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A retrospective study on malignant transformation of oral leukoplakia and its relationship with oral squamous cell carcinoma

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Abstract

Background: The term oral leukoplakia is defined as white plaques of debatable risk, diagnosed when other known diseases or disorders that bring no risk for oral cancer have been omitted. It has a malignant transformation rate of around 0.13%-34% whether it is measured as a premalignant lesion.

Objective: This study aimed to evaluate the demographic occurrence and malignant transformation of oral leukoplakia in the diagnosis of OSCC in an Indian sample.

Material and method: The study included gender, age, site of the lesion, clinical presence and histopathological analysis of 156 subjects. SPSS software is used for statistical calculation. For data analysis calculation of percentages, Shapiro-Wilk test and Chi-square test is used.

Result: This study was conducted on a total of 156 patients with oral leukoplakia. In which 142 subjects were found affected with oral leukoplakia and only 14 subjects were found affected with other premalignant disorders. However, male subjects of oral leukoplakia were 59.15% and female subjects were less in number only 40.85%. A total number of 81patients were found in the age range 40-50 years. Male patients had acquired 55.56% while female patient's attained 44.44%. The histopathological examination stated that out of 142 oral leukoplakia cases, 39.44% were existent with malignant transformation and the P-value was 0.0185, which was found to be statistically weighty.

Conclusion: Half of OSCCs are one way or another linked with or originated by oral leukoplakia. So biopsy of chronic lesions especially male patients (because of the higher risk to develop dysplastic changes) is recommended to check dysplastic alterations within the lesion and should be advised to leave the adversarial habit.

Keywords: Oral leukoplakia, oral squamous cell carcinoma and buccal lesion

Introduction

The term oral leukoplakia is defined as white plaques of debatable risk, diagnosed when other

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known diseases or disorders that bring no risk for oral cancer have been omitted [1]. It has a malignant transformation rate of around 0.13%-34% whether it is measured as a premalignant lesion [2]. The cause of oral leukoplakia is unknown but excessive smoking, tobacco chewing, betel nut chewing has the potential to develop it. Basically, it has no symptoms but it is allied with oral squamous cell carcinoma.

Classification of oral leukoplakia: There are two types of oral leukoplakia.

- **Homogenous oral leukoplakia:** It has a uniform white patch with a lower risk of turning into cancer.
- **Non-homogenous oral leukoplakia:** It contains a uniform red-white patch with a higher risk of converting into cancer.

There is another variant also which is rare and extensive, proliferative verrucous leukoplakia embroils in diverse parts of the mouth and is more communal in grown-up women; has a greater likelihood of turning into cancer [1].
Oral squamous cell carcinoma (OSCC) is extensively documented as the most communal type of head and neck cancer, with a ~50% continued existence in 5 years in spite of numerous treatments in the past [3, 4]. Oral squamous cell carcinoma (OSCC) has different variants like verrucous carcinoma (VC), adenoid/acantholytic/pseudoglandular SCC (AdSCC), spindle cell/sarcomatoid carcinoma (SCSC), adenosquamous carcinoma (ASC), basaloid SCC (BSCC) and papillary SCC (PSCC). Individually these variants have a distinctive history and morphology of cells [5]. Moreover, several diseases, including severe functional and cosmetic defects, mucositis, xerostomia and osteoradionecrosis are also connected with OSCC, which damage the quality of life of patients [6].
An estimated overall mean proportion rate of malignant transformation (MT) is 9.3% for OL according to recent studies [7]. Other studies have defined a twelve-monthly reappearance rate of about 13.5%-17%, following surgical removal [8, 9]. Therefore, this study aimed to evaluate the demographic occurrence and malignant transformation of oral leukoplakia in the diagnosis of OSCC in an Indian sample.

Materials and Methods

From the previous record of biopsy, data of 156 patients with oral leukoplakia were collected from the department of oral pathology. The study was conducted from March 2013 to Dec. 2013. The study included gender, age, site of the lesion, clinical presence and histopathological analysis. A duly filled consent form was obtained from all the participants.
The age range of 30-50 years of patients of both sex male and female were selected with oral leukoplakia. Patient with other oral problems and lesions which affect other tissue SPSS software is used for statistical calculation. For data analysis calculation of percentages, Shapiro-Wilk test and Chi-square test is used.

Results

This study was conducted on a total of 156 patients with oral leukoplakia. In which 142 subjects were found affected with oral leukoplakia and only 14 subjects were found affected with other premalignant disorders. However, male subjects of oral leukoplakia were 59.15% and female subjects were less in number only 40.85%. As described in table 1, female subjects had more other premalignant disorders 57.14% as likened to males (42.86%).

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Table 1: The distribution of mucosal lesions in males and females

Mucosal lesion	Male		Female		Total
	n	n%	n	n%	
Oral leukoplakia	84	59.15	58	40.85	142
Other premalignant disorders	6	42.86	8	57.14	14
Chi-square test	1.387				
P	0.238				

*Statistically significant, P≤0.05.

A total number of 81patients were found in the age range 40-50 years. Male patients had acquired 55.56% while female patients attained 44.44% as shown in Table 2.In Table 3, the most collective site tangled was buccal mucosa with 106 cases, shadowed by tongue 25 and floor of mouth 13 cases. Total 69.01% of oral leukoplakia cases were existing with buccal mucosa participation while 57.14% cases of other premalignant lesions were connecting buccal mucosa. The histopathological examination stated that out of 142 oral leukoplakia cases, 39.44% were existent with malignant transformation and the P-value was 0.0185, which was found to be statistically weighty [Table 4].

Table 2: The demographic data of study subjects

Gender	Age										Chi-square test	p
	20-30 years		30-40 years		40-50 years		50-60 years		60-70 years			
	n	n%	n	n%	n	n%	n	n%	n	n%		
Male	3	42.86	13	36.11	45	55.56	14	70.00	9	75.00	9.25	0.05
Female	4	57.14	23	63.89	36	44.44	6	30.00	3	25.00		
total	7	100	36	100	81	100	20	100	12	100		

*Statistically significant, P≤0.05

Table 3: The distribution of lesions in the oral cavity

Oral cavity	Oral leukoplakia (n=142)		Other premalignant Disorders(n=14)		Total
	n	n%	n	n%	
Buccal mucosa	98	69.01	8	57.14	106
Labial mucosa	3	2.11	1	7.14	4
Tongue	22	15.49	3	21.43	25
Floor of mouth	12	8.45	1	7.14	13
Other	7	4.93	1	7.14	8
Chi-square test	1.950				
P	0.741277				

*Statistically significant, P≤0.05

Table 4: The cytopathic distribution of oral leukoplakia and other premalignant disorders

Histopathy	Oral leukoplakia (n=142)		Other premalignant disorders (n=14)		Total
	n	n%	n	n%	
Mild dysplasia	25	17.61	1	7.14	26
Moderate dysplasia	61	42.96	2	14.29	63
Malignant transformation	56	39.44	11	78.57	67

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Chi-square test	7.975
P	0.0185

*Statistically significant, P≤0.05

Discussion

Oral squamous cell carcinoma is a communal malignancy globally and the record stumbles upon oral malignant tumors [10]. It has several causes but the most significant influences are tobacco and alcohol [11]. The term oral leukoplakia was first introduced by Ernő Schwimmer in 1877 [12]. The occurrence of oral leukoplakia diverse from 0.2% to 4.9%, whereas according to Petti the worldwide pervasiveness is 2.6% [13, 14]. The oral leukoplakia cases are predominant in an older age group beyond 40 years supports by different studies [15, 16]. In 2003 Petti, stated that the most shared age measured was fourth to the fifth decade followed by the seventh decade for oral leukoplakia which is similar to our study where 81 patients were found infected with this disease had age 40-50 years [14]. Additionally, this study is also allied with Silverman *et al.'s* report which describes that oral leukoplakia amplified with an increase in age [17].

In the present study, male subjects of oral leukoplakia were 59.15% and while female subjects were 40.85%.This disagrees with the study of Cerqueira JM, conducted on the Brazilian population where most of the patients were female (56%) [18] Although matches with A study by Gupta *et al*. in 1980 indicated oral leukoplakia incidence rate of 1.1-2.4/1000 people/year for males and 0.2-1.3/1000 people/year for females [19].

The buccal mucosa site is the most prominent site of occurrence of oral leukoplakia. The data of the current study shows buccal mucosa with 106 cases, shadowed by tongue 25 and floor of mouth 13 cases. As we know, oral leukoplakia is associated with oral squamous cell carcinoma, so we include a comparison of oral leukoplakia with malignant transformation. In 2003, Petti quantified that the annual rate of oral leukoplakia malignant transformation is 1.36% (95% confidence interval: 0.69%-2.03%) in several populations and terrestrial areas [14]. Numerous studies stated that 15.8%-48.0% of OSCC patients were related to oral leukoplakia when analyzed [20, 21], however in 2005, Mishra *et al*. identified that the risk of developing OSCC at lesion sites is five times more in patients with oral leukoplakia as compared to patients without them [16].

The dysplasia word represents abnormal growth [22]. In this study, moderate dysplasia was 42.96% and their conversion into the cancerous cell was 39.44% while more cases of oral leukoplakia converted into OSCC. Schepman *et al*. (1998) conclude that malignant transformation is more expected to arise within dysplastic lesions, dysplasia is not an obligatory form and the risk of malignant transformation spreads outside the limits of the recognized oral leukoplakia [23]. All the same, Silverman *et al*. states 31.4%-36.3% risk of malignancy for patients with any degree of dysplasia [17]. Previous literature recommended that oral leukoplakia is a premalignant lesion and an indicator of amplified carcinoma threat in the entire oral cavity [12].

Conclusion

The clinical diagnosis of Oral leukoplakia is relevant to white lesions of the oral cavity that is associated with habits like smoking, tobacco, or betel nut chewing. Half of OSCCs are one way or another linked with or originated by oral leukoplakia. So biopsy of chronic lesions is

recommended to check dysplastic alterations within the lesion. As discussed earlier, oral leukoplakia is linked with bad habits, so all oral leukoplakia patients and especially males patients (because of the higher risk to develop dysplastic changes), should be advised to leave the adversarial habit and strictly follow repeated biopsy schedule to avoid malignant transformation and expansion of OSCCs.

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