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RESEARCH ARTICLE

KERATOTIC LESIONS OF ORAL CAVITY

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Abstract

Lesions generally appear white intra orally for one of the several reasons like a thickened surface layer of keratin, an acanthotic epithelium or edematous epithelial cells. Exudates and adherent surface debris may also appear white. Identification of these lesions and making a definitive diagnosis using microscopic examination is extremely important, because some of these lesions may represent squamous cell carcinoma. This review lists the white lesions affecting the oral mucosa ranging from those that are genetically determined to those that are neoplastic and also highlights the specific features of each of these lesions.

Introduction:-

White lesions of the oral mucosa obtain their characteristic appearance from the scattering of light through an altered mucosal surface. Such alterations may be the result of a thickened layer of keratin (hyperkeratosis), thickening of spinous layer of the epithelium (acanthosis), intracellular edema of epithelial cells and reduced vascularity of subjacent connective tissue. White or yellow-white lesions may also be due to a fibrin exudate covering an ulcer, surface debris or fungal colonies.[1] With experience, the reason for a lesion's white appearance can be determined clinically, but the final diagnosis relies on microscopic examination.

White sponge nevus

White sponge nevus (WSN) is an autosomaldominant disorder characterized by asymptomatic white, thickened, and folded spongy plaques occurring symmetrically on the buccal mucosa. The tongue, labial mucosa, alveolar ridges, and floor of the mouth are also commonly affected. WSN presents at birth or early in childhood; rarely, the condition may appear during adulthood. Once they appear, they remain unchanged throughout the patient's life, except sometimes they become more pronounced during pregnanc. Some authors suggest that oral microflora could contribute to the stimulation of the lesion, because it has been demonstrated that WSN improved after tetracycline treatment. The histologic findings are characteristic but not pathognomonic. These findings include prominent hyperparakeratosis and acanthosis with clearing of the cytoplasm in the spinous layer. Diagnostic tests Exfoliative cytology of the epithelial cells stained with Papanicolaou's method show the characteristic eosinophilic perinuclear condensation better than histopathologic sections.No treatment is necessary.

Pachyonychia congenita

Pachyonychia congenita (Jackson-Lawler syndrome or Jadassohn-Lewandowsky syndrome) are a group of ectodermal dysplasias. The autosomal-dominantly inherited disorder is characterized by onychogryphosis; hyperkeratosis of the palms, soles, knees, and elbows; extensive tiny cutaneous horns; and leukokeratosis of the oral mucous membranes . Hyperhidrosis of the hands and feet is present frequently. Autosomal-recessive and late-onset pedigrees have been described. Oral leukokeratosis is similar in its appearance to that observed in dyskeratosis

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congenita. Dysplasia, however, does not develop.The genetic defect associated with pachyonychia congenita is localized to chromosome 17q12-q21 and chromosome 12q13. Both of these result in keratin defects.

Dyskeratosis Congenita

Dyskeratosis congenita is an inherited disorder with x-linked, autosomal-recessive and autosomaldominant pedigrees. Dyskeratosis congenita is characterized by skin pigmentation, leukoplakia, and nail dystrophy associated with a progressive bone marrow failure . Clinical manifestations in dyskeratosis congenita often appear during childhood. The skin pigmentation and nail changes typically appear first, usually by the age of 10 years[9,10]. Mucosal leukoplakia and epiphora appear later and by the midteens the serious complications of bone marrow failure and malignancy begin to develop. Rarely, marrow abnormalities may appear before the skin manifestations.An RNA component of telomerase is mutated[12].Treatment for this fatal disease remains unsatisfactory.

Leukoedema

Leukoedema is a common oral condition of unknown cause. The oral mucosa appears to have an asymptomatic, symmetric, opalescent milky-white film with accentuation of edematous folds or streaks (Fig. 1). Leukoedema most commonly occurs bilaterally on the buccal mucosa; it may also be noted on the floor of the mouth and palatopharyngeal tissues. Oral lesions of leukoedema show parakeratosis and an increase in thickness of the oral mucosa epithelium with intracellular edema of the spinous layer. The cells of the spinous layer are large with pyknotic nuclei. Rete ridges may be elongated. No dysplasia or hypergranulosis is evident. The white lesions of leukoedema do not rub off. Stretching of the oral mucosa and the resultant disappearance of the opalescence in the mucosa is diagnostic. Any diffuse white lesions of the oral mucosa should always be stretched out to rule out any other underlying lesions. No treatment is necessary.

Linea alba (buccalis)

It is a virtually inexplicable fact that some people have a white ridge on the buccal mucosa opposite the occlusal plane of the teeth, and others have not. In some mouths, it is tempting to attribute this line or ridge to an "edge to-edge" relationship of the posterior teeth with the implication that the cheek is pinched between the teeth as they come together. In other mouths, it seems that the thickness or plumpness of the cheeks pushes the buccal mucosa in between the teeth. Both theories are difficult to sustain because the factors just mentioned are not always obvious. Whatever the cause, linea alba has no pathological significance and must be regarded as a variant of normal.

Smokers' Keratosis

It is a well established fact that smoking (and the use of tobacco in other form causes abnormal changes in the oral mucosa. These changes are usually seen clinically as leukoplakia with various degrees of brown staining from the nicotine and other chemicals produced by burning tobacco, and the lesions are referred to as smokers' keratoses.The most common sites of cancer development due to smoking are the "pooling" areas, that is, floor of mouth, lingual and buccal sulci, undersurface and sides of tongue. This is probably due to the chemical products of tobacco combustion dissolving in saliva and pooling in the lower parts of the mouth.

Candidiasis (Candidosis, Moniliasis, Thrush)

The organism Candida albicans lives quietly and unobstrusively in the mouths of some 40% of the population, without ever producing an oral lesion. However, it is a great opportunist and under a variety of conditions that can render the oral mucosa (or other epithelial surfaces) abnormal, it can emerge as an extremely troublesome pathogen. In order to produce mucosal lesions it must invade the epithelium, but this invasion is superficial and limited to the cornified layer. Until recently a simplistic concept was accepted of the etiology of candidiasis but a number of workers in the field, notably Lehner*' and Gawson,'-- *•' have made important advances in understanding and categorizing the manifestations of this infection. Broadly speaking, candidiasis can be classified in two main categories, acute and chronic, with a number of subdivisions.

Discoid lupus erythematosus

Lupus erythematosus (LE) is an autoimmune condition with a broad spectrum of disease manifestations. LE may present in chronic form or it may present acutely. Occasionally, subacute lesions can be seen. Skin and oral lesions characterize chronic mucocutaneous LE. Subacute cutaneous LE is characterized by recurring superficial nonscarring annular skin lesions that are more disseminated and present more acute features both clinically and histologically than those seen in the chronic discoid type. Acute systemic LE lesions present as erythematous edematous plaques on the skin and erosions of the mucous membranes. Chronic cutaneous LE primarily affects the

skin but the oral mucosa can also be affected [18]. Patients with discoid lupus erythematosus (DLE) lesions typically have cutaneous findings and, rarely, they may also present with oral findings. DLE is used to describe both the skin and oral findings. Cutaneous DLE lesions are common on the scalp, face, and in the ears.

Syphilis mucous patches

Mucous patches are an oral manifestation of secondary syphilis. The plaques are usually oval and covered with white or gray membrane that is removed easily to reveal underlying raw connective tissue. Roughly 30% of patients with secondary syphilis present with superficial painless oral lesions with irregular, grayish mucosal necrosis. These patches are found on the tongue, lips, buccal mucosa, and palate. Mucous patches may heal spontaneously, but have a high incidence of recurrence (Fig. 8). Other findings associated with secondary syphilis include a papulosquamous eruption with prominent copper-colored scaly plaques involving the palms and soles; a moth-eaten alopecia; and genital condylomata lata lesions, which may be associated with a mild lymphadenopathy, hepatosplenomegaly, and a residual chancre.

Nicotinic stomatitis

Nicotinic stomatitis (smoker's palate) is a benign process with no malignant potential. Nicotinic stomatitis is always confined to the hard palate and begins as erythema of the palate. A characteristic finding is the appearance of multiple red dots, which represent the dilated and inflamed duct openings of the minor salivary glands. The lesions are asymptomatic and discovered during an oral examination. Nicotinic stomatitis occurs almost exclusively in heavy pipe smokers and rarely in cigarette or cigar smokers. It is also observed in reverse smokers (lit end placed in the mouth). This observation suggests that a thermal effect is the cause of the clinical changes.Tobacco pouch keratosis (smokeless tobacco pouch) Lesions induced by smokeless tobacco characteristically have a wrinkled surface that ranges from opaque white to translucent and develop on the mucosal surfaces that contact the tobacco products.

Squamous cell carcinoma

It is beyond the scope of this article to write a detailed overview of oral squamous cell carcinoma (SCC). A brief description is provided. Oral SCC appears in different clinical forms: leukoplakia, erythroplakia, and nonhealing ulcer.There is a higher incidence after the age of 40 years with a peak at 60 years.mostly occurring in the lips, tongue, floor of the mouth, palate, gingiva, alveolar and buccal mucosa, and oropharynx.

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