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Precancerous Lesions of the Oral Mucosa and the Vermilion Border of the Lips with a High Risk of Malignant Transformation

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Abstract: Early detection of diseases with a high risk of malignancy is a crucial aspect of medical practice. Lesions on the oral mucosa and the vermilion border of the lips are easily visible, which places significant responsibility on dentists and other healthcare professionals to promptly recognize potentially dangerous areas. This article discusses various precancerous conditions that carry a substantial likelihood of developing into cancer.

Keywords: oral mucosa, precancerous lesion, cheilitis.

In dental practice, stomatitis is classified based on the etiological factor (traumatic, bacterial, fungal, viral, etc.), localization (glossitis, cheilitis, skin diseases), and leading clinical symptoms (vesicular, hyperkeratosis, neoplasms) [1].

Some diseases are confined solely to the oral mucosa (OM), such as traumatic injuries and Vincent's ulceronecrotic stomatitis. In some cases, manifestations on the mucosa may reflect systemic diseases or their treatment effects (chronic recurrent herpetic stomatitis, fungal infections, mucositis during chemotherapy). A separate group includes oral manifestations of dermatological diseases (lichen planus, erythema multiforme, pemphigus) [2].

Another category includes oral mucosal lesions resulting from pathological systemic processes but are neither obligatory nor specific symptoms of the primary disease. These include manifestations related to gastrointestinal tract (GIT) diseases, cardiovascular system disorders, allergic reactions, neurogenic and endocrine disturbances (glossitis, glossodynia, bullous syndrome) [4].

The oral mucosa may also react to systemic infectious and non-infectious diseases that severely disrupt homeostasis (childhood infections, radiation sickness, myocardial infarction, blood pathologies). Some patients visiting the dentist may be carriers of contagious diseases such as AIDS, syphilis, or tuberculosis. In such cases, the dentist must refer the patient to specialized healthcare facilities for diagnosis and treatment. The specialist must be well aware of the signs of life-threatening diseases to make a preliminary diagnosis and timely refer the patient to a hematologist (for blood disorders), oncologist (in case of malignancy of tumors or ulcers), or dermatologist (for pemphigus) [3].

Diseases with a high tendency to malignancy (obligate precancers of the oral mucosa and vermilion border of the lips) deserve special consideration in dental practice. These conditions often lack objective signs of cancer but can rapidly progress to malignancy under unfavorable conditions [5].

To improve early diagnosis of precancerous diseases with a high risk of malignization, literature data, consultations, and clinical observations were analyzed. Over five years, 80 patients with oral mucosal lesions prone to malignization were examined. Most lesions were of traumatic origin. The majority could be classified as facultative precancers, with only a few clinical cases identified as obligate precancers. Patients with indications were referred for oncological consultation and laboratory testing.

The main clinical features of obligate precancers are described below.

Bowen's disease was first described by the American dermatologist James Bowen in 1912. It is considered one of the most malignant precancerous conditions and is classified as cancer in situ (intraepithelial carcinoma without invasive growth). It most commonly affects the posterior parts of the oral mucosa, such as the soft palate, uvula, retromolar area, and root of the tongue, but can also manifest on the cheeks, lateral tongue, and soft palate.

Clinically, the lesion usually appears as a single (rarely two or three) well-defined, congested red patch with a smooth or velvety surface, often with small papillary growths. When localized on the tongue, the papillae disappear in the affected area, and the lesion may appear slightly depressed due to mucosal atrophy. The disease course is variable: in some cases, it progresses rapidly to invasive growth, while in others, it remains in the cancer in situ stage for years.

Diagnosis is confirmed by histopathological examination, revealing features of intraepithelial carcinoma such as cellular polymorphism in the prickle cell layer, increased and abnormal mitoses, presence of giant multinucleated cells ("monster cells"), acanthosis, hyperkeratosis, and parakeratosis. The basement membrane and basal layer remain intact. In the upper part of the lamina propria, an infiltrate of lymphocytes and plasma cells is observed.

Early stages are characterized by the formation of a localized erythematous area, which can be nodular-spotted or smooth with a velvety surface due to small papillary proliferations. Later, areas of hyperkeratosis develop, with a tendency to erosion. When nodules form and merge into plaques, the lesion protrudes above the surrounding tissue; with prolonged disease, atrophy and depression of the lesion occur.

Diagnostic difficulties arise when a small erythematous patch becomes covered with scales, resembling leukoplakia or lichen planus. The diagnosis of Bowen's disease is based on histological findings, including giant multinucleated cells in the prickle cell layer.

The prognosis is unfavorable because lesion progression over 2–4 months usually leads to invasive growth without regression. Treatment involves complete surgical excision of the lesion with healthy surrounding tissue. When surgery is not possible, close-focus radiotherapy is used.

Verrucous precancerous lesion (verrucosus) was first described by A.L. Mashkilleison in 1965. It occurs almost exclusively on the red border of the lower lip and presents as a hemispherical nodule measuring 4 to 10 mm in diameter, rising 4–5 mm above the mucosal surface. The surface of the nodule is covered with tightly adherent gray scales that cannot be removed by scraping.

Patients typically complain of a cosmetic defect and mild discomfort. The lesion is usually solitary and located on the red border of the lower lip. Surrounding tissues remain unchanged. On palpation, the nodule is firm and painless.

Histopathologically, there is a pronounced, well-demarcated epithelial proliferation due to the expansion of the prickle cell layer. Hyperkeratosis often alternates with parakeratosis. Dyscomplexation and polymorphism of the prickle cells are observed. The basement membrane remains intact. The connective tissue shows a round-cell infiltrate.

Differential diagnosis includes papilloma, keratoacanthoma, and pyogenic granuloma. Unlike a wart, which is characterized by lobulated structure or papillary proliferations with a horny rim at the periphery, keratoacanthoma features a funnel-shaped crater filled with numerous keratinized

cells and a hyperemic mucosal border, while papilloma typically has a soft stalk.

Treatment of verrucous precancerous lesion is exclusively surgical, with complete excision of the lesion within healthy tissue margins and mandatory histological examination. This condition has a marked tendency toward malignancy, which may develop as soon as 1–2 months after lesion onset.

Localized Precancerous Hyperkeratosis of the Vermilion Border of the Lips

(*Hyperkeratosis praecancrosa circumscripta*) was first described by A.L. Mashkilleison in 1965. The condition predominantly affects middle-aged men and is typically localized on the lateral aspect of the lower lip's vermilion border.

Clinical presentation:

The lesion appears as a small, polygonal keratinized area, ranging from a few millimeters to 1.5 cm in size. It may be slightly depressed or, in some cases, subtly elevated due to the accumulation of tightly adherent grayish scales that cannot be scraped off. Upon palpation, the surface feels firm, though no deep induration is detected. Patients usually do not report symptoms, though some may note a cosmetic concern.

Histopathological features:

Microscopic examination reveals limited epithelial proliferation involving the prickle cell layer, marked hyperkeratosis, dyscomplexation, and cellular polymorphism. A polymorphocellular infiltrate is observed within the underlying connective tissue.

Disease progression:

This form of hyperkeratosis shows a relatively low potential for malignant transformation. The lesion may remain stable for months or even years. However, signs of malignant change—such as erosion, surface hardening, or increased keratinization—can appear abruptly, requiring close clinical surveillance.

Differential diagnosis:

It must be differentiated from leukoplakia, lichen planus, and discoid lupus erythematosus. Key distinguishing features include:

- smaller size and polygonal shape,
- tendency to form surface scaling,
- absence of characteristic findings such as Wickham’s striae (lichen planus) or atrophic scarring and diffuse inflammation (lupus erythematosus).

Diagnosis and treatment:

The gold standard for diagnosis is histological examination of a biopsy specimen. The presence of epithelial proliferation, hyperkeratosis, and cellular polymorphism confirms the condition. Treatment consists of complete surgical excision within healthy tissue margins.

Abrasive Precancerous Cheilitis Manganotti (*Cheilitis abrasiva praecancrosa Manganotti*) was first described in 1933 by the Italian dermatologist Manganotti. It predominantly affects men over 50 years old. The disease is influenced by age-related trophic changes in lip tissues—especially the lower lip—and by chronic irritants that provoke low-grade destructive processes in susceptible individuals. Metabolic disturbances, gastrointestinal dysfunction, and vitamin A deficiency are also considered contributory in its pathogenesis.

Clinical Presentation.

One (rarely two) erosive lesion develops on the vermilion border of the lip, typically oval, rounded, or irregular in shape, often offset from the midline. The lesion surface is smooth and bright red, sometimes covered by a thin epithelial layer or crusts. There is no induration at the

lesion's margins. These erosions may persist for long periods, sometimes spontaneously epithelializing and later recurring at the same or another site. Lesion size commonly ranges from 5 to 15 mm.

Histopathology.

Histologic examination reveals epithelial defects within the lesion. At lesion borders, the epithelium may proliferate and in some instances form epithelial islands. In the lower prickle and basal cell layers, there are signs of cellular discohesion (dyscomplexation). The underlying connective tissue shows dense infiltration by lymphocytes and plasma cells.

Differential Diagnosis.

Manganotti cheilitis must be distinguished from erosive variants of discoid lupus erythematosus, lichen planus, leukoplakia, pemphigus, erythema multiforme, herpetic erosion, and primary syphilitic lesions. Clinically, the hallmark is a bright red, often oval erosion without bleeding tendency, lacking papular formations, persistent hyperemia, or characteristic elements of these other disorders.

Treatment.

The first step is elimination of causative irritants and treatment of concomitant systemic diseases. In the absence of cytologic evidence for malignancy, conservative therapy is initiated: vitamin A and nicotinic acid, nerobol (if used locally), and topical applications of vitamin A, methyluracil, corticosteroid creams, and solcoseryl. If the erosion does not re-epithelialize within about one month, surgical excision is indicated. If no signs of malignant transformation are present, conservative therapy may be continued for 2–3 months. Recurrent lesions, increased severity, or any indication of malignancy warrant surgical removal with histological evaluation. Elimination of bad habits, removal of irritants, oral sanitation, and rational oral hygiene are mandatory.

Conclusion:

Key signs of malignant transformation include a sudden change in the clinical picture, such as rapid growth of a tumor or ulcer, exophytic tissue growth around the defect or ulceration of hyperplastic areas. Additional warning signs are bleeding of the lesion, development of hyperkeratosis, infiltration, and induration at the lesion base.

Failure to respond to conservative treatment within 7–10 days is a reason to refer the patient to an oncologist or maxillofacial surgeon. Malignancy is confirmed by morphological studies showing atypical cells in biopsy material.

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