

Dermatologic Manifestations of Oral Leukoplakia

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Background

The World Health Organization (WHO) first defined oral leukoplakia as a white patch or plaque that could not be characterized clinically or pathologically as any other disease; therefore, lichen planus, candidiasis, and white sponge nevus were excluded. At a 1983 international seminar, the following definition was proposed:

Leukoplakia is a whitish patch or plaque that cannot be characterized clinically or pathologically as any other disease, and is not associated with any physical or chemical causative agent, except the use of tobacco.

A more recent WHO workshop^[1] has amended the earlier WHO definition as follows: "The term leukoplakia should be used to recognize white plaques of questionable risk having excluded (other) known diseases or disorders that carry no risk for cancer." It has also recommended abandoning the distinction between the terms "potentially malignant lesions" and "potentially malignant conditions" and to use the term "potentially malignant disorders" instead. Leukoplakia and erythroplakia are the most common potentially malignant disorders. These diagnoses are still defined by exclusion of other known white or red lesions.

Oral white lesions include leukoplakias (as defined above), keratoses, leukoplakias of clear infective origin (candidal, syphilitic, hairy leukoplakia associated with Epstein-Barr virus), candidosis, lichen planus, oral submucous fibrosis, lupus erythematosus, congenital lesions (eg, white sponge nevus, dyskeratosis congenita, pachyonychia congenita), and frank carcinomas.

Pathophysiology

No etiologic factor can be identified for most persistent oral white plaques (ie, idiopathic leukoplakia). The histopathologic features are highly variable, ranging from hyperkeratosis and hyperplasia to atrophy and severe dysplasia.

Patients with idiopathic leukoplakia have the highest risk of developing cancer. In studies of these patients, 4-17% had malignant transformation of the lesions in less than 20 years. The risk of developing malignancies at lesion sites is 5 times greater in patients with leukoplakia than in patients without leukoplakia.

Dysplastic lesions do not have any specific clinical appearance; however, where erythroplakia is present, dysplasia is likely.

Dysplasia is evident in 17-25% of biopsy samples of leukoplakias. Erythroleukoplakias, verrucous leukoplakias, and nodular leukoplakias show an increasing frequency of dysplastic histologic changes or aneuploidy.

Leukoplakias that are speckled, or erythroleukoplakic, are usually dysplastic or frank carcinomas. Nodular or verrucous lesions are also sinister, but homogenous leukoplakias are far less likely to be potentially malignant.

Most idiopathic leukoplakias are homogenous leukoplakias and show little evidence of dysplastic histologic changes or aneuploidy. However, studies have revealed carcinoma or severe dysplasia in the excision specimens of approximately 5% of leukoplakias excised when the diagnostic biopsy specimens had revealed no dysplasia.

Carcinoma in situ is a controversial term used for severe dysplasia in which the abnormalities extend throughout the thickness of the epithelium. All the cellular abnormalities characteristic of malignancy may be present; only invasion of the underlying connective tissue is absent. Top-to-bottom epithelial dysplasia, like other dysplastic lesions, has no characteristic clinical appearance, although erythroplasia often proves to be carcinoma in situ or early invasive carcinoma.^[2]

Epidemiology

Frequency

Oral leukoplakia is uncommon, possibly occurring in less than 1% of adults.

Race

An increased prevalence is observed in communities and races with high tobacco

use, such as Southeast Asia.

Sex

Males have the highest incidence of leukoplakias.

Age

Leukoplakias are usually seen in adults older than 40 years.

Clinical Presentation

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