)	1 (10)		
Total	53 (100)	10 (100)		

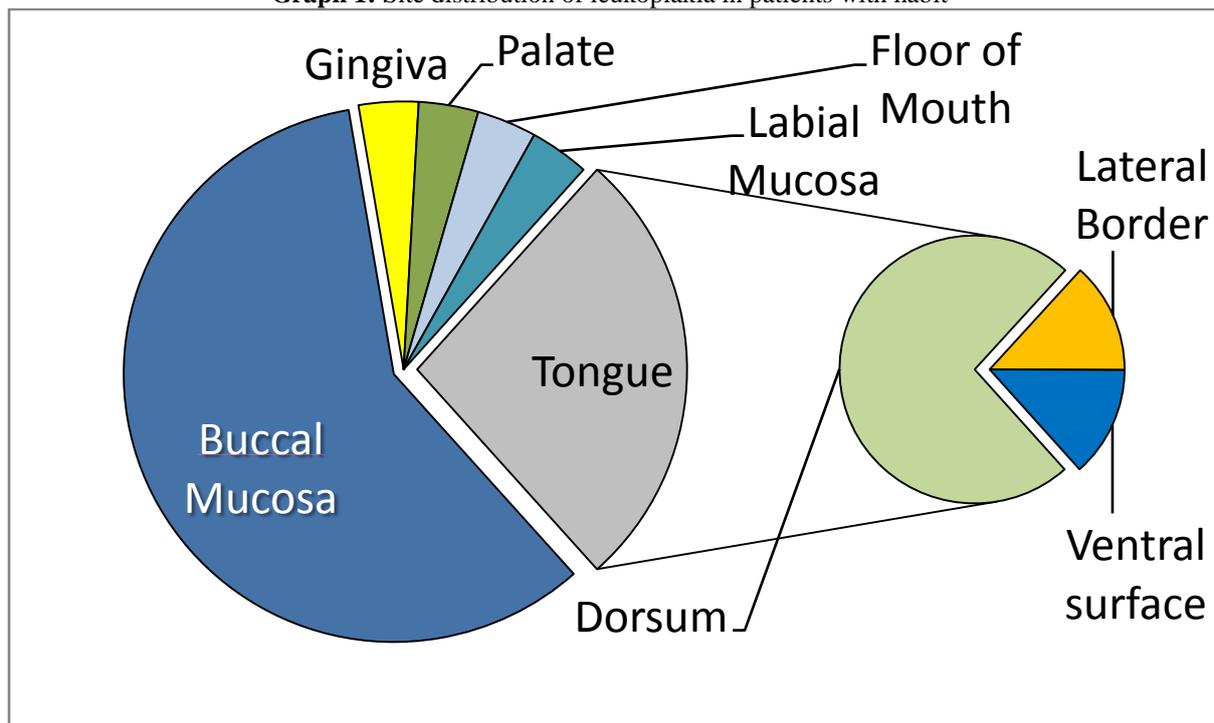
3. Site of leukoplakia

Table 3 and Graphs 1 & 2 show the difference in the site distribution between patients with and without habits. In patients with habits, most of the lesions were in buccal mucosa (58.9%) whereas the most common site among no-habit group was tongue (58.3%). Among tongue lesions in patients with habits, most common site was dorsum, whereas in no-habit group, no patients had lesions on dorsum, but lateral and ventral tongue involvement was common. The difference in site distribution between the two groups was found to be statistically significant (P < 0.05).

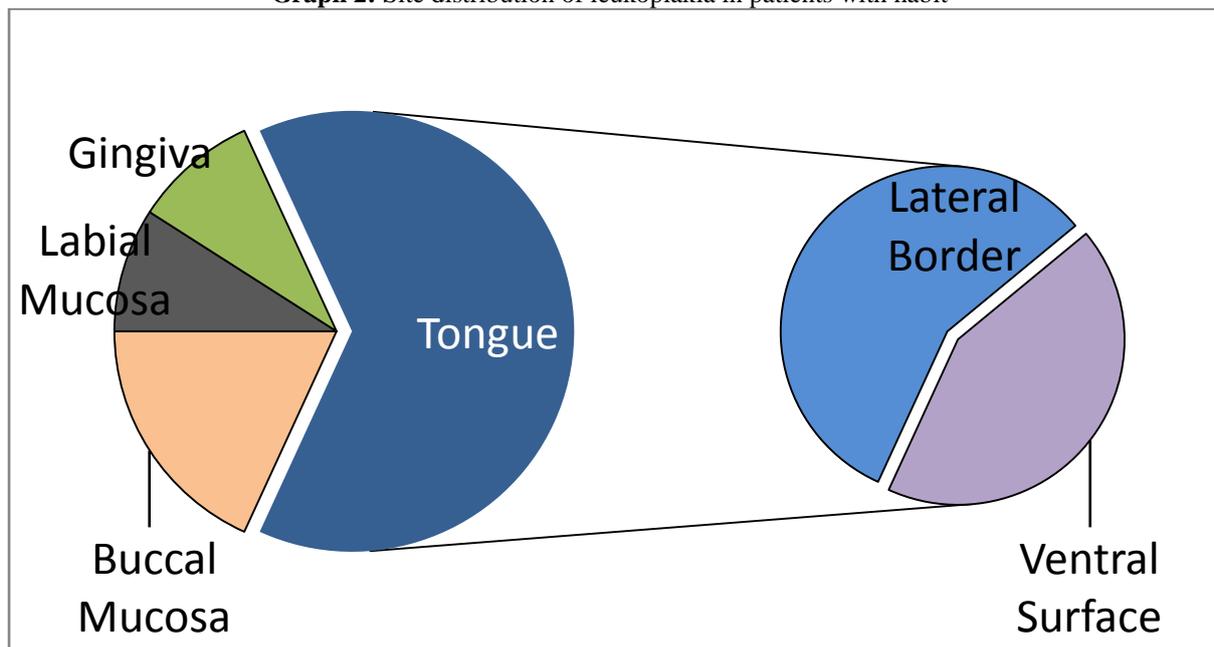
Table 3: Comparison of groups based on site of leukoplakia

Site	With habit N (%)	Without habits N (%)	Chi-square value	P value
Labial Mucosa	2 (3.6)	1 (8.3)	4.38	0.002*
Buccal Mucosa	33 (58.9)	3 (25)		
Palate	2 (3.6)	0		
Tongue dorsum	11 (19.6)	0		
Lateral tongue	2 (3.6)	4 (33.3)		
Ventral tongue	2 (3.6)	3 (25)		
Floor of mouth	2 (3.6)	0		
Gingiva	2 (3.6)	1 (8.3)		

Graph 1: Site distribution of leukoplakia in patients with habit



Graph 2: Site distribution of leukoplakia in patients with habit



4. Type of leukoplakia

Among 52 leukoplakia lesions in patients with habit, 42 were homogenous type and 10 were non homogenous type. However, all lesions among no-habit group were found to be of homogenous type. But the difference was not statistically significant ($P = 0.284$)

5. Degree of dysplasia

Table 4 shows the distribution of lesions based on the degree of dysplasia. Majority of the lesions in habit group had only mild dysplasia (58.5%), whereas 80% of no-habit group had moderate to severe dysplasia. The difference in the degree of dysplasia was not statistically significant ($P = 0.07$)

Table 4: comparison of degree of dysplasia

Degree of dysplasia	With habit N (%)	Without habits N (%)	Chi-square value	P value
Mild	31 (58.5)	2 (20.0)	5.215	0.07
Moderate	18 (34.0)	6 (60)		
Severe	4 (7.5)	2 (20.0)		

V. Discussion

The present study found that 10 among 62 leukoplakia patients (16%) did not have any history of habits: smoking, chewing or alcoholism. Even though there are a number of etiologic factors suggested for the development of oral squamous cell carcinoma (OSCC), including various oral habits, the major known etiological factors associated with leukoplakia are tobacco and alcohol.³ Genetic changes have been found to be occurring in leukoplakia⁵, but it is not clear whether it is the primary cause or secondary to tobacco use. Though there are a number of studies mentioning a causative role for HPV in OSCC, a recent study found no correlation between leukoplakia and HPV.⁶

The leukoplakia patients in the no-habit group had unique features when compared to those with habits. There was equal gender distribution in leukoplakia cases without habits. Females without habits have been found to be having increased risk of development of malignancy.⁴ The no-habit group had significantly lower age range than the habit group. Though the risk of malignancy is directly associated with increasing age, persistence of lesions for longer duration in patients without any habit, subjected to the same unknown etiological agent can increase the chances of development of malignancy in such patients.

Tongue was the most common site of involvement in no-habit group, especially lateral and ventral tongue. Lateral tongue lesions are known to be having higher risk for turning malignant.⁷ Even though all the lesions in no-habit group were homogenous leukoplakia, histologically most lesions had moderate to severe epithelial dysplasia, necessitating aggressive management and strict follow up.

All these findings could be correlated to the higher risk of malignancy in leukoplakia cases without any history of smoking, chewing or alcoholism, as suggested in the literature.⁸

VI. Summary

Though oral leukoplakia is commonly associated with tobacco and alcohol use, patients without any of these habits can at times present with leukoplakia. Such lesions have unique characteristics, occurring in younger individuals and tongue being the common site. Since the exact etiology in such cases is not known, and higher rates of malignancy have been found in such lesions, especially in females, aggressive management and thorough follow up is necessary to ensure prevention of development of malignancy in cases of leukoplakia in non tobacco, non alcohol users.

Acknowledgement

The author would like to acknowledge the help offered by Dr. Arun Paul, Reader, Community Dentistry, KMCT Dental College, Kozhikode, in doing the statistical analyses

Reference

- [1]. van der Waal I. Oral leukoplakia, the ongoing discussion on definition and terminology. *Med Oral Patol Oral Cir Bucal*. 2015 Nov 1; 20(6): e685-92.
- [2]. Mello FW, Miguel AFP, Dutra KL, Porporatti AL, Warnakulasuriya S, Guerra ENS, Rivero ERC. Prevalence of oral potentially malignant disorders: a systematic review and meta-analysis. *J Oral Pathol Med*. 2018 May 8. doi: 10.1111/jop.12726. [Epub ahead of print]
- [3]. Parlatescu I, Gheorghe C, Coculescu E, Tovu S. Oral leukoplakia – An update. *Maedica – J Clin Med*. 2014; 9(1): 88-93
- [4]. Qasrdashti AB, Habashi MS, Aresteh P, Ardakani MT, Abdoli Z, Eghbali SS. Malignant transformation in leukoplakia and its associated factors in Southern Iran: A hospital based experience. *Iran J Pub Health*. Aug 2017; 46(8): pp1110-1117
- [5]. Singla S, Singla G, Zaheer S, Rawat DS, Mandal AK. Expression of p53, epidermal growth factor receptor, c-erbB2 in oral leukoplakias and oral squamous cell carcinomas. *J Can Res Ther* 2018;14:388-93.
- [6]. Bhargava A, Shakeel M, Srivastava AN, Raza TS, Rizvi S, Varshney P. Role of human papilloma virus in oral leukoplakia. *Indian J Cancer* 2016; 53: 206-9.
- [7]. Waldron CA, Shafer WG. Leukoplakia revisited. A clinicopathologic study 3256 oral leukoplakias. *Cancer* 1975; 36(4): 1386-92.
- [8]. van der Waal I. Potentially malignant disorders of the oral and oropharyngeal mucosa; terminology, classification and present concepts of management. *Oral Oncol* 2009;45(4-5):317-23.

Padippurakkakath Salim Haris "Oral Leukoplakia in Non-Smoking, Non-Chewing, Non-Alcoholic patients: A retrospective study." *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)*, vol. 17, no. 6, 2018, pp 52-55.