

A Long Shadow from Childhood: Oral Leukoplakia with Epithelial Dysplasia in a 25-Year-Old with Early-Onset Tobacco Use

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Abstract

Background: Oral leukoplakia is the most common potentially malignant disorder of the oral mucosa, and its risk of malignant transformation is closely linked to the presence and severity of epithelial dysplasia. Tobacco use is a major modifiable risk factor, and early-onset exposure may result in a longer cumulative carcinogenic burden. Potentially malignant oral lesions occurring at a young age are less commonly reported and warrant clinical attention. We present a case of biopsy-confirmed oral leukoplakia with epithelial dysplasia in a young adult with a history of tobacco use beginning in childhood. **Methods:** A 25-year-old male presented with a persistent, non-scrapable white lesion on the lateral border of the tongue, with a reported history of tobacco use beginning at approximately 10 years of age. Clinical examination did not reveal an identifiable local mechanical or frictional cause. An incisional biopsy was performed, and the specimen was evaluated histopathologically. Based on clinical and histopathological findings, the patient was managed with a chemopreventive pharmacological regimen combined with tobacco-cessation counselling and structured clinical surveillance. **Results:** Histopathological examination demonstrated hyperkeratosis with orthokeratinization, acanthosis, loss of basal-cell polarity, premature and individual-cell keratinization, nuclear hyperchromatism, cellular pleomorphism, an increased nuclear-to-cytoplasmic ratio, atypical mitotic figures, and dyskeratosis, along with chronic subepithelial inflammatory infiltration and focal fibrosis findings consistent with oral leukoplakia with epithelial dysplasia. The patient was managed with oral lycopene, vitamin A (retinol), vitamin E, and topical tretinoin, alongside counselling for complete tobacco cessation and scheduled follow-up for lesion monitoring. **Conclusion:** This case highlights the clinical significance of early-onset tobacco exposure and the occurrence of potentially malignant oral lesions at a young age. It underscores the importance of timely biopsy and histopathological evaluation of persistent oral white lesions, particularly when conventional local causes are absent, along with long-term surveillance given the risk of malignant transformation.

Keywords: Oral leukoplakia, epithelial dysplasia, tobacco use, early tobacco exposure, chemoprevention, lycopene, retinol

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Introduction

Oral leukoplakia is defined as a predominantly white lesion of the oral mucosa that cannot be characterized as another definable disorder. It is the most frequently encountered potentially malignant disorder of the oral cavity. Its clinical importance is related to the possibility of underlying epithelial dysplasia and subsequent malignant transformation. The risk of progression varies according to several factors, including lesion characteristics,

anatomical site, clinical subtype, and, importantly, the presence and grade of epithelial dysplasia.

Tobacco exposure is an important modifiable risk factor for oral leukoplakia and other potentially malignant oral disorders. Both smoked and smokeless tobacco products have been associated with adverse oral mucosal changes and increased risk of oral malignancy. The timing and duration of exposure may be relevant because early

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initiation can result in prolonged cumulative exposure to tobacco-related carcinogens. However, the development of an individual lesion is multifactorial, and the presence of early tobacco exposure alone cannot establish a direct causal relationship between childhood tobacco use and epithelial dysplasia.

The present case involves a 25-year-old male who reported initiating tobacco use at approximately 10 years of age and subsequently developed biopsy-confirmed epithelial dysplasia within a persistent leukoplakic lesion. The unusually early reported age of tobacco initiation and the young age at presentation provide a clinically relevant context for discussing the potential consequences of prolonged tobacco exposure.

This case is also relevant to the broader regional context of adolescent tobacco use. Previous school-based research conducted by our group in the Mathura region of Uttar Pradesh has documented tobacco and arecanut use among adolescents and identified associated oral mucosal findings [1]. Additional studies from the same population have highlighted the potential contribution of peer and family influences to early initiation of smokeless tobacco use [2] and substantial exposure to tobacco-related marketing relative to preventive health messaging [3]. These findings provide epidemiological context for the early exposure history reported in the present case but do not establish that the patient's clinical condition resulted from the same population-level pathways.

Case Presentation

A 25-year-old male presented to our clinic with a persistent white lesion involving the lateral border of the tongue. The patient reported a history of tobacco use beginning at approximately 10 years of age and continuing thereafter. Details regarding the specific tobacco products used, frequency of use, cumulative exposure, and periods of cessation should be reported where available because these factors are relevant to assessment of tobacco-related risk.

Clinical Examination

Intraoral examination revealed a well-demarcated, white, leathery lesion involving the lateral border of the tongue that was non-scrapable on attempted removal. Examination of the adjacent dentition and oral tissues did not identify an obvious mechanical or frictional source, such as a sharp cusp, irregular restoration margin, or other apparent local irritant. No additional clinically evident mucosal lesions were identified at the time of presentation. Given the persistence and clinical characteristics of the lesion, together with the absence of an identifiable local traumatic cause, an incisional biopsy was performed for histopathological evaluation (Figure 1).

Investigations

Histopathological examination of the incisional biopsy demonstrated several epithelial, cytological, and connective tissue abnormalities. The epithelial changes included hyperkeratosis with orthokeratinization,



Figure 1. Intraoral Clinical Photograph Showing a Well-demarcated, White, Leathery Leukoplakic Lesion Involving the Lateral Border of the Tongue.

characterized by the absence of nuclei within the keratin layer, acanthosis, loss of basal-cell polarity, and premature keratinization, including individual-cell keratinization. The cytological abnormalities included nuclear hyperchromatism, cellular pleomorphism, an increased nuclear-to-cytoplasmic ratio, increased and atypical mitotic figures, and dyskeratosis. The connective tissue also showed chronic inflammatory cell infiltration and focal subepithelial fibrosis. Taken together, the clinical and histopathological findings were consistent with oral leukoplakia with epithelial dysplasia. The specific grade of dysplasia (mild, moderate, or severe) should be reported according to the final histopathological assessment and the applicable World Health Organization classification. Because the degree of epithelial dysplasia is an important factor in risk stratification and clinical management, the complete pathology report should be reviewed and the confirmed dysplasia grade documented where available.

Management

The patient was managed with a chemopreventive regimen consisting of lycopene 8 mg orally twice daily for 3 months, vitamin A (retinol) 25,000 IU orally once daily for 6–8 weeks, vitamin E 400 IU orally once daily, and topical tretinoin 0.05% gel applied to the lesion. The treatment regimen was selected in consideration of previously published studies evaluating chemopreventive approaches for oral leukoplakia. Clinical trials have reported improvements in the clinical and, in some studies, histological characteristics of leukoplakic lesions following treatment with agents such as lycopene and vitamin A [4, 5]. Topical retinoids, including tretinoin, have also been investigated for the management of oral leukoplakia and have demonstrated clinical responses in some controlled studies [6]. However, evidence that these interventions prevent malignant transformation remains limited. Therefore, the regimen should be considered an adjunctive management approach rather than a substitute for elimination of established risk factors or appropriate management of epithelial dysplasia. The patient received counselling regarding complete tobacco cessation, which represents a central component of management, and the importance of avoiding continued tobacco and arecanut

exposure was emphasized. Clinical follow-up was scheduled to assess treatment response and monitor the lesion for changes in size, surface characteristics, colour, induration, or other features that might warrant repeat biopsy or surgical management.

Discussion

This case is clinically notable because of the combination of a young age at presentation, a reported history of tobacco initiation during childhood, and biopsy-confirmed epithelial dysplasia within an oral leukoplakic lesion. The patient reported beginning tobacco use at approximately 10 years of age, representing approximately 15 years of exposure before presentation. Although this temporal relationship is clinically concerning, the development of oral epithelial dysplasia is multifactorial, and the case does not by itself establish a direct causal relationship between the age of tobacco initiation and the development of dysplasia.

The case nevertheless provides an important clinical perspective on the potential consequences of prolonged tobacco exposure beginning during childhood. This is particularly relevant in the context of our group's school-based research in the Mathura region, which has documented tobacco and arecanut use among adolescents and associated oral mucosal findings [1]. Additional studies from the same population have identified peer and family influences on early tobacco initiation [2] and substantial exposure to tobacco-related marketing relative to preventive health messaging [3]. These population-level findings provide contextual evidence for the early exposure history reported in the present case, although they cannot be used to establish that the same causal pathway occurred in this individual.

The histopathological findings in this patient are consistent with epithelial dysplasia, a clinically important feature of oral leukoplakia because dysplastic lesions generally carry a greater risk of malignant transformation than lesions without dysplasia. The degree of dysplasia, together with lesion site, size, clinical appearance, and other patient- and lesion-related factors, should therefore be considered when determining surveillance and treatment strategies. In the present case, documentation of the final dysplasia grade is particularly important for appropriate risk stratification.

Role of Chemoprevention

The use of chemopreventive agents in oral leukoplakia requires cautious interpretation. Individual clinical trials have reported regression or clinical and histological improvement with agents including lycopene, vitamin A, and retinoids [4–6]. However, improvement in lesion size or histological appearance does not necessarily demonstrate prevention of oral cancer.

Evidence syntheses have highlighted uncertainty regarding whether pharmacological interventions for oral leukoplakia reliably reduce the risk of malignant transformation. Accordingly, chemoprevention should not be presented as a definitive strategy for preventing

progression to oral cancer. In this patient, pharmacological treatment was used as an adjunct to risk-factor modification and clinical surveillance.

The most important modifiable intervention remains complete cessation of tobacco exposure. Continued exposure could potentially reduce the likelihood of sustained lesion regression and may contribute to the development of additional oral potentially malignant disorders. Appropriate follow-up is therefore essential, particularly given the presence of epithelial dysplasia.

Any increase in lesion size, development of induration, ulceration, changes in surface characteristics or colour, or other clinically suspicious features should prompt reassessment and consideration of repeat biopsy. The need for surgical excision should be determined according to the confirmed dysplasia grade, lesion characteristics, anatomical site, response to conservative management, and multidisciplinary clinical assessment.

Implications for Early Prevention

The case also highlights the potential importance of interventions occurring well before the development of clinically detectable oral disease. By the time this patient presented, tobacco exposure had reportedly continued for approximately 15 years and epithelial dysplasia had already developed. Consequently, clinical management at this stage represents secondary prevention rather than prevention of the initial exposure.

This distinction is particularly relevant to adolescent tobacco-control strategies. Research from the same region has identified early peer- and family-related pathways to tobacco and arecanut use, as well as substantial exposure to tobacco-related marketing [1–3]. These findings support the importance of prevention during early adolescence, before tobacco-use patterns become established.

At the population level, measures such as effective enforcement of age-related tobacco-sales restrictions, reduction of adolescents' exposure to tobacco marketing, improved identification of less conventional tobacco products such as tobacco-containing dentifrices, and rigorous evaluation of school-based prevention programs may help reduce early exposure. Such approaches should complement, rather than replace, clinical surveillance and management of individuals who already present with oral potentially malignant disorders.

In conclusion, this case describes biopsy-confirmed oral leukoplakia with epithelial dysplasia in a 25-year-old male who reported initiating tobacco use at approximately 10 years of age. The combination of early tobacco exposure, a persistent lateral-tongue lesion, and epithelial dysplasia emphasizes the importance of prompt clinical assessment and histopathological evaluation of persistent oral white lesions in young tobacco users.

Management should prioritize complete tobacco cessation, appropriate treatment based on the clinical and histopathological characteristics of the lesion, and structured long-term surveillance. Although chemopreventive agents such as lycopene, vitamin A, and topical retinoids have demonstrated clinical or histological responses in selected studies, their role in preventing

malignant transformation remains uncertain and should not replace definitive management or surveillance.

More broadly, the case illustrates the potential clinical relevance of preventing tobacco exposure during childhood and early adolescence. Population-level tobacco-control measures that reduce early access and exposure may have an important role in preventing the prolonged exposure trajectories that place young individuals at risk of tobacco-related oral disease.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and any accompanying clinical images.

Conflict of Interest

The authors declare that they have no conflict of interest.

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