

Original Research Article

Prevalence and Distribution of Oral Leukoplakia in Patients Attending Oral Medicine Department at Dentistry college in Tishreen University

Abstract

BACKGROUND: To find out the prevalence and distribution of oral leukoplakia in patients who are visiting the Department of Oral Medicine at Tishreen University is necessary to assess oral health and identify the risks of malignant transformation.

OBJECTIVES: The aim of this study was to find out the prevalence and distribution of oral leukoplakia in the patients who visited the Department of Oral Medicine at Tishreen University.

MATERIALS AND METHODS: The study was conducted on 500 patients of the Tishreen Oral Medicine Department at Tishreen University. The number of males was 348 and females 152. The number of who drink alcohol was 117 and non-alcoholic 383. The number of smokers was 279 and non-smokers 221. The average age of the sample was 52 years.

RESALTS: We found that the percentage of leukoplakia in the sample was 2.8%. There was a statistically significant correlation between leukoplakia and smoking, drinking alcohol, increase in age and sex, and no relation was found with general diseases.

CONCLUSIONS: Increased incidence and associated risk factors (smoking, drinking alcohol, increasing age and sex) require dentists to carefully examine Oral mucosa for early detection of precancerous changes and therefore early treatment.

Leukoplakia, smoking, drinking alcohol

Introduction :

Leukoplakia is "The lesion is often a white lesion on the mucous membrane of the mouth that can not be classified as any other disease". [1] As such, it is not a specific disease in itself, where there is a clinical similarity and variable tissue manifestations. [2] Which are strongly attached to the mucosa and associated with an increased risk of cancer [4,3], the lesion has clear and

Comment [U1]: Spelling error needs to be corrected

32 variable edges over time [5,3]. Advanced models have developed red spots and
33 there are no other symptoms. [5] Mucosa sometimes though to other parts of
34 the gastrointestinal tract or urinary tract Genitals may be affected [6,7,8].

35 leukoplakia is a descriptive term that should be launched only after excluding
36 other possible causes. The cause of the episode is not known but the risk factors
37 include smoking chewing tobacco, excessive drinking alcohol, viruses and chronic
38 irritation and the use of nuts [9,3]. It is a pre-cancerous lesion where tissue
39 biopsy generally shows an increase in correlations with or without abnormal cells
40 [5,3] and is mixed with lichen planus , hyperkeratosis, and white candidiasis [3].

41 Treatment recommendations depend on the clinical appearance and histological
42 examination of the lesion. When abnormal cells are present, simple surgical
43 removal is one possible solution. In other cases, monitoring for periods of three
44 to six months may be sufficient. [3] People are advised to stop smoking and
45 reduce alcohol intake. [3] In half the cases, When smoking continues, 66% of
46 cases Increase thick and white. [5] These cases are more common with age and
47 usually do not occur until after 30. [3] Rates may be as high as 8% in men over
48 the age of 70. Several studies have been conducted on their prevalence and risk
49 factors in several studies in different communities to determine the risk rate,
50 which increases the predictability and ease of treatment

51

52 **materials and methods :**

53 – The sample consists of 500 patients who visite the Department of Oral
54 Medicine at the Faculty of Dentistry at Tishreen University, who are over 16
55 years of age. The number of males is 348 and females 152, and 14 cases have
56 been diagnosed as leukoplakia.

Comment [U2]: Aracanurt has been associated with oral submucous fibrosis. It would be beneficial if the authors could mention what nuts are mentioned here

- 57 – The number of smokers 279 and non-smokers 221.
- 58 – The number of who drink alcohol was 117 and non-alcoholic 383.
- 59 – The number of people with systemic diseases 101 and the number of non-
60 infected 399.
- 61 – The average ages were 52 years, while the age of those infected was between
62 49–62 years.
- 63 – A research form was designed in which the researcher recorded the patient's
64 personal information (age and gender), Smoking (intensity, duration), Drinking
65 Alcohol (Quantity, Frequency, Duration).
- 66 – The existence of general diseases through the use of indirect and directed
67 questions.
- 68 – WHO standards for clinical diagnosis to leukoplakia were adopted.
- 69 – The statistical SPSS program was used to analyze the results.

70

71

72 3. Results:

73 3.1 Prevalence leukoplakia of the sample:

Statistics		
leukoplakia		
N	Valid	500
Mean		.028
Std. Deviation		.165
Sum		14

74

Table (3.1) Some descriptive statistics for the rate of leukoplakia

75 The previous table shows some metadata for the variable variable. The sample
 76 in question was 500. The number of individuals who had leukoplakia 14 was
 77 2.8% and a standard deviation 0.16.

78 **3.2 Relationship between leukoplakia and age:**

79 The average ages were 52 years, while the age of those infected was
 80 between 49 – 62. To test whether there was a relationship between leukoplakia
 81 and the age, we used the Point Biserial Correlation Coefficient, which is
 82 expressed in Pearson Correlation, next one:

83

Correlations

		leukoplakia	age
leukoplakia	Pearson Correlation	1	.115 [*]
	Sig. (2-tailed)		.010
	N	500	500
age	Pearson Correlation	.115 [*]	1
	Sig. (2-tailed)	.010	
	N	500	500

84 Table (3.2) Correlation is significant at the 0.05 level (2-tailed).
 85 Note from the table that the correlation coefficient value is 0.115. The correlation
 86 is linear in the sense that the longer the age, the greater the probability of a
 87 coating. Although this coefficient is relatively small, the correlation is significant or
 88 significant at the significance level of 0.05 (Sig = 0.01 <0.05).

89 **3.3 Relationship between leukoplakia and sex:**

90 The number of males who do not have a diploma is 334 and the number
 91 of females is 152. However, it should be noted that those who have a class are
 92 male only.

93 To study the relationship between sex and class, a correlation coefficient can be
 94 used.

95 * Crosstabulation sex

Count

		sex		Total
		male	female	
leukoplakia	Non-leukoplakia	334	152	486
	leukoplakia	14	0	14
Total		348	152	500

96 Table (3.3): Some descriptive statistics on the rate of sex-related relationship

97

Symmetric Measures

	Value	Approximate Significance
Phi	.112	.012
N of Valid Cases	500	

98 Table (3.4) Relationship between leukoplakia and sex using Phi coefficient

99 Note from the above table that the value of the Fay correlation coefficient is
 100 0.112, ie the correlation is positive or negative, which is a significant correlation
 101 (Sig = 0.012 <0.05).

102 **3.4 The relationship between leukoplakia and smoking:**

103 leukoplakia was distributed as follows:

104 There are two non-smokers and twelve smokers .

105 To study the relationship between smoking and class, a correlation coefficient
 106 was used.

107 * Smoking Crosstabulation

	Total	
	Non-smokers	smokers

Leukoplakia	Non-leukoplakia	219	267	486
	leukoplakia	2	12	14
Total		221	279	500

Table (3.5): The relationship between leukoplakia and smoking

108

109

Symmetric Measures

	Value	Approximate Significance
Phi	.154	.008
Cramer's V	.154	.008
N of Valid Cases	500	

110 Table (3.6): The relationship between leukoplakia and smoking using the laboratory Fay

111 Note from the above table that the value of the coefficient of Fay correlation is

112 0.154, ie the correlation is positive or negative, which is a significant correlation

113 (Sig = 0.008 < 0.05). In the sense that those who are increasing their smoking

114 are more likely to have a class.

115 3.5 The relationship between leukoplakia and drinking alcohol:

116

117 Six people in the sample do not drink alcohol and eight drink . To study the

118 relationship between drinking alcohol and leukoplakia can be used Chi-Square

119 test.

120 * Alcohol consumption Crosstabulation

121

		Total		
		Non-alcoholic	alcoholic	
leukoplakia	Non-leukoplakia	377	109	486
	Leukoplakia	6	8	14
Total		383	117	500

Table (3.7) Relation between drinking alcohol and leukoplakia

122

Chi-Square Tests

	Value	df	Asymptotic Significance (2- sided)
Pearson Chi-Square	14.890 ^a	2	.001
Likelihood Ratio	9.971	2	.007
Linear-by-Linear Association	13.800	1	.000
N of Valid Cases	500		

Table (3.8) Relation between drinking alcohol and leukoplakia using Chi-Square Test

124 From the table above, the value of the Chi-Square test index is 14.89 and the
 125 test is significant at the significance level of 0.05 (Sig = 0.001 < 0.05). That is,
 126 there is a link between drinking alcohol and leukoplakia.

127

128 3.6 The relationship between leukoplakia and systemic diseases:

129 The distribution leukoplakia among sample is as follows: Thirteen
 130 people are without systemic diseases and only one is with. To study the
 131 relationship between leukoplakia and systemic diseases, the correlation
 132 coefficients can be used coefficient of Phi

Crosstabulation

Count		Systemic diseases		Total
		without	with	
leukoplakia	Non-leukoplakia	386	100	486
	leukoplakia	13	1	14
Total		399	101	500

133

(3.9)

134

135

136

Symmetric Measures

	Value	Approximate Significance
Phi	-.055-	.220
N of Valid Cases	500	

137

(3.10)

138 Note from the above table that the value of the coefficient of Phi correlation is
139 equal to 0.055, ie, the correlation is weak and is insignificant at the level of 0.05
140 (Sig = 0.22 > 0.05). It is not clear that those who have leukoplakia have a
141 disease.

142 **4. Discussion:**

143 The prevalence of our study was 2.8% while the prevalence was
144 0.9% in a study by Reichart et al [10] While a high proportion was observed in
145 Zhang et al. 9.18% [11] and 9.3% in a study conducted by Kumars et al [12]
146 among the tribal population of Kundam province. In a study by Granero et al [13]
147 in Mallorca it was 5,1%and It was 22% at Patil s et al [14].

148 The difference between prevalence rates in different studies is explained by a
149 number of factors, including: sample size, the nature of the studied society,
150 common habits (smoking and drinking alcohol) and the age of the studied
151 sample, where we see a significant increase in prevalence in studies conducted
152 on older persons. [14] The nature and climate of the region may also play a
153 role.[12]

154 We found in our study that there was a positive correlation between the
155 prevalence of the leukoplakia and the increase in age. Reichart et al. [15] agreed
156 with us because he studied the German elders to a similar result while R
157 Chandran et al. [16] Kassab et al [17] disagreed with us in a study conducted at
158 the Lebanese University found no difference in the distribution of oral lesions
159 among age groups. Several studies [15] [10] have found a positive correlation
160 between age and leukoplakia. This may be explained by histological changes
161 that occur with increase in age, as well as by prolonged use of oral habits
162 (smoking, drinking alcohol).

163 In our study, we found that only males were affected by leukoplakia, indicating
164 their association with sex. PA Reichart et al. [10] agreed that males are more
165 affected than females 1.6% to 0.2% .

166 A study conducted in Budapest by J.Banoczy et al. [10] Where the ratio of
167 males to females was 3.2% to 1% and It also reached the same conclusion
168 Sujathy et al.[19] and Patil S et al. [14] In a study carried out by Cebeci Ar et
169 al. in Ankara [52], the number of men was four times greater than that of
170 women. These results may explain the different oral habits of the sexes (smoking
171 and drinking alcohol) and may be the cause of occupational stress [21] and sex
172 there are no studies to prove a direct relationship.

173 We found a positive correlation between prevalence of leukoplakia and smoking
174 and this is consistent with many studies Femopase FI et al. [22] Gary et al.[23]
175 Saraswathi et al. [24] Zhang et al. [11] and Mathewall et al [25].The agreement
176 between studies on the presence of such a relationship may explain the effect of
177 nicotine on the oral mucous and the changes it causes in mucous membranes.

178 We found that there was a positive correlation between drinkink alcohol and
179 leukoplakia, and we agreed with that, Zhang et al. [11], Saraswathi et al [26],
180 and Sujathd et al.[19] and Rooban et al.[27]

181 While Cebeci Ar et al. [20] did not find a relationship between drinking alcohol
182 and the risk of developing oral lesions. Explanation of the effect of drinking
183 alcohol on the oral mucous where the excessive use of high alcohol, which
184 contains (more than 25%) to the presence of gray board [28].

185 In our study, there was no relationship between the prevalence of leukoplakia
186 and the presence of systemic diseases , Agreed with us Cebeci Ar et al. [20]
187 This may be due to the low age of the sample and the nature of the studied
188 society while Reichart et al. [15] disagreed with us This may be because he
189 studied German elders with a high proportion of systemic diseases as a result of
190 age.

191

192 **Conclusions:**

193 The prevalence and distribution of oral leukoplakia were influenced by
194 a range of factors (smoking, drinking alcohol, sex and age), but no association

Comment [U3]: Other authors such as Madiyal et al have also found similar results. The manuscript may benefit from the mention of similar studies conducted recently- Status of thiocyanate levels in the serum and saliva of non-smokers, ex-smokers and smokers. Ananya Madiyal, Vidya Ajila, Subhas G Babu, Shruthi Hegde, Suchetha Kumari, Medhini Madi, Sonika Achalli, Priyadharshini Alva, Harshini Ullal. African Health Sciences. Vol 18, No 3 (2018)

Comment [U4]: Spelling error needs to be corrected

195 was found with systemic disease, which should prompt dentists to examine the
196 oral mucous in the most high risk factor groups for early detection of pre-
197 cancerous lesions .
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Reference

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- 200 1. Neville BW; Damm DD; Allen CM; Bouquot JE. (2002). Oral &
201 maxillofacial pathology (2. ed.). Philadelphia: W.B. Saunders. pp. 337–345.
202 ISBN 0-7216-9003 .
- 203 2. Greenberg MS, Glick M (2003). Burket's oral medicine diagnosis & treatment
204 (10th ed.). Hamilton, Ont.: BC Decker. pp. 87,88,90–93,101–105. ISBN 1-
205 55009-186-7 .
- 206 3. Villa, A; Woo, SB (26 October 2016). "Leukoplakia–A Diagnostic and
207 Management Algorithm". Journal of Oral and Maxillofacial Surgery. 75: 723–
208 734. doi:10.1016/j.joms.2016.10.012. PMID 27865803 .
- 209 4. Scully, C; Porter, S (Jul 22, 2000). "ABC of oral health. Swellings and red,
210 white, and pigmented lesions". BMJ (Clinical research ed.). 321 (7255):
211 225–8. doi:10.1136/bmj.321.7255.225. PMC 1118223 . PMID 10903660 .
- 212 5. Neville, Brad W.; Damm, Douglas D.; Chi, Angela C.; Allen, Carl M.
213 (2015). Oral and Maxillofacial Pathology (4 ed.). Elsevier Health Sciences.
214 pp. 355–358. ISBN 9781455770526. Archived from the original on 2017-
215 09-10 .
- 216 6. Banfalvi, Gaspar (2013). Homeostasis – Tumor – Metastasis. Springer
217 Science & Business Media. p. 156. ISBN 9789400773356. Archived from
218 the original on 2017-09-10 .
- 219 7. Wein, Alan J.; Kavoussi, Louis R.; Novick, Andrew C.; Partin, Alan W.;
220 Peters, Craig A. (2011). Campbell–Walsh Urology: Expert Consult Premium
221 Edition: Enhanced Online Features and Print, 4-Volume Set. Elsevier

- 222 Health Sciences. p. 2309. ISBN 9781416069119. Archived from the
223 original on 2017-09-10 .
- 224 8. Montgomery, Elizabeth A.; Voltaggio, Lysandra (2012). Biopsy
225 Interpretation of the Gastrointestinal Tract Mucosa: Volume 1: Non-
226 Neoplastic (2 ed.). Lippincott Williams & Wilkins. p. 10. ISBN
227 9781451180589 .
- 228 9. Underner, M; Perriot, J; Peiffer, G (January 2012). "[Smokeless tobacco]".
229 Presse Médicale. 41 (1): 3-9. doi:10.1016/j.lpm.2011.06.005. PMID
230 21840161 .
- 231 10. PA Reichart* and H Kohn, Oral Dis dec,2(4):291-4 1996
- 232 11. Zhang X1, Li C, Song Y, Reichart PA Oral Maxillofac Surg dec,14(4):195-
233 202 2008
- 234 12. Kumar S1, Muniyandi M , Asian Pac J Cancer Rev . 16(4):15-8 2015
- 235 13. Granero Fernandez M1, Lopez-Jornet P2 Aust Dent J mar,62(1) :47-51 .2016
- 236 14. Patil S1, Doni B2, Maheshwari S3 Can Geriatr J mar31 ,18(1) :11-40 2015
- 237 15. Reichart PA et al, Oral Dis nov,9(6):302-4 1996
- 238 16. R Chandran, S Meer, L Feller Oral Dis sep,19(6):592-7 2012
- 239 17. El Toum S1, Cassia A1, Bouchi N2, Kassab I2. Int J Dent 4030134 2018
- 240 18. Department of Conservative Dentistry (Director: Prof. J. Bánóczy, M.D.,
241 C.Sc.), Faculty of Dentistry, Semmelweis Medical University, Budapest,
242 Hungary 2005
- 243 19. Sujatha D1, Hebbar PB, Pai A. 2012 The Oxford Dental College and
244 Hospital, Bangalore, India
- 245 20. Cebeci AR1, Gülşahi A, Kamburoglu K, Orhan BK, Oztaş B Med Oral Patol
246 Oral Cir Bucal jun1,(416):e272-7 2009
- 247 21. Dagli RJ1, Kumar S, Mathur A, Balasubramanyam G, Duraiswamy P,
248 Kulkarni S Med Oral Patol Oral Cir Bucal nov1,13(11):e687-92 2008

- 249 22.Femopase FL1, Binagui MV, López de Blanc S, Gandolfo M, Lanfranchi HE.
250 Acta Odontol Latinoam 10(2):89–99 1997
- 251 23. GARY CHADMARTIND.D.S., M.P.H.1JOHN P.BROWNB.D.S.,
252 PH.D.2CLAYTON W.EIFLERPH.D.3GLEN D.HOUSTOND.D.S.,
253 M.S.D.1999
- 254 24. Splieth CH1, Sümrig W, Bessel F, John U, Kocher T Quintessence Int.V104
255 2007
- 256 25.Mathewal , Indian J Dent Res apr–jun,19(2):99–103 2008
- 257 26.Saraswathi TR1, Ranganathan K, Shanmugam S, Sowmya R, Narasimhan
258 PD, Gunaseelan R Indean J Dent Res sep,17(3):121–5 .2006
- 259 27.Rooban Thavarajah Arch Oral Biol jun,51(6):512–9 2006
- 260 28.Højgaard, AD; Jessen, AL (Aug 26, 1991). "[Bladder leukoplakia]". Ugeskrift
261 for læger. 153 (35): 2408–9. PMID 1949238 .
262