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AN UNUSUAL PRESENTATION OF IRRITATION FIBROMA ASSOCIATED WITH MILD EPITHELIAL DYSPLASIA: A CASE REPORT

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ABSTRACT

Leukoplakia is the most common oral potentially malignant disorder, characterized by a predominantly white plaque that cannot be clinically or histopathologically classified as any other disease. It carries a variable risk of malignant transformation depending on its clinical and histopathological features. In contrast, irritation (traumatic) fibroma is a common benign reactive lesion of the oral soft tissues that develops secondary to chronic local irritation or trauma, such as plaque accumulation, calculus, sharp tooth edges, overhanging restorations, or ill-fitting prostheses. Although benign, these lesions may cause esthetic and functional impairment depending on their size and location. Irritation fibroma can occur at any age and may arise at virtually any oral soft-tissue site, including the buccal mucosa, tongue, lips, and gingiva. Among these sites, the buccal mucosa is the most frequently affected due to its susceptibility to repeated mechanical trauma. The present case report showed a 60 years old female patient with traumatic fibroma associated with dysplastic feature at right lateral border of tongue. After surgery post-operative healing was good without any recurrence.

KEYWORDS: Epithelial dysplasia, Traumatic fibroma, Ulcer.

INTRODUCTION

By definition, oral epithelial dysplasia (OED) is an epithelial tissue in which the prevalence of squamous cell carcinoma is more likely to appear than in its healthy counterpart.[1] In OED, normal maturation and stratification pattern of epithelium are replaced by cytological and/or architectural alterations. OED prevalence is infrequently reported.[2] In a retrospective study by Singh et al.[3], the mean preva lence of OED in the Indian population was 5.7%. According to the literature, the most common site of OED is the tongue and buccal mucosa. Habits such as smoking and alcohol consumption are regarded as risk factors for oral squamous cell carcinoma (OSCC), and therefore, for OED.[4] Traditionally, OED is graded into three cate gories by increasing severity: mild, moderate, and severe. A newer grading system categorizing OED into two grades, low and high risk, has been suggested in 2017 by the World Health Organization, and it aims to increase objectivity of histopathological grading.[5]

OED is a known risk factor for OSCC development, with a malignant transformation rate of 5% to 36% depending on the severity of dysplasia. In a meta-analysis by Mehanna et al.[6], the malignant transformation rate for mild to moderate oral epithelial squamous cell dysplasia was 10.3% and for severe oral epithelial squamous cell dysplasia 24.1%. Women seem to have a higher risk for malignant transformation despite OED lesions being less common in females. Several studies have evaluated how OED grading correlates with OSCC transformation. Results vary, from no correlation to a clear correlation. No consensus of the risk of malignant transformation based on histopathology has been reached.[7]

In addition to dysplastic changes themselves, OED often shows other histopathological features such as lichenoid inflammation, candidiasis, or ulcer. Two studies have reported lichenoid features in OEDs in almost one-third of samples. Lichenoid features in chronic inflammation related to OED complicate the diagnosis of dysplasia. Inflammation may cause a dysplastic feature described as

reactive epithelial atypia or dysplasia. Despite the presence of lichenoid inflammation in some OEDs, its dysplastic potential and as sociation with malignant potential remain unknown.[8]

CASE REPORT

A sixty year old female patient reported with pain in left lateral border of tongue since 2 months. There was no other relevant medical and family history. There was no history of any tobacco chewing or smoking. Dental history revealed extraction with respect to 36 without any post-operative complication. Extra oral examination showed no significant abnormality at maxillofacial region. Intraoral findings showed ulcerative lesion present on lateral border of left tongue measuring about 12mm antero-posteriorly and 9mm supero inferiorly. The margin of the lesion was elevated with well-defined border. Central portion of the lesion was ulcerated and have a high tendency for bleeding. **[Figure 1]** Overall oral hygiene was poor with multiple root stump at upper and lower arch. Sharp cusp was present to opposite of that ulcerated area. Based on the clinical finding a provisional diagnosis of traumatic ulcer was made. For final diagnosis incisional biopsy was performed under local anaesthesia after taking written approval from the patient. The incised tissue specimen measures about 9mm in length x 8mm in breadth x 4mm in height fixed in 10% buffer formalin. Under light microscope submitted Haematoxylin & Eosin stained soft tissue sections shows parakeratinized stratified squamous surface epithelium with broad and blunt rete ridges overlying a fibro-cellular connective tissue stroma along with an ulcerated area. This epithelium shows features of dysplasia like basal cell hyperplasia, loss of polarity of the basal cells, nuclear hyperchromatism, altered nuclear-cytoplasmic ratio, increased mitotic activity and loss of cellular adhesion on the lower 1/3rd of epithelium. Epithelium connective tissue junction is intact. This connective tissue stroma shows loosely arranged collagen fibers in association with fibroblasts, dense and diffuse chronic inflammatory cells infiltrate predominantly consisting of lymphocytes and plasma cells in association with endothelial lined blood vessels filled with RBCs. **[Figure 2 & 3]** Based on histological findings the final diagnosis was made as Traumatic ulcer with Mild Oral Epithelial Dysplasia. Smoothening of the cusp at upper arch and root stump extraction at lower arch was performed. Tropical application of anti-inflammatory gel was prescribed. After 15 days of follow-up showed improvement of the lesion with healing of the lesion.

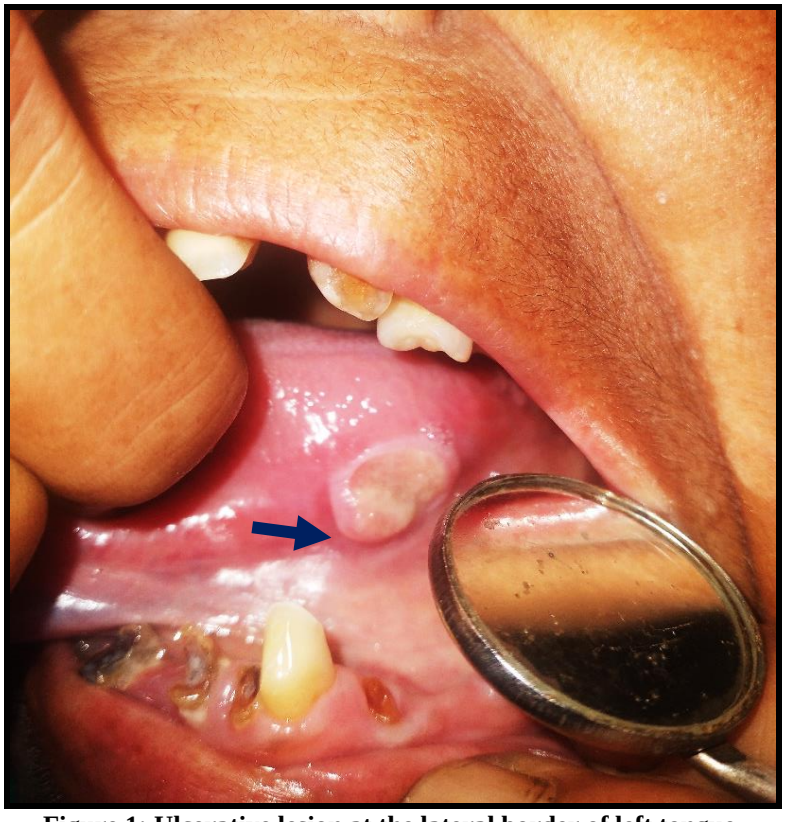

Figure 1: Ulcerative lesion at the lateral border of left tongue.

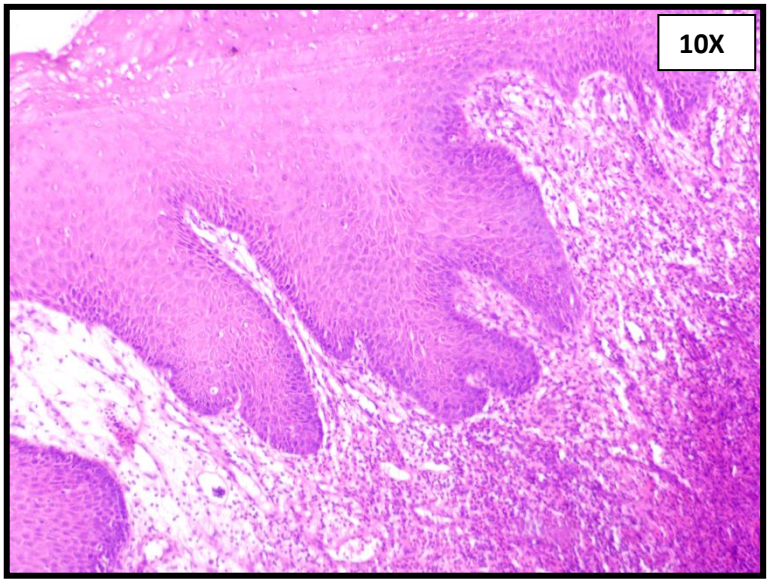

Figure 2: Photomicrograph showed broad rete ridges with inflammatory cells infiltration.

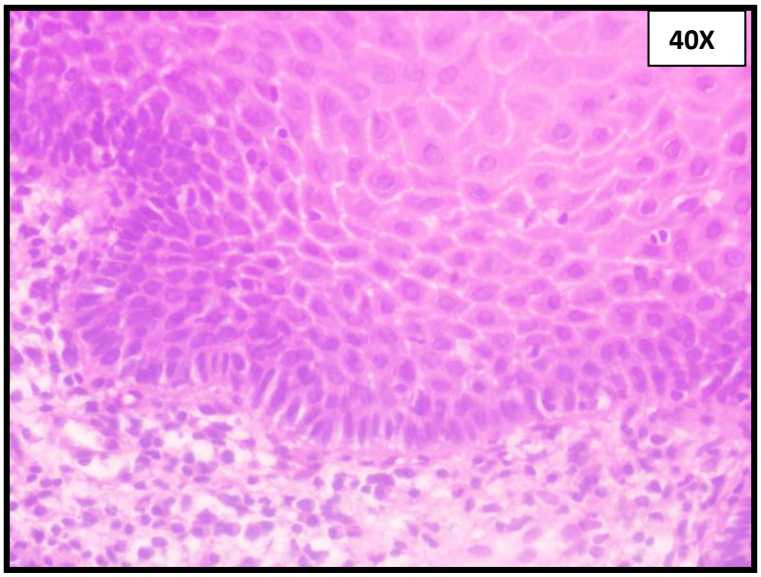

Figure 3: Photomicrograph showed dysplasia at basal one third of epithelium.

DISCUSSION

Fibroma is a reactive lesion that develops as a result of chronic irritation or trauma, leading to a prolonged reparative process characterized by granulation tissue formation and subsequent fibrosis, ultimately producing a localized fibrous submucosal mass. A study conducted by Santiago Torres-Domingo et al. in 2008 evaluated the prevalence and characteristics of common benign oral mucosal tumors in 300 patients. Of these, 153 cases (53.3%) were histopathologically diagnosed as fibroma, demonstrating that fibroma was the most frequently occurring benign tumor of the oral cavity.[9]

Prognostication of oral epithelial dysplasia (OED) remains challenging because its assessment is largely subjective and reliable predictive biomarkers are lacking. Although several grading systems have been proposed, none adequately incorporate the etiological factors or the host inflammatory response associated with the lesion. This conventional approach is largely based on the assumption that OED and oral squamous cell carcinoma (OSCC) represent sequential stages of chemical carcinogenesis.[10]

In 1863, Virchow supported the earlier hypothesis proposed by Galen, suggesting a link between chronic inflammation and carcinogenesis. Later, Dvorak demonstrated that tumor tissues closely resemble healing wounds, as both contain similar stromal cell populations and exhibit comparable tissue responses, leading to the concept that tumors are "wounds that do not heal." In 1970, Prehn further advanced the theory of inflammation-associated carcinogenesis after observing the development of malignant tumors in individuals without exposure to conventional risk factors.[6]

The reported malignant transformation rate of oral epithelial dysplasia (OED) ranges from 1.4% to 36% in

both habitués and non-habitués. Notably, non-smokers have been reported to be 7.1 times more likely to experience disease progression and malignant transformation than smokers. In non-habitués, oral potentially malignant disorders (OPMDs) may arise secondary to chronic trauma, immune dysregulation, or persistent inflammation rather than traditional carcinogenic exposures.[9]

Clinical assessment remains a key component in predicting malignant potential. Lesions exhibiting surface alterations, such as a granular or verruciform appearance, demonstrate a malignant transformation rate of approximately 4%–15%, whereas homogeneous, thick leukoplakias show a comparatively lower transformation rate of 1%–7%. Therefore, clinicians should maintain a high index of suspicion for OPMDs in both habitués and non-habitués when lesions exhibit changes in surface texture, color, size, contour, mobility, or loss of surface integrity involving intraoral or extraoral tissues.[11]

Data comparing habit-associated and non-habit-associated oral epithelial dysplasia (OED) remain limited. However, several studies suggest that OED in non-habitués carries a greater risk of malignant transformation. Rock et al. (2018) reported that non-smokers were approximately twice as likely to develop malignant transformation compared with smokers, and the progression from OED to oral squamous cell carcinoma occurred more rapidly in non-smokers. Similarly, Jaber (2010) observed a higher rate of malignant transformation among non-users of tobacco, although the long-term outcomes of users and non-users were comparable.[3]

The absence of established risk factors, such as smoked or smokeless tobacco use, in non-habitués suggests that distinct molecular and genetic mechanisms may contribute to carcinogenesis in this population. In these individuals, chronic trauma or persistent irritation may act as initiating stimuli, eliciting early epithelial responses including hyperplasia and hyperkeratosis, while acute and chronic inflammation constitute the primary stromal responses. Persistent inflammation activates nuclear factor-kappa B (NF-κB) signaling and promotes the release of pro-inflammatory cytokines, including interleukin (IL)-1, IL-4, IL-6, and tumor necrosis factor-alpha (TNF-α). These mediators enhance oxidative stress, leading to DNA adduct formation, nucleotide transversions, impaired DNA repair, and genomic instability. In addition, activation of key signaling pathways, such as the non-canonical transforming growth factor-beta (TGF-β), mitogen-activated protein kinase (MAPK), and phosphatidylinositol 3-kinase/protein kinase B (PI3K/Akt) pathways, promotes abnormal cell proliferation, architectural disorganization, and cytological atypia, which are recognized histopathologically as the characteristic features of oral epithelial dysplasia.[7]

Inflammation-associated carcinogenesis has been proposed to explain the development of malignancy in individuals without exposure to conventional carcinogens. Chronic inflammation promotes the recruitment of immune cells and the generation of reactive oxygen and nitrogen species, creating a state of persistent oxidative stress. This oxidative environment induces DNA damage, genetic mutations, and genomic instability. Furthermore, failure of DNA repair mechanisms to accurately correct replication errors further contributes to carcinogenesis.[8]

Ho et al. (2012) identified several factors associated with an increased risk of malignant transformation, including idiopathic leukoplakia in non-smokers, female sex, and higher grades of epithelial dysplasia. These findings suggest that non-habit-associated oral epithelial dysplasia (OED) may possess a distinct biological behavior with a greater propensity for malignant progression.[4]

Clinical intervention during the stage of mild epithelial dysplasia has been reported to achieve favorable outcomes, whereas the management of moderate and severe dysplasia remains less predictable. Although no universally accepted treatment protocol for OED currently exists, it is essential to investigate management strategies tailored to both habit-associated and non-habit-associated lesions. Such an approach is particularly important because OED in non-habitués has demonstrated a higher risk of malignant transformation, despite habitués having a greater overall prevalence of oral potentially malignant disorders (OPMDs).[8]

CONCLUSION

Oral epithelial dysplasia commonly presents as white patches (leukoplakia) on the oral mucosa, with an overall malignant transformation rate of approximately 5%. Histopathological detection of epithelial dysplasia remains the gold standard for assessing malignant potential, although diagnosis is subjective despite WHO grading criteria. Only about 50% of clinically diagnosed leukoplakias exhibit dysplasia on biopsy. The risk of progression increases with dysplasia severity, reaching approximately 50% for severe, 30% for moderate, and 5% for mild dysplasia.

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