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ORIGINAL ARTICLE

Oral Frictional Hyperkeratosis: A brief review

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Abstract

Introduction: Among various white oral lesions, frictional keratosis is a commonly reported condition. Thus, it is essential for clinicians and pathologists to be familiar with the diagnosis and proper management of this lesion to ensure optimal patient care and outcomes.

Methods: This review analysed full-length papers gathered from PubMed and PubMed Central (PMC) from 2015 to 2025 regarding the demographic information, clinical features, and histopathological findings of oral frictional keratosis.

Results: A total of 56 articles were initially identified and evaluated by the authors; those aligned with the specific scope of the review were selected for further analysis. Finally, demographic information, clinical findings, and histopathological data were extracted from the included studies.

Conclusion: When addressing oral white lesions, the primary step is to identify and eliminate the underlying cause while allowing adequate time for the lesion to heal. If the lesion persists or worsens despite removing the etiological factor, a biopsy should be performed. Furthermore, if the cause remains unclear or if the keratotic lesion is situated in a high-risk area for malignancy, a biopsy is crucial to ensure an accurate diagnosis and appropriate management.

Key words: Leukoplakia; Oral keratosis; Biopsy; Mouth, Frictional keratosis.

Introduction

White lesions in the oral cavity are frequent clinical findings that can manifest with various appearances and arise from a wide array of underlying causes. These lesions can be attributed to both benign and malignant conditions (1). Oral white lesions are categorized into several major groups: developmental (e.g., White sponge nevus, Dyskeratosis congenita), reactive (e.g., Traumatic lesions/keratoses, Morsicatio mucosae oris, Benign alveolar ridge keratosis), infectious (e.g., candidiasis, Oral secondary syphilis), Immune-mediated and autoimmune (e.g., Idiopathic oral lichen planus, Lupus erythematosus), Metabolic (e.g., Uremic stomatitis, Migratory glossitis) and Preneoplastic and neoplastic lesions (e.g., Leukoplakia, Oral squamous cell carcinoma) (2-7).

Among these, frictional keratosis is a highly prevalent lesion (1). However, since a subset of white lesions is categorized as oral potentially malignant disorders (OPMDs), which are difficult to distinguish from benign conditions a careful and thorough evaluation is critical due to the increased risk of progression to oral squamous cell carcinoma (OSCC) (8-11). Specifically, OPMDs comprise a heterogeneous group

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of disorders with varying causes, most commonly associated with tobacco use (12). These changes may or may not be accompanied by clinical or histomorphological alterations. Yet, they pose a significant risk for the development of OSCC Examples of OPMDs include leukoplakia, erythroplakia, Oral submucous fibrosis (OSMF), lichen planus, actinic keratosis, discoid lupus erythematosus, and palatal lesions seen in reverse smokers (13-15). Thus, clinicians must remain vigilant in the assessment and management of white lesions, as early diagnosis and intervention can significantly influence patient outcomes (16). Understanding the characteristics, etiologies, and potential implications of oral white lesions is essential for effective treatment and patient care (1,17,18).

A biopsy remains the gold standard for the definitive diagnosis of oral white lesions. This essential procedure allows healthcare professionals to accurately distinguish between various types of lesions, including reactive, infectious, and benign conditions, as well as potentially malignant or malignant pathologies. However, enhancing familiarity with the clinical presentation of these lesions can substantially minimize the reliance on biopsy procedures. Improved clinical recognition enables clinicians to initially pursue conservative management, such as targeted topical medications, lifestyle modifications, and improved oral hygiene. When such conservative approaches fail to resolve the lesion or when there is a lack of improvement, a biopsy becomes essential. This strategic progression not only enables precise identification of the underlying condition but also allows for a tailored management plan that meets the specific needs of the patient (19).

This review aims to comprehensively identify and analyze the clinical and histopathological characteristics associated with frictional keratosis, a commonly observed oral white lesion. By examining findings from various studies, this review seeks to enhance the diagnostic accuracy of clinicians and pathologists regarding this specific lesion. Key aspects explored include clinical variations, underlying etiologies, histological features, and practical implications for treatment, ultimately facilitating improved patient care and outcomes.

Method

A literature search of the PubMed database was conducted to retrieve full-length articles published between 2015 and 2025. The search utilized the keywords ‘oral reactive,’ ‘factitial,’ and ‘frictional hyperkeratoses.’ Inclusion criteria were strictly limited to articles with full texts available in PubMed and PubMed Central (PMC). A total of 56 articles were initially identified and evaluated by the authors; those aligning with the specific scope of the review were selected for further analysis. Finally, demographic information, clinical findings, and histopathological data were extracted from the included studies.

Discussion

This review analysed studies regarding keratotic lesions of the oral cavity, with a primary focus on frictional keratosis, to identify effective clinical management strategies. The differential diagnosis of oral white lesions presents significant challenges due to their diverse clinical manifestations, which frequently overlap with other conditions, including OPMDs (1-7).

Prevalence

Müller (1) noted that the true prevalence of frictional keratosis remains unknown because epidemiological studies of oral mucosal lesions typically rely solely on clinical evaluations. This reliance frequently leads to the misclassification of leukoplakia as frictional

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keratosis, or vice versa. However, in a study investigating the prevalence of white lesions among dental patients in India, Nautiyal et al. (20) found that 71.72%of the identified lesions were classified as frictional keratosis

Etiology

Parafunctional habits, such as constant rubbing, chewing, or sucking of the oral mucosa against the teeth, can lead to keratosis of the lips, buccal mucosa (known as Morsicatio buccarum), and the tongue (Morsicatio linguarum) with various clinical appearance based on the extent of trauma (1). Shiragur et al. (21) in 2024 state that frictional keratosis is frequently initiated by mechanical friction from the teeth on the buccal mucosa at the occlusal level or on the dorsal surface of the tongue, mostly occurring in adult females, particularly those diagnosed with temporomandibular joint dysfunction. Nautiyal et al. (20) believe the etiology is different in relation to the location of the lesion. Sharp tooth edges, vigorous tooth brushing, and irritation from dental appliances, such as retainers or dentures, have been proposed as etiologic factors for frictional keratosis by authors emphasizing the need for careful monitoring of oral hygiene practices and dental health. In 2020, Almazyad et al. (22) conducted a study on benign alveolar ridge keratosis, characterizing it as a specific variant of reactive hyperkeratosis that develops as a result of chronic trauma or friction applied to the oral mucosa. Thompson et al. (24) also believes chronic mechanical irritation and trauma, as mentioned by other studies, habitual biting, aggressive tooth brushing, and other forms of abrasion related to dental appliances such as orthodontic devices and dentures are the underlying etiologic factors of frictional hyperkeratosis. Additionally, these authors believe parasitic infections causing bruxism can also contribute to the development of this condition.

Clinical features

Clinically, frictional keratosis presents as ill-defined areas of gray or white non-scrapable patches, papules, and plaques exhibiting varying degrees of ulceration and shredded keratin. The reported locations for these lesions include the cheek, lips, alveolar ridge, retromolar pad, and facial attached gingiva (1,22,23). In some patients, frictional keratoses can be extensive, affecting the entire cheek and extending to the lips (1). Woo and Lin (2) noted that the clinical appearance of frictional keratosis can vary depending on the extent of trauma, presenting unilaterally or bilaterally with varying color intensity and thickness. Occasionally, the lesional surface may feel rough with shredded keratin and peeling, indicating a cycle wherein the patient attempts to remove the rough tags with their teeth, subsequently resulting in the formation of additional tags.

Some authors believe this lesion is seen most commonly in women, some in males (20, 21). In the study conducted by Almazyad et al. (22) 67.1% of benign alveolar ridge keratosis occurred in males, with a median age of 56 years (ranging from 15 to 86 years). Woo and Lin (2) reviewed 584 cases of clinical leukoplakia and found that cases associated with frictional keratosis were predominantly in patients during their fifth and sixth decades of life.

Microscopic findings

Elongated rete ridges, acanthosis, epithelial hyperplasia, hyperparakeratosis, and significant intracellular edema with surface microbial colonization are microscopic features observed primarily in frictional keratosis (21). In the study by Almazyad et al. (22) hyperkeratosis, often accompanied by wedge-shaped hypergranulosis and occasional focal parakeratosis, surface corrugation, and tapered rete ridges that were often interconnected at their tips were reported.

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Diagnosis, treatment, and prognosis

These lesions typically resolve following the cessation of the causative habit (1, 11). Generally, clinical findings are sufficient to determine the diagnosis and etiology of these white lesions, often eliminating the need for a confirmatory biopsy. However, if the cause is unclear, or if the keratotic lesion is located in a high-risk area for OPMDs, such as the lateral border of the tongue, a biopsy is advisable to rule out malignancy (11). Special attention is required for lesions with poorly defined borders and a rough, irregular surface texture, which might signal a heightened risk for developing an OPMD (20). If there is no noticeable clinical improvement within four weeks after the removal of identified irritants, the diagnosis of frictional keratosis should be re-evaluated (21).

Benign alveolar ridge keratosis is particularly prevalent among edentulous individuals, as the absence of dental structures may lead to increased mechanical irritation in the affected areas. Immunohistochemical evaluation of P53 in these lesions has demonstrated less than 25% nuclear positivity, supporting the conclusion that benign alveolar ridge keratosis is a distinct, benign clinicopathologic entity caused by friction that must be clearly distinguished from true leukoplakia(22). Furthermore, if a lesion persists and fails to regress after eliminating the frictional source, the clinical designation of “leukoplakia” should be applied (23).

Conclusion

Keratotic lesions are common oral anomalies categorized into reactive conditions (such as frictional/factitial hyperkeratosis and benign alveolar ridge keratosis), keratinizing epithelial dysplasia, and other entities such as lichen planus, candidiasis, and white sponge nevus (25). Frictional keratosis is a strictly benign condition; its primary management involves protecting the oral mucosa from the causative chronic frictional trauma (26).

Clinical presentation varies with the severity of trauma; while it can manifest at multiple anatomical sites, it most frequently occurs on the buccal mucosa (27). The condition typically results from chronic mechanical irritation secondary to habitual biting, aggressive toothbrushing, or abrasion from dental appliances such as orthodontic devices and dentures. Furthermore, bruxism often linked to temporomandibular joint (TMJ) dysfunction or parasitic infections, particularly in females plays a significant role in its development. Initial management requires identifying and eliminating the etiological factor, allowing sufficient time for clinical resolution. However, biopsy is indicated if the etiology remains undetermined, the lesion persists or worsens despite removing the suspected cause, or the keratosis is located in a high-risk area for malignancy (24).

While histopathological features vary by anatomical site, common findings include marked hyperkeratosis (frequently accompanied by microbial colonies), epithelial hyperplasia, and intracellular edema. Inflammation is generally absent in the superficial connective tissue unless secondary ulceration is present. Normal mitotic figures may appear within the basal or parabasal layers, but features indicative of epithelial dysplasia are strictly absent (24, 25, 28).

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