ISSN: 2581-5989
PubMed - National Library of Medicine - ID: 101738774

International Journal of Dental Science and Innovative Research (IJDSIR)

IJDSIR : Dental Publication Service

Available Online at: www.ijdsir.com

Volume – 4, Issue – 4, August - 2021, Page No. : 297 - 303

Oral keratotic lesions in children

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Citation of this Article: Shreya Shrivastava, Ankur Jain, Rinky Sisodia, Satish Maran, Anaya Kale, Krishna Sagar, "Oral keratotic lesions in children", IJDSIR- August - 2021, Vol. – 4, Issue - 4, P. No. 297 – 303.

Type of Publication: Case Report

Conflicts of Interest: Nil

Abstract

Background: White lesions of the oral cavity are quite common and can have a variety of etiologies, both benign and malignant. Although the vast majority of publications focus on leukoplakia and other potentially malignant lesions, most oral lesions that appear white are benign. This review will focus exclusively on reactive white oral lesions, aimly frictional keratosis its types and its various causes and sites. Keywords Leukoplakia · Frictional keratosis can be found in any children with a variety of causes, how to differentiate between different keratosis and their eradication or treatment is discussed brefly in this review.

Conclusion: Keratotic lesions are benign lesions which can be scraped of easily or can be treated by removing the irritating factor or the causative agent for it.

Keywords: Frictional keratosis, leucoplakia morsicatio mucosae oris, breastfeeding keratosis

Introduction

Oral lesions can be classified into four groups comprising of ulcerations, pigmentations, exophytic lesions, and red-

white lesions [1]. Although white lesions constitute only 5% of oral pathoses, some of these lesions such as leukoplakia, lichen planus, and proliferative verrucous leukoplakia have malignant potential as high as 0.5–100%[2]. Therefore, white lesions mandate an appropriate clinical diagnostic approach to exclude the possibility of malignancy. White mucosal lesions may result from thickening of one or several layers of the oral epithelium. They vary in size and depth, generally have an irregular outline, and may be solitary or multifocal. Common sites are the buccal mucosa, lateral border of the tongue, floor of the mouth, and hard palate. The remainder of the tongue, soft palate, lips, and gingiva are less often involved [3].

Keratin is a fibrous protein produced by the body, when an excessive growth is reported in this protein its results in hyperkeratosis or simply keratosis. It may happen for a number of reasons, and may be genetic (runs in the family), physiological e.g. due to friction from a sharp tooth, pre-malignant (pre-cancerous) and malignant (cancerous). The change may result from chemical, heat or physical irritants.

Hyperkeratosis (focal keratosis)

is a microscopic term meaning increased thickness of the keratin layer of stratified squamous epithelium with no microscopic evidence of atypical epithelial cells Clinically, hyperkeratotic lesions appear as white, rough, non-painful patches that do not rub off. They are often secondary to chronic irritation, such as biting or tobacco use.

Friction (the constant rubbing of two surfaces against each other) in the mouth may result in benign (non-cancerous) white patches also known as frictional keratosis (FK)[4].

Various names have been used to describe particular examples of FK, including those resulting from excessive tooth-brushing force (toothbrush keratosis), the constant rubbing of the tongue against the teeth (tongue thrust keratosis)[4].Such keratotic lesions when occurred on specific sites have been referred to as “morsicatio buccarum” when it occurs on the buccal mucosa, “morsicatio labiorum” when on the labial mucosa and “morsicatio linguarum” when it is on the lateral borders of the tongue. These lesions have been referred to as “pathominia mucosae oris,” “morsicatio mucosae oris,” “morsicatio buccarum,” or “morsicatio labiarum,” depending on the location (morsus bite). These injuries will be referred to as “morsicatio mucosae oris” or MMO [5].

Epidemiology

The true prevalence of frictional keratosis is unknown as studies that review oral mucosal lesions are generally clinically based and may mis-categorize leukoplakia as frictional keratosis or vice versa. In some published series in children and adolescents the reported range is 0.26–5.3% [6]. White lesions near rough dental restorations, a sharp tooth, or due to biting because of unsuitable prosthesis were registered as frictional keratosis.[3] The occurrence of frictional keratosis was in 5.79% of all subjects [7]. MMO has been reported to occur in young patients in the second and third decade. The prevalence has been estimated at 6% in those under age 12 with an equal gender distribution, 4.6% among reform school students aged 12 to 24 (most were under age 20), 1.7% among the general population, and 1.8% among patients aged 15 to 19 years [5].

Mortality/morbidity

Frictional keratosis and its variants do not cause symptoms and are benign mucosal lesions that remain localized with no associated mortality or morbidity.

Race - No racial predilection seems apparent for oral frictional keratosis.

Sex - In general, frictional keratosis has no known sex predilection, except for cheek biting and lip biting, which are twice as prevalent in women compared with men.

Age - Oral frictional keratosis affects persons from a wide range of ages, and contributing factors determine which age group is more commonly affected. In general, oral frictional keratosis lesions are more common in adults[4].

Pathophysiology

The white patches of frictional keratosis that develop in the oral cavity represent a chronic, low-grade, mechanical process that is analogous to the formation of a callus on the skin. The most common local factors involved in this process are tissue chewing (mainly on the buccal mucosa or lips), ill-fitting or irregularly surfaced removable dental prostheses (dentures), fractured or malposed teeth, poorly adapted dental restorations, orthodontic appliances, improper toothbrushing, and constant mastication on edentulous alveolar ridges. The constant irritation stimulates the production of excessive keratin, with a subsequent change in the thickness and the color of the involved mucosa [4].

Parafunctional habits whereby there is constant rubbing, chewing or sucking of the oral mucosa against the teeth can result in keratosis of the buccal mucosa (morsicatio buccarum), tongue (morsicatio linguarum) and lip. Chronic biting (nibbling) of the buccal mucosa often leads to loose threads such as keratin shreds, tissue tags or desquamative areas on the mucosal surface [7].A newer form of keratosis recently reported is breastfeeding keratosis

Breastfeeding keratosis

Focal regions of thickened labial mucosa are not uncommon in breastfeeding infants, but these appear more like pale pink “pads” than white keratotic, leukoplakia-like plaques. This can occur due to sucking habit developed for breastfeeding, with an unusually active lip sucking habit while breast feeding and especially between feeding sessions. This habit became less pronounced over time, and with cessation of the habit the lip lesion disappeared. We have called this type of frictional keratosis “breastfeeding keratosis,” but we assume that the lesion cannot develop without exuberant lip sucking activity between feedings.

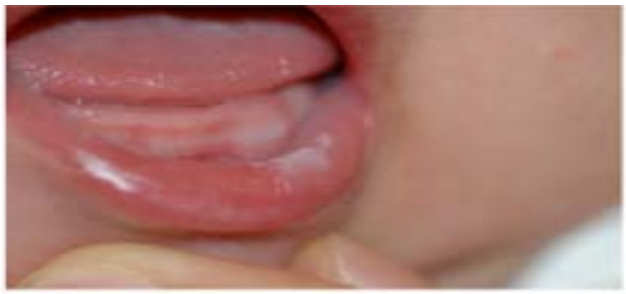

FIGURE 1
White, moderately well demarcated keratotic plaque of the lower left lip mucosa had a roughened surface and could not be scraped off.

The lesion is, of course, innocuous, and we believe it will always disappear as the sucking habit becomes less pronounced, just as other forms of adult-onset frictional keratosis disappear when the offending tooth or denture edge is made less traumatic. The major problem, probably the only problem, is mistaking the keratosis for thrush and treating accordingly without success, leading to the potential use of hepatotoxic systemic antifungal agents that have not been approved for neonatal infants. The recognition of the proper diagnosis will prevent this situation from occurring.

Active sucking during and between breastfeeding can produce a transient frictional keratosis of the lip mucosa. This lesion requires no treatment and should be differentiated from the more common sloughing white plaque of infancy, thrush. We propose the diagnostic term “breastfeeding keratosis” for this entity.[8]

Clinical features

These lesions are sometimes distinctive enough for a diagnosis based on clinical features alone. They present as

whitish gray papules and plaques on the buccal mucosa and labial mucosa (usually lower), often associated with leukoedema and a macerated appearance; 67% to 72% are bilateral.[11,12] Loose thread-like keratin shreds, tissue tags, or desquamative areas are often seen on the surface, and there may be ulcers and erosions. Lesions are evanescent and may resolve and recur.[5,6] Treatment with protective screening devices is of limited value.1 In some cases, the surface keratin may be peeled off, leaving behind normal-appearing mucosa, unlike candidiasis or vesiculobullous conditions. Their diffuse, poorly demarcated, peeling, thready appearance usually makes the clinical diagnosis straightforward. However, MMO may sometimes appear as distinct, well-demarcated plaques.[7]

Histological features

The histologic features of frictional keratosis from the tongue, lip or buccal mucosa vary slightly depending on the site of the biopsy. With few exceptions, marked hyperparakeratosis with a shaggy or shredded keratin surface is noted. Corresponding to the clinical presentation, the surface keratin can have a macerated appearance with fissures and clefting[7]. Bacteria is usually present on the keratin surface in biopsies from the tongue, but not as often on the buccal mucosa or lip. Although candidal hyphae may be present this is uncommon and unrelated to the underlying etiology. The epithelium exhibits epithelial hyperplasia and intracellular edema is common presenting as ballooned cells in the spinous layer. Generally, there is a lack of inflammation in the superficial connective tissue with the exception of cases where secondary ulceration is present. In these instances, normal mitotic figures may be present in the basal or parabasal layer, but the features of epithelial dysplasia are absent.[14]

Diagnosis

Diagnosing MMO as merely "hyperortho- or –parakeratosis with acanthosis" (referred to in many pathology circles as the histologic sign-out), although histologically correct, does not provide an accurate interpretation of this condition because this is the same histologic sign-out offered for nondysplastic leukoplakias. MMO has much more specific histology than nondysplastic leukoplakias. Smokeless tobacco keratosis for example, also exhibits "hyperorthoor –parakeratosis and acanthosis," and although this is histologically correct, it is more accurate and helpful to the clinician to diagnose and sign it out as smokeless tobacco keratosis.[15]

Table 1

Entity	Age	Gender	Common Location(s)	Clinical Features	Treatment	Premalignant
Frictional keratosis	NA	NA	NA	white plaque with rough and frayed surface	removal of irritants	NA
Oral leukoplakia	>50 years	M	buccal mucosa, lip vermilion and gingivae	white patch or plaque	NA	potentially malignant lesion
OHL	NA	M	borders of the tongue unilaterally or bilaterally	from slight, white vertical bands to thickened, furrowed areas with a shaggy surface	systemic anti-herpes virus drugs, topical retinoids or podophyllum resin, combination therapy with acyclovir cream and podophyllumresin, gentian violet, surgical excision or cryotherapy	no potential for malignant transformation

Entity	Age	Gender	Common Location(s)	Clinical Features	Treatment	Premalignant
PVL	mean age: 60 years	F	Gingivae	non-homogeneous multifocal areas with speckled and rough surface in the form of exophytic, wart-like, verrucous, polypoid projections or erythematous components	surgery, carbon dioxide laser ablation, topical photodynamic therapy, oral retinoids, topical bleomycin solution, beta-carotene, methisoprinol (a synthetic antiviral agent), radiation, chemotherapy	malignant transformation
OSCC	>65 years	M	floor of the mouth, posterior lateral borders and ventral surface of the tongue	red, white, or combined red-and-white lesion; alteration of surface texture into granular, rough, fungating, papillary, and verruciform or crusted lesion; or existence of a mass or irregular ulceration with rolled border and induration on palpation.	radiation therapy or combined chemo radiation therapy with or without surgery	NA
Verrucous carcinoma	elderly	M	mandibular vestibule, buccal mucosa, gingivae, tongue, and hard palate	asymptomatic, diffuse, well demarcated, thick white plaque with papillary or verruciform projections	NA	NA
Nicotinic stomatitis	>45	M	Palate	diffuse leathery grayish-white palatal plaque with red points, “dried mud” appearance	regression after cessation of smoking	not a premalignant condition
Actinic cheilitis	old age	M	lower lip vermilion	dryness, swelling, cracks, atrophic regions, crusting regions, keratotic plaques, chronic ulceration	Surgery, cryotherapy, electrosurgery, topical retinoids, 5-flurouracil cream, photodynamic therapy, CO_2 laser ablation and vermilionectomy	Premalignant condition
Chronic mucocutaneous candidiasis	begins during infancy	NA	nails, skin, oral and genital mucosae	chronic whitish plaques, along with crusts and ulcers	antifungal therapy	NA
chronic hyperplastic candidiasis (Candidal leukoplakia)	over 50	NA	retro commisures bilaterally, tongue, palate and lips	white patches or plaques	antifungal therapy, topical retinoids, betacarotene, bleomyin, several surgical techniques	NA

Differential diagnosis

Leukoedema

Linea alba

Smokeless tobacco keratosis

Cinnamon contact stomatitis

Lupus erythematosus

Treatment

The lesion subsides after removal of the causative factor. In case of doubt, when removal of the causing habit does not result. In clinical improvement, a biopsy should be performed[15]

Outcome

The treatment results are excellent with no malignant potential.

Discussion

Frictional (traumatic) keratosis is defined as white plaques with a rough and frayed surface clearly related to an identifiable source of mechanical irritation. These lesions can occasionally mimic dysplastic leukoplakia.

Morsicatio originates from the Latin word morsus, meaning “bite”, which also called morsicatio mucosa oris or chronic mucosal chewing.[7] This lesion is caused by self-induced injury and chronic tissue irritation like habitual chewing of buccal mucosa, chronic nibbling, biting, or sucking.

Most patients deny the self-inflicted injury or do it subconsciously. Habitual biting of cheeks and the lower lip. Most patients deny the self-inflicted injury or do it subconsciously. Glass blowers develop similar changes in their buccal mucosa due to chronic irritation as well.

The clinical features include asymptomatic shaggy and thickened macerated gray-white patches or plaques with keratin shreds, tissue tags, or desquamated areas on the mucosal surface, which gradually merge the adjacent mucosa[16.17.18]. A peeling irregular ragged surface with erythema or erosion—but not ulceration—might accompany white areas, and the patient may report the ability to peel shreds of the white portion from the periphery of the lesions[17.18,19]. If the clinical presentation of morsicatio is typical and history taking reveals patients’ habit of mucosal biting the diagnosis will be established. In case of any doubt, a biopsy seems to be necessary[17.18.19]. This condition has no long-term negative consequences and generally no treatment is recommended. As morsicatio usually occurs subconsciously, the patients should give consultation for their parafunctional.

Sometimes morsicatio might appear as unilateral lip and/or tongue lesions[16]. The clinical features include asymptomatic shaggy and thickened macerated gray-white patches or plaques with keratin shreds, tissue tags, or desquamated areas on the mucosal surface, which gradually merge the adjacent mucosa[18,19,20]. A peeling irregular ragged surface with erythema or erosion—but not ulceration—might accompany white areas, and the patient may report the ability to peel shreds of the white portion from the periphery of the lesions[17,18,19]. If the clinical presentation of morsicatio is typical and history taking reveals patients’ habit of mucosal biting the diagnosis will be established. In case of any doubt, a biopsy seems to be necessary[1718,19]. This condition has no long-term negative consequences and generally no treatment is recommended. As morsicatio usually occurs subconsciously, the patients should give consultation for their parafunctional behavior to resolve it. Nonetheless in some patients whose chewing habit is difficult to quit use of night guard has been suggested to eliminate the injury of adjacent teeth to oral mucosa[17,18, 20].

Conclusion

Keratotic lesions are benign lesions which can be scraped of easily or can be treated by removing the irritating factor or the causative agent for it.

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