

# Leukoplakia Transforming into Carcinoma in Situ: A Case Report

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## Abstract

Malignancies are preceded by potentially malignant disorders. Leukoplakia is one of the precursors of oral squamous cell carcinoma. Leukoplakia is one of the types of potentially malignant disorder that have potential to convert into carcinoma frequently. Leukoplakia poses a major diagnostic and therapeutic challenge. We present a case of leukoplakia in left side of buccal mucosa which is converted into carcinoma in situ along with review in light of current information from the literature. We also attempt to present the clinical relevance, and the therapeutic modalities available for the management of the disease.

**Keywords:** Malignancies, potentially malignant disorders, Leukoplakia, Carcinoma.

## Introduction

Public awareness of lesions that can potentially be a malignancy in oral cavity has been increasing. One lesion that can be found in the oral cavity is leukoplakia. Leukoplakia is derived from the word “leuko” which means white, and “plakia” which refers to the word plaques or patches. Thus, leukoplakia can be defined as a white plaque that cannot be scraped off. Its etiology, however, is still questionable after eliminating all risk factors that do not have a tendency toward malignancy [1]. Approximately, 3% of the worldwide population has suffered from leukoplakia, 5-25% of which are pre-malignant lesions. After verified through histopathological examination, all lesions of leukoplakia can be considered as a potentially malignant lesion. [2] In a meeting supported by the World Health Organization, oral leukoplakia has been defined as “a predominantly white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer.[3] It has been added that leukoplakia is a clinical term and has no specific histology. In a recent report, a suggestion has been made to somewhat simplify the definition of leukoplakia into “a predominantly white, non-wipable lesion of the oral mucosa having excluded other well-defined predominantly white lesions clinically, histopathologically or by the use of other diagnostic aids.[4]

## Case Report

A 48 year old male patient reported to the outpatient department of Rama Dental College, Kanpur with a white patch on left side of buccal mucosa. The white patch was first noticed 6 months ago which showed gradual enlargement causing discomfort and burning sensation. He had been smoking since the age of approximately 22 years, but he has stopped the habit since 4 years ago. Along with this the patient also consumed alcohol occasionally. Intraoral Examination of left buccal mucosa revealed non tender, non scrapable, white patch measuring 4 x 2 cm. Surface appears rough and slightly elevated which clinically resembled leukoplakia in appearance. The lesion was typically gives the cracked mud appearance. Chair side investigation, toluidine blue staining was carried out to select the area of biopsy to be made. The selected area was then biopsied.

Histopathologic examination with H and E stained sections showed squamous epithelium overlying connective tissue stroma. The epithelium shows Dysplastic features such as broad rete ridges, acanthosis, hyperchromatism, basilar hyperplasia, cellular and nuclear pleomorphism, and abnormal mitosis, Individual cell keratinization and intraepithelial keratin pearls formation. The dysplastic epithelial changes has extended top to bottom. Connective tissue stroma is highly cellular with dense infiltration of chronic inflammatory cells, dilated and proliferated capillaries. The overall clinical and histopathological findings were

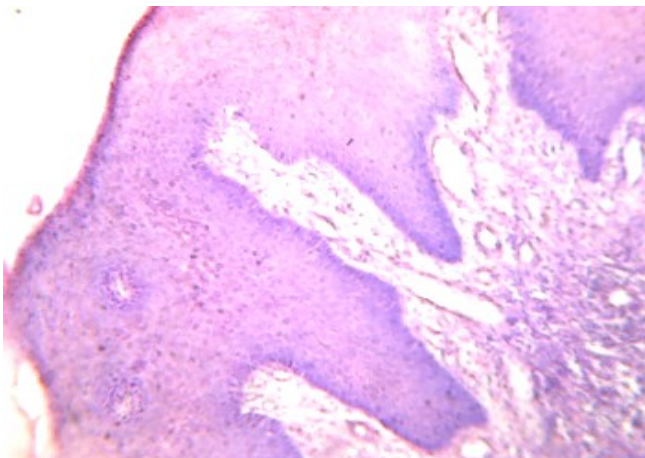
considered diagnostic for leukoplakia transforming into carcinoma in situ.



**Figure 1: Showing leukoplakia in left side of buccal mucosa.**



**Figure 2: Grossing image showing tissue of biopsy.**



**Figure 3: Histopathology showing speckled leukoplakia transforming into Carcinoma in situ [H & E X10]**

## Discussion

WHO defines leukoplakia as a whitish patch or plaque that cannot be characterized, clinically or pathologically, as any other disease and which is not associated with any other physical or chemical causative agent except the use of tobacco.[5] The literature, however, strongly indicates the role of alcohol, viruses and systemic conditions. The World Health Organization (WHO) employs the term Leukoplakia (SL) to describe the presence of white patches on the oral mucosa.[6] The two main clinical types of leukoplakia are homogeneous and non homogeneous leukoplakia. Etiological factors involved are alcohol use and smoking, diets lacking antioxidants (such as vitamins C, E, and beta-carotenes), occupational exposure to carcinogens, viral infections, and genetic and hereditary factors. Smoking of tobacco was found to be the strongest independent risk factor. Other forms of tobacco, hyperacidity, lipstick, and ill-fitting dentures were found to be a causative factor, which shows that socioeconomic status and lifestyle are involved in causing premalignant lesions. Various designations have been used to describe the presence of both white and red patches. Lesions appearing completely red are named as Erythroplakia. Leukoplakia is indicated when white patches are present over the mucosa.[4]

Leukoplakia carries a risk of developing into malignancy. So early diagnosis by biopsy has to be done to avoid the dangerous malignant transformation.[8] Approximately 80 percent of leukoplakia progress to oral carcinoma over a period of time in spite of a variety of interventions. This feature contrasts with homogenous leukoplakia in which approximately 5-10 percent will transform into a carcinoma. [4] Leukoplakia is resistant to most of the available treatment modalities, including surgery. Therefore, total excision with free surgical margins is critical combined with a lifelong follow-up. [3] Malignant potential of leukoplakia is higher in women (6%) than in men (3.9%). Leukoplakia associated with habit of chewing tobacco shows higher rate of malignant transformation as compared to others.[7] In buccal mucosa and commissure region 1.8 percent malignant transformation can occur. In lip and tongue region 16 to 38.8 percent malignant transformation has been reported. The annual malignant transformation rate has been determined to be 0.1% to 17%. [9]

## Management

The degree of epithelial dysplasia plays a pivotal role while deciding the nature of treatment to the patient. Martorell- Calatayud. [10] Defined two risk groups and the subsequent treatment options:

**Group 1: Those with low risk of malignisation:**

Those leukoplakias lacking dysplasia, and those that show mild dysplasia located in low risk areas or those with a thickness of less than 200 mm or that present clinically as homogenous leukoplakia. A range of therapeutic approaches can be taken in this group:

Regular patient follow-up. The interval between follow-up visits should not exceed 12 months in order to detect any change, suggestive of malignant transformation.

Treatment of lesions with topical or oral retinoid {eg: 13-Cis-Retinoic Acid (1.5 to 2 mg/kg body weight for 3 months)} [10]

Treatments using nonsurgical ablative techniques, such as cryotherapy and carbon dioxide laser ablation. Of these options, the use of laser light has shown better results in terms of controlling the lesions, and so it is considered the treatment of choice in this low risk group.

**Group 2: Those with high-risk of malignant transformation, which comprises:**

Those leukoplakias with mild dysplasia located in high-risk areas measuring more than 200 mm, or those associated with a nonhomogenous clinical form; Leukoplakias with moderate or severe dysplasia;

Veracious leukoplakias. In this group, aggressive surgical treatment, consisting of excision of the entire thickness of the mucosa at the site of the leukoplakia is recommended. This is similar to the present case.

Among the many therapeutic options available, however, eliminating risk factors (smoking, alcohol) and etiological factors (sharp broken teeth, faulty metal restorations and metal bridges) are preventive measure applicable to all patients with these lesions. [8]

Regular check-up of these patients is essential, probably every 3, 6 and then 12 months, both in treated and untreated patients.

**Conclusion**

Leukoplakia is a potentially malignant disorder. Patient suffering from any potentially malignant disorder should be treated aggressively.

Since malignant transformation rate of leukoplakia is high early diagnosis plays a very important role in treatment as well as prognosis of leukoplakia. Hence biopsy has been advised in every cases of potentially malignant disorder.

**References**

1. Kardam P, Rehani S, Mehendiratta M, Sahay K, Mathias Y, Sharma R. Journey of leukoplakia so far - an insight on shortcomings of definitions and

classifications. *J Dent Oral Disord Ther.* 2015; 3(2):1-6.

2. Lingen MW. Head and neck. In: Kumar V, Abbas AK, Aster, JC. Editors. Robbins and cotran-pathologic basis of disease. 9th ed. Philadelphia: Elsevier; 2015. p. 731.
3. Warnakulasuriya S, Johnson NW, van derWaal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. *J Oral Pathol Med.* 2007; 36(10):575–80.
4. Van der Waal I. Historical perspective and nomenclature of potentially malignant or potentially premalignant oral epithelial lesions with emphasis on oral leukoplakia; some suggestions for modifications. *Oral Surg Oral Med Oral Pathol Oral Radiol.* 2018; 125(6):577–81.
5. Pindborg J.J., Reichart P., Smith C.J. and Van der Waal I. World Health Organization: histological typing of cancer and precancer of the oral mucosa. Berlin: Springer-Verlag; 1997.
6. Eversole LR. Dysplasia of the Upper Aero digestive Tract Squamous Epithelium. *Head and Neck Pathol.* 2009; 3:63–8.
7. Reibel J. Prognosis of oral premalignant lesions: significance of clinical, histopathological, and molecular biological characteristics. *Critical Reviews in Oral Biology and Medicine* 2003; 14(1):47–62.
8. Scuba J.J. Oral leukoplakia. *Critical Rev Oral Biol Med* 1995 ;( 2):147-160.
9. Lodi G. and Porter S. Management of potentially malignant disorders: evidence and critique. *Journal of Oral Pathology and Medicine* 2008; 37(2), 63–69.
10. Martorell-Calatayud,a R. Botella- Estrada,a J.V. Bagán-Sebastián,b O. Sanmartín-Jiménez,a and Guillén- Baróna C. Oral Leukoplakia: Clinical, Histopathologic, and Molecular Features and Therapeutic Approach. *Acta Dermosifiliogr.* 2009; 100:669-84.

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