ejpmr, 2025, 12(4), 116-118

SJIF Impact Factor 7.065

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Review Article

ISSN 2394-3211

EJPMR

IDIOPATHIC LEUKOPLAKIA OF TONGUE: A RARE ORAL LESION WITH MALIGNANT POTENTIAL

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Article Received on 25/01/2025 **Article Revised on 15/02/2025** **Article Published on 07/03/2025**

ABSTRACT

Oral leukoplakia, the most prevalent premalignant lesion of the oral cavity, is broadly categorized into homogeneous and nonhomogeneous types, with idiopathic leukoplakia representing a unique subset unassociated with tobacco or other identifiable risk factors. Idiopathic leukoplakia is more frequently observed on the tongue and carries a higher risk of malignant transformation compared to tobacco-associated lesions. This report details the case of a 47-year-old female presenting with a hyperkeratotic lesion on the right lateral border of the tongue, diagnosed as Idiopathic leukoplakia of tongue. The lesion's idiopathic nature was emphasized by the patient's lack of deleterious habits and relevant systemic conditions. The discussion highlights the multifactorial etiology of oral leukoplakia, including potential links to HPV infection, genetic alterations, and other unidentified factors. The case reinforces the significance of early diagnosis, detailed clinical evaluation, and histopathological examination for accurate differentiation and management.

KEYWORDS: Oral leukoplakia, Idiopathic leucoplakia, Hyperkeratosis, Premalignant lesion, Dysplasia, Malignant potential.

INTRODUCTION

Oral leukoplakia is a common premalignant lesion which is defined by the World Health Organization (WHO) as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease".[1]

Leukoplakia can be broadly categorized as homogeneous and nonhomogeneous types. Homogeneous leukoplakia is typically identified as a white, well-defined plaque with a consistent appearance throughout the lesion. Its surface may vary from smooth and thin to rough and leathery, sometimes showing cracks resembling "cracked mud".[3]

Non homogeneous leukoplakia, in contrast, features white patches or plaques mixed with red areas. This combination of white and red regions is also referred to as erythroleukoplakia or speckled leukoplakia. Leukoplakias with verrucous, nodular (papillary), or outward-growing (exophytic) features, even if their surface texture is uniform, are also classified as nonhomogeneous. Both forms of leukoplakia can appear on any sites in the oral mucosa.[3]

Smoking is the primary risk factor for leukoplakia, with various forms of tobacco use, including cigarettes, cigars, beedis, and pipes, significantly contributing to its development. Other contributing factors can amplify the risk, such as excessive alcohol intake, persistent oral irritation, fungal infections like candidiasis, oral galvanism from dental restorations, and bacterial infections. Sexually transmitted diseases such as syphilis, combined micronutrient deficiencies, viral infections, hormonal imbalances, and prolonged exposure to ultraviolet radiation, also play a role in its development.[10]

Additionally, a rare type of leukoplakia which is not associated with tobacco use is known as nontobacco or idiopathic leucoplakia.[2] Nonsmokers are more likely to develop idiopathic leukoplakias at the tongue's border compared to smokers. High-risk sites for malignant transformation include the floor of the mouth and the lateral borders of the tongue. These areas also exhibit a higher frequency of loss of heterozygosity compared to low-risk sites.[3]

CASE REPORT

A 47 years old Female patient presented to the Department of Oral Medicine and Radiology at Tagore Dental College and Hospital with a chief complaint of missing tooth in her upper and lower right back tooth region for past 2 years and wants to replace it. Patient give history of food lodgement in her lower right back tooth region for past 3 months. Patient did not give any relevant past medical history. Patient did not have deleterious habits history such as smoking, alcohol consumption, tobacco chewing etc. Patient gave no history of any prior dental treatments.

On examination of tongue, Inspection revealed a whitish hyperkeratotic patch in the right lateral aspect of tongue which extended 1 cm away from the tip of the tongue to the junction of anterior & middle third of the tongue, elliptical in shape and approximately 0.5 cm x 1 cm in size. The lesion had well defined border demarcating it from the adjacent surrounding tissues. The surface of the lesion was smooth and "cracked mud" appearance was evident. On palpation, all inspectory findings were confirmed. Lesion was non-tender and non-scrapable [Fig 1]. Lips, buccal mucosa, Labial mucosa, palate, Extra oral soft tissue findings were normal. Lymphadenopathy was absent. Hard tissue findings were Missing teeth 16, 23,38,46,47,48; Dental caries in 13,24,25,37; Root stumps in relation to 14,15,18,26,27,28,36; Mild calculus and stains; Sharp teeth in 43,44.

Taking into consideration the characteristic clinical features, the condition was diagnosed as Idiopathic Leukoplakia. The differential diagnoses include Oral candidiasis, Oral lichen planus, Frictional keratosis, Leukoedema, White sponge nevus, and even surface debris. Enameloplasty of sharp tooth was done in 43,44. Patient was explained about the potential risk of malignant transformation of the lesion. Patient was advised for biopsy. Patient was not willing for biopsy and did not return for follow up.

Figure 1: Idiopathic leukoplakia seen on the right lateral border of tongue

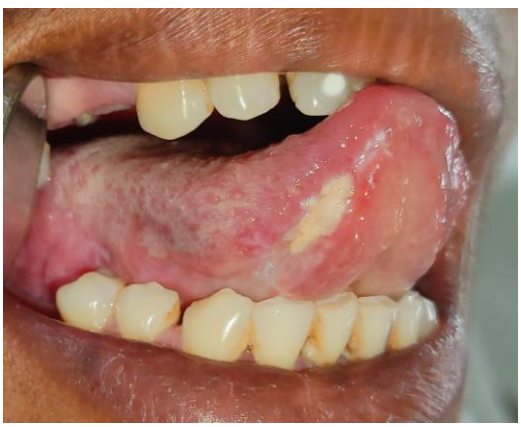

Fig. 1: Idiopathic leukoplakia seen on the right lateral border of tongue.

DISCUSSION

Leukoplakia is the most common form of oral pre malignant lesion, accounting for 85% of the lesions. Studies estimate its global prevalence to range from 1.5% to 4.3%.[1] When encountering oral leukoplakia with unknown cause, it is important to thoroughly evaluate the patient's history, oral habits, systemic conditions, and histological findings to arrive at an accurate diagnosis. A differential diagnosis helps systematically eliminate other potential causes based on clinical and laboratory data.[4]

Approximately 70–90% of oral leukoplakias are linked to smoking tobacco or areca nut usage, individually or combined. There is a clear correlation between the frequency and duration of tobacco use and the occurrence of leukoplakia. However, the underlying mechanisms of idiopathic leukoplakia remain unknown.[5] While human papillomavirus (HPV) infection and excessive alcohol consumption may be associated with leukoplakia, there is limited evidence to suggest a direct causal relationship.[5]

The progression of oral leukoplakia into premalignant lesions involves various genetic changes. Studies indicate that different genetic markers are expressed in different leukoplakias. Oncogene activation, suppressor gene deletion, and DNA repair gene damage contribute to genomic dysfunction, ultimately leading to malignant transformation after a series of mutations.[5]

Notably, idiopathic leukoplakia has a higher risk of malignant transformation compared to tobacco-associated leucoplakia.[5]

A study by Freitas et al. found that dysplastic lesions were more frequent in non-smokers than smokers, a statistically significant finding ($P = 0.026$).[6] Idiopathic leukoplakia most commonly occurs on the tongue. Shesha Prasad R et al. reported a case of idiopathic leukoplakia on the lateral border of the tongue in a 78-year-old male, while R.S. Faecher et al. documented a nonspecific case in a 67-year-old male on the tongue.[7,8] Though rare, idiopathic leukoplakia can appear in other parts of the oral cavity, as highlighted by Sapna et al., who observed linear gingival leukoplakia in a 40-year-old male. These lesions exhibit a high likelihood of recurrence and malignant transformation.[9]

CONCLUSION

In summary, leukoplakia remains the most prevalent oral precancerous lesion, with a significant proportion linked to modifiable risk factors such as smoking and areca nut use. However, idiopathic leukoplakia, characterized by its unknown etiology and higher potential for malignant transformation, presents unique diagnostic and management challenges. Thorough evaluation of patient history, habits, systemic conditions, and histological findings is critical for accurate diagnosis and differentiation from other lesions. The genetic basis of

leukoplakia highlights the complexity of its progression to malignancy, underscoring the need for early detection and close monitoring. Continued research and case studies are essential to better understand and manage this potentially malignant condition.

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