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White Lesions of the Oral Cavity: A Review of Literature

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Abstract:
White lesions in the oral cavity may be benign, premalignant or malignant. The oral white lesions can be acquired or congenital. Some of these lesions are leukoplakia, lichen planus, and proliferative verrucous leukoplakia which has high malignant potential.Leukoplakia is the most frequent precancerous lesion of the mouth. Oral candidiasis is the commonest human fungal infection. Early diagnosis can minimize the progression of oral cancer.

Keywords: ***White lesions, Oral leukoplakia, Oral candidiasis, Lichen planus.***

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I. Introduction:

Oral lesions are classified into four groups.They are ulcerations, pigmentations,exophytic lesions, and red-white lesions.The white lesions consists of only 5% of oral pathoses,some of these lesions such as leukoplakia, lichen planus, and proliferative verrucous leukoplakia which has high malignant potential 0.5–100%. The oral white lesions can be acquired or congenital.Oral white lesions are caused by a thickened keratotic layer or an accumulation caused by fungal infections or caustic chemicals. White lesions can be attributed to increased thickness of keratin layer, which is induced by local irritation, immunologic reactions, premalignant or malignant transformation. [18]

Classification of White Lesions:

Variation in structure and appearance of normal mucosa:

1.Leukoedema
2.Fordyce's granules
3.Linea alba

White lesion with definite precancerous potential:

1.Leukoplakia
2.Erythroplakia
3.Tobacco keratosis, actinic keratosis
4.Lesion associated with electrogalvanism
5.Carcinoma in situ
6.Verrucous carcinoma
7.Lichen planus
8.Lichenoid reaction
9.Oral submucous fibrosis
10.Dyskeratosis congenita
11.Lupus erythematosus
12.Acanthosis nigricans

White lesion without precancerous potential:

1.Traumatic keratosis
2.Focal epithelial hyperplasia
3.Psoriasis
4.Geographic tongue
5.Pachyonychia congenita
6.White sponge nevus
7.Hereditary benign epithelial dysplasia
8.Stomatitis nicotina
9.Hyperkeratosis palmoplantaris with gingival hyperkeratosis

10.Darrier's disease
11.Intraoral skin grafts
12.Pseudoxanthoma elasticum
13.Hyalinosis cutis et mucosa oris
14.Oral condyloma acuminatum
15.Hairy leukoplakia

Nonkeratotic white lesion
1.White hairy tongue
2.Burns-thermal, chemical, electrical
3.Desquamative gingivitis
4.Epidermolysis bullosa
5.Pemphigus
6.Benign mucous membrane pemphigoid (BMMP)
7.Diphtheria
8.Syphilitic mucus patches
9.Acute necrotizing ulcerative gingivitis (ANUG)
10.Koplik's spot of measles
11.Candidiasis
12.Median rhomboidal glossitis
13.Veruca vulgaris

ORAL LEUKOPLAKIA:

The World Health Organization (1978) has defined oral leukoplakia as a white patch or plaque which cannot otherwise be characterized clinically or pathologically as any other disease.[2] Leukoplakiais the most frequent precancerous lesion of the mouth.Pindborg et al. (1963) differentiate two main groups according to clinical appearance: Homogenous leukoplakia and non-homogenous leukoplakia.The Clinical features of leukoplakia are focal and smooth or homogeneous to "pumice-like", with an associated low risk of malignant transformation. Other lesions may be heterogeneous or non-homogeneous (speckled, erosive, ulcerative, verrucous), which may be interspersed with red or atrophic areas and are associated with a higher risk of dysplasia and malignant transformation. If there is a shift from a homogeneous to a nonhomogeneous form, re-biopsy is mandatory.[1][2]The Histopathological examination reveals[12]

Candidalleukoplakia:

The presentation of chronic candidal infection in the form of leukoplakia is termed as "chronic hyperplastic candidosis" (CHC) and candidalleukoplakia. Oral candidosis lesions are subdivided into two main groups: Group I, or primary oral candidoses confined to lesions localized to the oral cavity with no involvement of skin or other mucosa, and Group II or secondary oral candidoses, where the lesions are present in the oral and extra-oral sites such as skin.[11]

Proliferative verrucous leukoplakia:

Proliferative verrucous leukoplakia (PVL) and verrucous hyperplasia (VH) are two interrelated oral mucosal lesions. It is a slow growing diffuse, multifocal exophytic lesion, with or without an erythematous component. The lesions are described as persistent, progressive, relentless, and non-reversible. Biopsy specimens of the lesions, overtime, showed varying degrees of epithelial hyperplasia and dysplasia and eventually squamous carcinoma (verrucous or conventional squamous cell).[17]

Sanguinaria-associated oral leukoplakia:

It is caused by the use of a dentifrice or mouthrinse containing the herbal additive sanguinaria. Clinically, most of these lesions are in the anterior maxillary mucobuccal fold and attached gingiva. Histologically, they are usually hyperorthokeratotic, and the cells occupying the lower strata exhibit mild cytologic atypical features, including nuclear polarization, nuclear enlargement, and mild pleomorphism; the condition thus qualifies as a mild dysplasia. None of these lesions have disclosed microscopic evidence of severe dysplasia, nor have any instances of carcinoma been reported in sanguinaria-associated leukoplakia.[4]The treatment of sanguinaria associated oral leukoplakiaareElimination of predisposing factors, use of beta-carotene, lycopene, ascorbic acid, α-Tocopherol (Vitamin E), topical and systemic retinoic acid (Vitamin A), topical bleomycin, cold-knife surgical excision, laser surgery along with regular follow up.[18]

ORAL CANDIDIASIS:

Oral candidiasis is the commonest human fungal infection. If untreated it can lead to poor nutrition and prolonged recovery.In extreme cases can be fatal when it becomes disseminated. It is caused by the fungus **Candida albicans.**

Oral candidiasis can be classified as follows:

1.Acute candidiasis

a)Acute pseudomembranous candidiasis (thrush).

b)Acute atrophic (erythematous) candidiasis.

2.Chronic candidiasis

a)Chronic hyperplastic candidiasis (candidalleukoplakia).

b)Denture induced candidiasis (chronic atrophic (erythematous) candidiasis).

c)Median rhomboid glossitis.

3.Angular cheilitis (stomatitis) [7]

The clinical featuresis characterized by thick, white patches of different magnitude, which may coalesce and form a continuous coverage of the oral mucosa. The white plaques are removable by strong rubbing, leaving a reddened, erythematous, sometimes bleeding surface.[1]The histopathological examination reveals that the epithelium is thickened, shows hyperplasia, parakeratosis, and infiltration of polymorphonuclear leukocytes. The Candidal hyphae made visible by PAS staining in the superficial layers of the epithelium. Dysplastic changes occur especially in association with oral leukoplakia.[1]The management of candidiasis includes the elimination of Predisposing factors, maintenance of good oral hygiene, administration of Topical antifungals for two weeks.Systemic antifungals should be administered in some cases.[7]

FORDYCE GRANULES:

Fordyce granules appears in vermilion border of lips & oral mucosa as yellow spots which forms discrete/ confluent nodules or forms large plaques appearing as multiple yellow or yellow white papular lesions. These are called as "ectopic" sebaceous glands because of its distribution in the oral cavity and dermal structures. It is also called as exocrine (holocrine) glands, these sebaceous glands diminishes water permeability of hair, skin & eyelids which aids in lubrication.Theclinical featuresare asymptomatic, heterogeneous patch of yellowish white dots presents on both sides of buccal mucosa. These maculopapulesare 1-3 mm in diameter and are clinically consistent.The histopathological examination reveals clusters in hyperparakeratinized stratified squamous epithelium overlying a fibro cellular connective tissue stroma. Superficial sebaceous glands with lobules are seen with flattened and darkly stained peripheral layer, pale and foamy inner layer. There is a direct opening into the surface by short keratinized ducts. Ducts show keratin plugging and connective tissue with loosely arranged collagen fibers.Since it is asymptomatic no treatment is necessary.[5]

ORAL LICHEN PLANUS:

Lichen planus is a chronic inflammatory disease that affects skin and mucosa. It is one of the most common dermatological conditions involving the oral cavity. [9] It is a T-cell mediated autoimmune disease in which cytotoxic CD8+ T-cell trigger the apoptosis of oral epithelial cells.The Clinical features of oral lichen planus presents in one of twoforms - the reticular form and the erosive form. According to Mollaoglu19 (2000), four other forms were originally described: the papular,plate-like, bullous and atrophic forms. The reticular form is more frequently occurs and it is characterized by white lacy streaks known as **Wickham's striae**, which is surrounded by erythematous borders. The reticular form usually causes no symptoms.[9]The Histopathological examination reveals the lichenification of the basement layer, followed by a marked layered lymphocytic infiltrate immediately underlying the epithelium; the presence of numerous eosinophilic colloid bodies along the epithelial-connective tissue interface **Civatte bodies**absent, hyperplasic, saw tooth-shaped interpapillary ridges, variable thickness of the spinous layer, and variable degrees of ortho or parakeratosis.[9]The treatment includes Corticosteroids which is drugs of choice due to their ability to modulate the inflammatory and immunological responses.Topical use and local injection of steroids are used successfully for controlling lichen planus. Systemic steroids may be used for severe cases.[9]

LUPUS ERYTHEMATOSUS:

Lupus erythematosus (LE) is an autoimmune disease among other connective tissue diseases like scleroderma, rheumatoid arthritis,polymyositis, and mixed connective tissue disease.[15]In discoid erythematous plaques with firmly adherent follicular hyperkeratoses and hyperesthesia,active border with erythema and hyperpigmentation.Discoid lesions are more frequently occurs in the vermillion border than the buccal mucosa,mutilation around nose and mouth, perioral pitted scars.[13]The histopathological examination shows hyperparakeratosis, liquefaction degeneration of basal layer, focal or perivascular infiltrates of lymphocytes.The treatment includes potent topical steroids and antimalarials.In some cases, retinoids, thalidomide, and topical

tacrolimus offer alternatives, as do immunosuppressives like azathioprine, cyclosporine, mycophenolate mofetil, and methotrexate.[13]

WHITE SPONGE NEVUS:

White sponge nevus (WSN) is a rare autosomal dominant genetic disease of oral mucosa. The lesion is white or greyish, thickened, folded and spongy in nature. It is also called as familial white folded dysplasia. The condition affects labial mucosa, ventral surface of the tongue, soft palate, alveolar mucosa, and floor of the mouth.The extra oral sites are the nose,esophagus, larynx and anogenital mucosa.TheClinical features are characterized by symmetric, thickened, white, corrugated or velvety, diffuse plaques affects buccal mucosa presents bilaterally. The plaques are smooth with velvety texture and irregular, well-defined borders.The lesions are asymptomatic and painless.TheHistopathological examination reveals prominent hyperparakeratosis and acanthosis with clearing of the cytoplasm of the cells in the spinous layer. A unique feature of WSN iseosinophilic condensation in the perinuclear area of the cells in the superficial layers of the epithelium.TheCurative treatments with Nystatin, antihistamines, vitamins, and mouth rinses. Azithromycin, tetracycline and penicillin have few clinical effects. Following penicillin administration, Victoria A acid and tetracycline mouth rinse a significant improvement is seen.[16]

HEREDITARY BENIGN INTRAEPITHELIAL DYSKERATOSIS:

Hereditary benign intraepithelial dyskeratosis (HBID) is a rare hereditary disorder affecting the oral and ocular mucosa.It is characterized by white spongy plaques in the buccal mucosa associated with the typical ocular findings of white conjunctival plaques.The disorder affects the mucosa of the mouth and lip, ventral surface of the tongue, and floor of the mouth. White, spongy plaques that may vary in thickness form in the mouth and may be thrown into folds.There is no Malignant transformation in the oral or ocular lesions. The findings of HBID are similar to clinical and histopathologic appearance to the white sponge nevus.The treatment includes the genetic cause of HBID has not been elucidated and there is no effective treatment for the oral or ocular lesions. After excision, the abnormal epithelium almost invariably recurs.

DYSKERATOSIS CONGENITA:

Dyskeratosis congenita (DC) is a rare, inherited, disorder, with premature ageing, bone marrow failure and malignancy. It consists of the triad of nail dystrophy increased skin pigmentation and mucosal leukoplakia. The syndrome often proves fatal due to progressive bone marrow failure (or malignant change within areas of mucosal leukoplakia).This can occur on any mucosal surface, but most frequently occurs in the oral mucosa. The specific intraoral sites previously published include, lingual mucosa, buccal mucosa and the palate, with the tongue being the most frequently affected.The oral manisfestation other than leukoplakia, include hyperpigmentation of the buccal mucosa, severe periodontal disease, hypocalcified teeth and taurodontism..[3]The histopathological examination shows abnormal epidermal cell keratinization, apoptosis of keratinocytes, nuclear pyknosis.Thetreatment includes bone marrow transplants (transplant of blood forming stem cells), Androgen therapy (artificial hormones to increase red blood cells).

II. Conclusion:

The role of the oral white lesions in the possible development of oral carcinoma, the precancerous nature of oral leukoplakia is today established. The clinically erosive and histologically dysplastic leukoplakias show a greater tendency for malignant transformation, and might therefore be considered as the "high risk" group among oral leukoplakias. Some clinical types of oral lichen planus, especially the atrophic and erosive forms, because of their possible association with oral carcinoma, care should be taken in the treatment and supervision of these lesions, however the precancerous nature of oral lichen planus requires further investigation. In candidiasis and discoid lupus erythematosus, epithelial dysplasia may occur, but no direct evidence of malignancy has been established. With the aid of clinical, histological and ultrastructural methods the differential diagnosis of these "white lesions" no longer causes problems to the expert clinicopathologist.

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