ORAL LEUCOPLAKIA: DIAGNOSTICS AND TREATMENT

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Abstract

Leukoplakia of the oral mucosa (OM) is the most common potentially malignant disease characterized by the appearance of white, non-erasable spots that have no alternative clinical and histological diagnosis. The article presents modern concepts of the etiology, pathogenesis, classification, clinical forms, diagnostics and treatment of leukoplakia. Particular attention is paid to the issues of dysplasia and precancerous potential of the disease. A review of modern approaches to therapy and dispensary observation of patients with this disease is conducted.

Key words: leukoplakia, oral mucosa, dysplasia, precancerous disease, treatment

Introduction

Leukoplakia of the oral mucosa (OM) is a chronic disease with a high oncogenic potential. According to the WHO definition (2005), it is "a white spot or plaque that cannot be characterized as any other disease." The disease is widespread and

occurs in 0.2–11% of the population, more often in men after 40 years of age, especially in the presence of bad habits (smoking, alcohol). Timely detection and treatment of leukoplakia helps prevent the development of oral cancer.

Leukoplakia is considered a multifactorial disease, but smoking is considered to be the most common factor in its development. It is much more common among smokers than non -smokers [4]. According to some authors, alcohol is considered to be an independent risk factor [5], but there is still no definitive data. There are conflicting results of studies related to the possible role of human papillomavirus infection. Since leukoplakia can mimic a large number of lesions, in case of suspicion of a possible causative factor, such as dental restoration, mechanical irritation. In a later case, a follow-up assessment after 4 weeks is necessary [6].

Etiology and pathogenesis

The main predisposing factors are:

Smoking and alcohol

Mechanical trauma (sharp edges of teeth, incorrectly installed dentures)

Vitamin deficiency , especially group A and B

Immune disorders

Human papillomavirus (HPV) infection

Pathogenesis includes disruption of the processes of differentiation and keratinization of the epithelium, which can lead to dysplasia and malignant transformation.

Classification and clinical forms

The most commonly used classification is the WHO classification, which is divided into the following forms:

Flat (simple) - white spots with a smooth surface, without signs of inflammation.

Verrucous - a thickened, folded surface with signs of keratinization.

Erosive-ulcerative - painful areas with erosions, against which hyperkeratosis zones develop.

Hyperplastic - dense white plaques, often with signs of dysplasia.

Diagnostics

Modern diagnostics of leukoplakia is based on a comprehensive approach that combines clinical examination, morphological and molecular tissue studies.

1. Imaging techniques and optical biopsy

Technologies such as optical coherence tomography (OCT), fluorescence imaging, reflectance spectroscopy are used to assess the degree of dysplasia in vivo before performing invasive biopsy.

Farah CS, McCullough MJ. Oral mucosal imaging: an overview. Aust Dent J 2007 ;52 (Suppl 1):S58–S63.

2. Biopsy and histology

The gold standard remains incisional biopsy followed by histological analysis. According to WHO Collaborating Centre (2017), mild, moderate and severe dysplasia are the determining risk factors for malignancy.

Speight PM, Khurram SA, Kujan O. Oral potentially malignant disorders: risk of progression to malignancy. Oral Surg Oral Med Oral Pathol Oral Radiol . 2018;125(6):612–627.

3. Cytological methods

Liquid cytology and express cytology (including with computer image analysis) are used as a screening method, especially in patients with multiple foci.

4. Immunohistochemical markers

The most studied markers:

p53 , Ki-67 , Cyclin D1 are indicators of proliferation and mutational activity.

E- cadherin , Podoplanin are markers of early transformation.

HPV 16/18 and p16INK4a - especially in lesions of the tongue and floor of the mouth.

Kujan O, Sloan P, Duxbury A, et al. Evaluation of immunohistochemical expression of biomarkers in oral epithelial dysplasia: a systematic review and meta-analysis. J Oral Pathol Med . 2017;46(6):387–396.

Treatment

1. Drug treatment

Drug therapy is relevant for uncomplicated forms and as preparation before surgical intervention.

Retinoids (topical and systemic) reduce keratinization and dysplasia. However, if treatment is stopped, there is a high risk of relapse.

Lodi G, et al . Interventions for treating oral leukoplakia to prevent oral cancer. Cochrane Database Syst Rev. 2016 ;2016 (7):CD001829.

Solcoseryl gel - activates tissue regeneration, promotes normalization of microcirculation and reduces inflammation. Effective in complex therapy, especially in combination with vitamins A and E.

Immunomodulators and antioxidants - local and systemic forms (eg, imudon , lysobact , α- lipoic acid) reduce oxidative stress and increase local immunity.

2. Surgical treatment

Laser ablation (CO_2 and diode lasers) is minimally invasive, has low pain and recurrence rates.

Van der Hem PS, Nauta JM, Roodenburg JL, Vermey A. The treatment of oral leukoplakia with CO_2 laser. Oral Surg Oral Med Oral Pathol Oral Radiol Endod . 1999;87(5):598–602.

Cryosurgery is effective for small lesions. It allows tissue destruction with minimal pain and bleeding.

Surgical excision with a scalpel is recommended when severe dysplasia is detected. In most cases, it is accompanied by histological control of the resection margins.

3. New methods and clinical trials

Photodynamic therapy (PDT) is a promising non-invasive method. Active Photosensitization followed by light irradiation leads to the destruction of atypical cells.

Fukuhara K, Yamamoto H. Photodynamic therapy for oral leukoplakia and squamous cell carcinoma: a review. Int J Mol Sci . 2021;22(15):8200.

Nanotherapy and drug delivery - the use of liposomal forms of vitamins, retinoids and antioxidants increases their effectiveness when applied topically.

Prognosis and prevention

The risk of malignant degeneration of leukoplakia is from 3 to 20% depending on the form and degree of dysplasia. The most dangerous are warty and erosive forms. The basis of prevention is the elimination of bad habits, oral sanitation, taking vitamins and regular monitoring.

Conclusion

Leukoplakia of the oral mucosa is an important object of observation for a dentist due to its potential malignant nature. A comprehensive approach, including timely diagnosis, treatment and observation, can reduce the risk of cancer and improve the quality of life of patients.

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