White Lesions

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KEYWORDS

• White lesions • Oral mucosa • Leukoplakia • Hyperkeratosis
• Dysplasia

Increased thickness of the epithelium imparts a white appearance to the oral mucosa by increasing the distance to the underlying blood vessels. Usually this thickening is a result of the increased formation of keratin. Some other less common causes of white lesions are acanthosis or a thickening of the spinous cell layer, edema of the epithelium, or increased fibrosis of the connective tissue thereby reducing blood vessels. Occasionally the surface of an ulcer may appear white, due to collection of fibrin on the surface. In this article the authors discuss white lesions based on putative etiology, that is, hereditary, reactive, inflammation related, immunologic, traumatic, infection related, and idiopathic. Because many other lesions that may appear white have overlapping etiology and/or clinical nature (eg, candidiasis, carcinoma, smokeless tobacco associated lesions), they are discussed in other articles.

DEVELOPMENTAL WHITE LESIONS

Leukoedema

Leukoedema is a common developmental mucosal alteration of unknown cause, rather than a true pathologic change.[1,2] Leukoedema is seen in 90% of all black adults and 50% of black teenagers.[3] This condition has also been reported over a wide range in white adults (10%–90%) but the changes are far less pronounced.[3] Tobacco smoking and chewing are reported to enhance the whiteness and extent of the lesion.[4,5] Similar edematous changes have been seen in the mucosa of the larynx, the vagina, and other mucosal surfaces.[5]

Clinical features

Leukoedema typically presents as a bilateral, diffuse, milky, gray-white asymptomatic area with numerous surface folds, on the buccal mucosa (**Fig. 1**). Rarely it may involve the floor of the mouth and palatopharyngeal tissues.[6] Mucosal changes start as early as age 3 to 5 years and by the end of the teen years, 50% of blacks present with mucosal alterations.[3] An important diagnostic feature of leukoedema stems from its disappearance on stretching.

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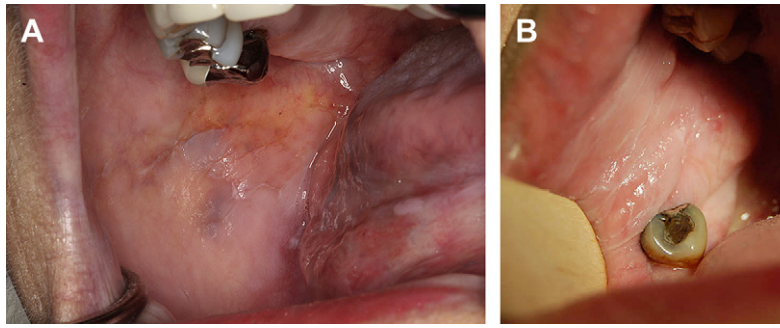

Fig. 1. (*A*) Diffuse, gray-white appearance of the buccal mucosa in leukoedema. (*B*) Wrinkled, white appearance in leukoedema may extend throughout the buccal mucosa.

Histopathologic features
Epithelial hyperplasia with significant intracellular edema of the spinous cells, and broad and elongated rete ridges are evident. Keratinization of the surface is also noted.

Differential diagnosis
White sponge nevus, frictional keratosis, smokeless tobacco keratosis, and hereditary benign intraepithelial dyskeratosis.

Treatment
None indicated. No malignant changes have been reported.[3,6]

White Sponge Nevus

White sponge nevus is a rare autosomal dominant disorder resulting from point mutation of either keratin 4 or keratin 13 genes.[6–8] These mutations result in defective keratinization of the oral mucosa, with alterations that may also be seen in the nasal, esophageal, laryngeal, and anogenital mucosa.[6,9,10] A high degree of penetration and variable expression is seen. White sponge nevus typically presents early in life and there is no gender predilection.[9]

Clinical features
White sponge nevus presents as asymptomatic, white, soft and spongy bilateral plaques or macules, typically on the buccal mucosa, but other sites such as the lip, ventral surface of the tongue, and floor of the mouth may also be involved.[11,12] Rare cases of mild discomfort due to secondary infections have been reported.[12]

Histopathologic features
Parakeratosis, marked epithelial thickening, and intracellular edema with perinuclear condensation of keratin is seen. Clear cell changes begin at the parabasalar layer and extend close to the surface.

Differential diagnosis
Leukoplakia, candidiasis, pachyonychia congenita, hereditary benign intraepithelial dyskeratosis, Darier disease, dyskeratosis congenita, lichen planus, lupus erythematosus, chemical burns, and syphilis.