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ORAL LEUKOPLAKIA: A REVIEW

Dr. Veena B. Pujari[1]* and Dr. Savita Thakkannavar[2]

[1]Department of Oral Medicine and Radiology Tatyasaheb Kore Dental College and Research Center New Pargaon, Kolhapur, Maharashtra. India.
[2]Department of Oral Pathology and Microbiology Tatyasaheb Kore Dental College and Research Center New Pargaon, Kolhapur, Maharashtra. India.

***Corresponding Author: Dr. Veena B. Pujari**
Department of Oral Medicine and Radiology Tatyasaheb Kore Dental College and Research Center New Pargaon, Kolhapur, Maharashtra. India.

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ABSTRACT
Oral cavity presents various lesions depending on the etiology, site and usage of tobacco and alcohol. Among all the lesions known oral leukoplakia is the most common potentially malignant disorder of the oral mucosa. It is a predominantly white lesion which cannot be characterized clinicopathologically as any other definable lesion. The greater risk of malignant change is seen in women than men. Hence, a complete knowledge of this lesion by the clinician helps early diagnosis and appropriate treatment facilitating a good prognosis and a better quality of life

KEYWORDS: White lesion, Potentially malignant, Tobacco, homogenous, verrucous, Erythroleukoplakia.

INTRODUCTION
Leukoplakia is derived from Greek word- Leucos means white and plakia means patch. Oral leukoplakia is the most common premalignant or "potentially malignant" lesion of the oral mucosa. It has been defined as "a predominantly white lesion of the oral mucosa that cannot be characterized as any other definable lesion."[1] WHO defined leukoplakia as "a white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.[2] Leukoplakia is broadly categorized into two forms: Homogeneous, which is predominantly white flat smooth lesion of uniform, thin appearance, having a wrinkled or corrugated surface throughout the lesion and the non-homogeneous type, which is a mixed white-and-red lesion that may be either irregularly flat, nodular, or verrucous.[3] Leukoplakia is a clinical term and the lesion has no specific histology. As malignant transformation rate of leukoplakia increases up to 17.5% with time, there is a need for continued observations no matter how long the lesions have been present.

EPIDEMIOLOGY
There is a wide range of prevalence of leukoplakia among various populations. Martorell-Calatayud *et al*. found the prevalence of leukoplakia to be 0.4% to 0.7% of the population.[4] In India, prevalence of leukoplakia was about 0.2% to 4.9% of the population over the age of 15 years [5] and less than 1.3% of leukoplakias in India are found to be idiopathic.[6] According to Bánóczy the adult population prevalence varied between 0.6% and 3.6%.[7] According to Feller and Lemmer the prevalence of leukoplakia ranged from 0.5% to 3.46%, and also the malignant transformation of leukoplakia was stated to be 0.7% to 2.9%,[8] whereas Downer and Petti stated a malignant transformation of leukoplakia annually between 6.2 and 29.1 cases per 100,000 people.[9] Similarly Brouns *et al*. found the prevalence of leukoplakia as approximately 2% with a malignant transformation of approximately 1% annually.[10] Men are affected more frequently than women, and majority of leukoplakia occurs in the third and fourth decade of life. Bánóczy found that the incidence of leukoplakia was higher in patients who were habitual smokers or drinker, and also a ratio of 3.2:1 was seen in sex distribution.[11] Brouns *et al*. found the mean age of 57 years among 275 patients among whom 112 were men and 163 were women.[10] A follow up study was conducted on 166 patients on hospital-based population by Axéll *et al*. and it was found that leukoplakia was commonly present in buccal mucosa (76%), alveolar sulcus (19%), and tongue (5%).[12] On the contrary, Liu *et al*. conducted a study on 218 patients and found that tongue was affected in 51.4% patients and 32.6% in buccal mucosa.[13]

CLASSIFICATION
Clinically oral leukoplakia is classified into four types:
- Early or thin
- Homogenous or thick
- Granular or verruciform and
- Speckled or erythroleukoplakia.

Each subdivision has a different malignant transformation potential.[14]

VARIOUS FORMS OF LEUKOPLAKIA

1. Homogenous leukoplakia- the lesion is uniformly flat, thin and exhibits shallow surface cracks. The risk of malignant transformation is relatively low.[1]
2. Oral erythroleukoplakia/ speckled leukoplakia - It is a non-homogenous lesion consisting both white and red components and shows higher malignant transformation than homogenous leukoplakia..[1]
3. Proliferative verrucous leukoplakia – most aggressive form of leukoplakia and was first described by Hansen et al. in 1985. It is multifocal has a wrinkled appearance, spreads slowly and often presents with surface projections. Most frequently affected area are the mandibular gingiva, tongue, buccal mucosa, and alveolar ridge.[1]
4. Candidial leukoplakia- is a chronic, discrete, translucent or opaque elevated lesion that is palpable and rough to touch.[15]
5. Oral hairy leukoplakia - is also known as Greenspan lesion. It is most commonly present on the lateral borders of the tongue and is characterized by white patches with a corrugated or hairy surface caused by Epstein-Barr virus infection.[16]
6. A provisional diagnosis of leukoplakia is made whe n a predominantly white lesion at clinical examination cannot be clearly diagnosed as any other disease or disorder of the oral mucosa (Table 3). A biopsy is mandatory.
7. A definitive diagnosis is made when any aetiological cause other than tobacco /areca nut use has been excluded and histopathology has not confirmed any other specifi.
8. A provisional diagnosis of leukoplakia is made whe n a predominantly white lesion at clinical examination cannot be clearly diagnosed as any other disease or disorder of the oral mucosa (Table 3). A biopsy is mandatory.
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DIAGNOSIS

A 2-4 weeks observational period should be allowed for regression of the white lesion after elimination of the causative factor. Clinically, a provisional diagnosis of leukoplakia is made when a predominantly white lesion at clinical examination cannot be clearly diagnosed as any other disease or disorder of the oral mucosa (Table 1). [1] Histopathologic evaluation is the gold standard for diagnosis and t is mandatory to take the biopsy at the most clinically suspicious area. Excisional biopsy can be considered in case of smaller size lesion field mapping in a widespread and multifocal lesion. Histological report should include presence or absence of epithelial dysplasia. A final diagnosis is made by excluding other etiological causes other than tobacco/areca nut use and histopathology has not confirmed any other specific disorder (fig.1)[1]

Table 1: Exclusion of lesions/disorders to diagnose leukoplakia.

Disorder	Diagnostic features	Investigation
Frictional keratosis	History of low grade chronic trauma, along the occlusal plane with an apparent etiology, mostly reversible on removing the cause	Biopsy if lesion persists after elimination of cause
Morsicatio buccarum	Habitual cheek/lip biting , irregular white lesion with jagged out line	Biopsy not indicated
Chemical injury	Known history, presence of pseudomembrane, painful, resolves rapidly	Not indicated
Lichen planus (plaque type)	Other forms of lichen planus (reticular) found in association	Biopsy consistent with lichen planus
Lichenoid reaction	Drug history, lesion in contact to an amalgam restoration	Biopsy consistent with lichenoid reaction
Smoker's palate	Smoking history, greyish white palate	Not indicated
White sponge nevus	Family history, Noted in early life, family history, large areas involved, genital mucosa may be affected	Biopsy not indicated
Discoid lupus erythematoses	Circumscribed lesion with central erythema and white lines radiating	Biopsy consistent with DLE,immunofloresence

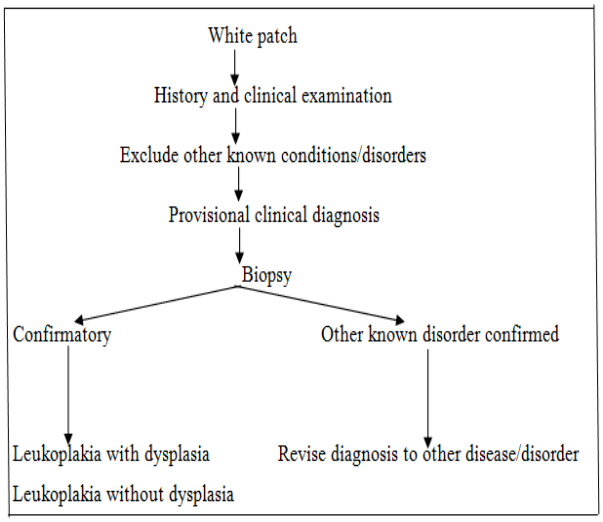

FIGURE 1: A schematic diagram for diagnosis of oral leukoplakia.

LOW RISK LEUKOPLAKIA

Lesions which are
a. not in high risk area
b. less than 200 mm in size
c. Homogenous clinical form
d. no dysplastic features

HIGH RISK LEUKOPLAKIA

Lesions which are
a. occur in high risk area
b. greater than 200mm in size
c. Non homogenous clinical form
d. presence of dysplastic features

STAGING OF LEUKOPLAKIA

A staging system for oral leukoplakia was recommended by WHO in 2005 on the lines of TNM staging considering the size of the lesion (L) and the histopathological features (P).[17]
L1 -Size of leukoplakia is < 2cm
L2 - Size of leukoplakia is 2 - 4 cm
L3 - Size of leukoplakia is >4cm
Lx - Size of leukoplakia is not specified

(P - Pathology)
Px - Dysplasia not specified in pathology report
P0 - No epithelial dysplasia
P1 - Mild to moderate epithelial dysplasia
P2 - Severe epithelial dysplasia

OLEP STAGING SYSTEM (L-size of the lesion, P-histopathological feature)
Stage I: L1P0
Stage II: L2P0
Stage III: L3P0 or L1/L2P1
Stage IV: L3P1or any LP2.

MANAGEMENT

Elimination of risk factors such as tobacco and alcohol abuse, betel chewing, superimposed candida infection over the lesion is the primary step in treatment of leukoplakia. A thorough clinical examination should be repeated every fortnight to assess the regression of the size of the lesion after habit cessation. If the size of leukoplakia shows reduction, follow up is done initially every three months followed by every 6-12 months.[18] Low risk leukoplakia may be either completely removed depending on the location, size and cessation of habit, For high risk leukoplakia surgical treatment is recommended.[19] Non-surgical treatment is indicated in patients with widespread lesion involving a large area of the oral mucosa or medically compromised patients unfit or at risk for surgery, or when the patients refuse surgical intervention.[20] Conservative treatment includes use of vitamins A, C, E, carotenoids such as beta-carotene, lycopene, bleomycin, protease inhibitor, anti-inflammatory drugs, green tea, curcumin etc.[21]

CONCLUSION

It is a well known fact that leukoplakia has the potential for malignant transformation and hence, for an oral physician a complete knowledge of the lesion from the etiology, features, diagnosis and its appropriate treatment gives a better prognosis and plays a key role in saving the patient's life.

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