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POTENTIALLY MALIGNANT DISORDERS OF THE ORAL CAVITY – CLINICAL FEATURES

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პირის ღრუს პოტენციურად ავთვისებიანი მდგომარეობები - კლინიკური მახასიათებლები

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doi: https://doi.org/10.52340/jecm.2022.03.13

რეზიუმე

პირის ღრუს კიბო წარმოადგენს მნიშვნელოვან პრობლემას მთელს მსოფლიოში. პირის ღრუს კვლევის ზოგადი ხელმისაწვდომობის მიუხედავად, ბევრი ავთვისებიანი დაავადება დიაგნოსტირდება გვიან ეტაპზე. კიბოს განვითარებას პრეკანცერული მდგომარეობების ფართო სპექტრი უწყობს ხელს, როგორიცაა ლეიკოპლაკია, ერითროპლაკია, ორალური ბრტყელი ლიქენი, დისკოიდური წითელი მგლურა, მემკვიდრეობითი დარღვევები, როგორიცაა თანდაყოლილი დისკერატოზი და ბულოზური ეპიდერმოლიზი.

კიბოსწინარე მდგომარეობების ავთვისებიანი ტრანსფორმაციის თავიდან ასაცილებლად, შემუშავდა მრავალი სკრინინგი და დიაგნოსტიკური ტექნიკა. კიბოს ადრეული გამოვლენა კრიტიკულად მნიშვნელოვანია, რადგან გადარჩენის მაჩვენებლები მნიშვნელოვნად უმჯობესდება დაავადების ადრეულ ეტაპზე დიაგნოზირების დროს.

Cancers of the oral cavity represent approximately three percent of all malignancies in men and two percent of all malignancies in women and is a significant worldwide health problem [1]. Squamous cell carcinoma, which arises from the oral mucosal lining, accounts for over 90 percent of these tumors. Despite remarkable advances in treatment modalities, the 5-year survival rate has not significantly improved over the past several decades and still hovers at about 50-60% [2].

A wide array of conditions has been implicated in the development of oral cancer, including leukoplakia, erythroplakia, palatal lesion of reverse cigar smoking, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, and hereditary disorders such as dyskeratosis congenital and epidermolysis bullosa [3].

Despite the general accessibility of the oral cavity during physical examination, many malignancies are not diagnosed until late stages of disease. In order to prevent malignant transformation of these precursor lesions, multiple screening and detection techniques have been developed to address this problem. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage.

The etiology of precancerous lesions of oral mucosa is not well-known. Some risk factors such as tobacco chewing, tobacco smoking, and alcohol play an important role in development of potentially malignant oral conditions. While tobacco chewing is a major risk factor for oral leukoplakia, OSMF, and erythroplakia, tobacco smoking may be a risk factor for oral leukoplakia. Alcohol drinking may increase the risk by 1.5-fold for oral leukoplakia, by 2-fold for OSMF, and 3-fold for erythroplakia. [4,6]. Studies have shown that moderate to heavy drinkers have a 3-9 times greater risk of developing cancer. In fact, the heavy use of alcohol and tobacco combined may convey a risk greater than 100 times the general population [7].

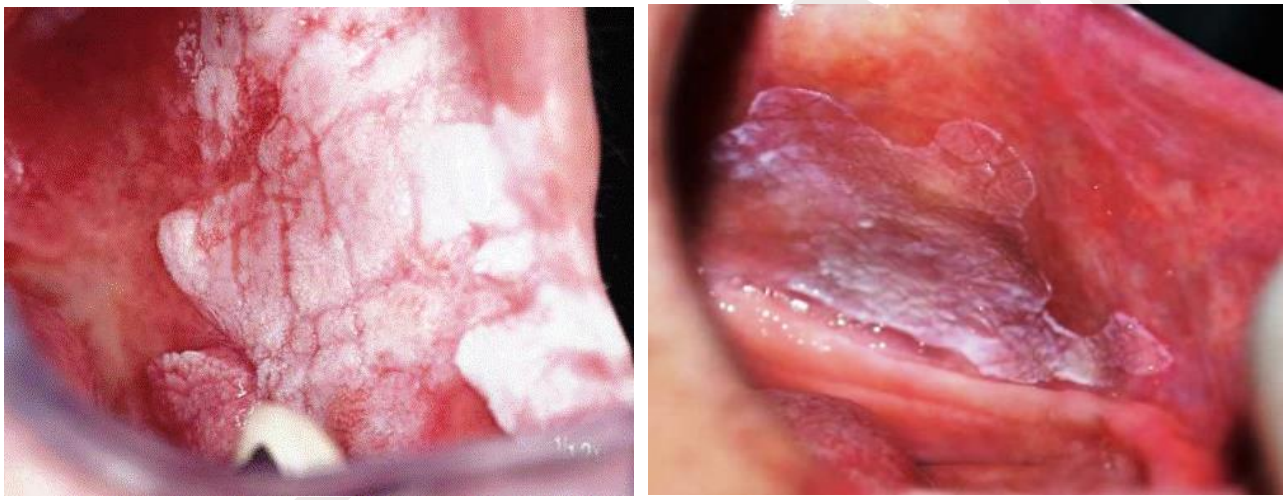
Early detection of premalignant lesions and oral cancer is very important. Therefore, miscellaneous modalities such as oral cavity examination, supravital staining, oral cytology and optical technologies including spectroscopy, fluorescence spectroscopy, elastic scattering (reflectance)

spectroscopy, Raman spectroscopy, fluorescence imaging, optical coherence tomography, narrow-band imaging, and multimodal optical imaging may be used [5].

The following criteria should be taken into consideration in terms of the importance of early diagnosis: (1) symptomatic and/or non-symptomatic non-healing lesions of oral mucosa; (2) history of smoking, chewing tobacco, alcohol consumption, oral HPV infection, drug use, long-term exposure to sunlight; (3) advanced age; (4) the presence of immunodeficiency; (5) the presence of genetic disease; and (6) poor oral hygiene.

ORAL LEUKOPLAKIA

The term *leukoplakia* was first used by Schwimmer in 1877 to describe a white lesion of the tongue, which probably represented a syphilitic glossitis. The definition of leukoplakia has often been confusing and controversial. In the evaluation of the patient, leukoplakia is a clinical diagnosis of exclusion. If an oral white patch can be diagnosed as some other condition (e.g., candidiasis, lichen planus, etc.), then the lesion should not be considered to be an example of leukoplakia. Sometimes a white patch is initially believed to represent leukoplakia, but the biopsy reveals another specific diagnosis. [8,9,30].



Photos by Prof. A.Katsitadze

In studies reported in recent years, the prevalence of oral leukoplakia varies between 1.1% and 11.7%, with a mean value of 2.9%. Although leukoplakia can occur at any age, it often occurs in individuals under the age of 40. Leukoplakia is seen six times more among smokers than among non-smokers [11].

Table 1. Histopathological Nature of Leukoplakia by Site (3,360 Biopsy Specimens) [10].

Site	% Of Leukoplakias at this site	% Of Leukoplakias at this site that showed dysplasia or carcinoma
Lips	10.3	24.0
Maxillary mucosa and sulcus	10.7	14.8
Mandibular mucosa and sulcus	25.2	14.6
Palate	10.7	18.8
Buccal mucosa	21.9	16.5
Tongue	6.8	24.2
Floor of mouth	8.6	42.9
Retromolar	5.9	11.7
Total	100.0	19.9

Clinically, leukoplakia may be affecting any part of the oral and oropharyngeal cavity and can be divided into two subtypes including homogeneous and non-homogeneous types. Homogenous lesions are characterized by uniformly flat, thin, uniformly white in color and shows shallow cracks of the surface

keratin. Non-homogenous lesions have been defined as a white and red lesion (known as *erythroleukoplakia*) that may be either irregularly flat (speckled) or nodular. Verrucous leukoplakia is yet another type of non-homogenous leukoplakia. Proliferative verrucous leukoplakia, which is a form of verrucous leukoplakia, was first described by Hansen et al. in 1985 and characterized by multifocal presentation. It has a strong potential for malignant transformation and is resistance to treatment [12].

Histopathologically, two distinct appearances may be seen as dysplastic or non-dysplastic leukoplakia. Risk factors of malignant transformation are shown in Table 2.

Once a definitive diagnosis of leukoplakia has been made, the risk of malignant transformation should be evaluated. The rate of all clinical subtypes of leukoplakia is estimated to be approximately 2% to 3% per year. Table 2 lists the numerous identified risk factors for malignant transformation. Of these factors, epithelial dysplasia and nonhomogeneous clinical subtype are the most important indicators for malignant transformation. However, it should be recognized that not all dysplastic lesions progress to malignancy. Some remain clinically unchanged and others may regress spontaneously or after elimination of the causative agent, such as cessation of smoking. In addition, malignant transformation may also occur in nondysplastic leukoplakia [28,29].

Table 2. Risk Factors for the Conversion of Oral Leukoplakia into Oral Squamous Cell Carcinoma

Main risk factors	Presence of epithelial dysplasia
	Nonhomogeneous clinical subtype
	Large size
	Location on the tongue and/or floor of the mouth
Other risk factors	Female gender
	Long duration of oral leukoplakia
	Leukoplakia in nonsmokers

Oral leukoplakia should be distinguished from miscellaneous benign and/or potentially malignant disorders that may be seen white or predominantly white diseases of the oral mucosa. The diseases should be considered in the differential diagnosis including aspirin burn, chemical injury, oral pseudomembranous and hyperplastic candidiasis, frictional lesions, oral hairy leukoplakia, leukoedema, linea alba, lupus erythematosus, morsicatio buccarum, papilloma and allied lesions, mucous patches in secondary syphilis, tobacco-induced lesions, smoker's palate (nicotinic stomatitis), stuff-induced lesion, white sponge nevus, oral lichen planus (OLP), and lichenoid reaction [13].

Oral leukoplakia should be confirmed by mucosal biopsy. But before biopsy, some staining methods may be used as a diagnostic aid. Chen et al. used methylene blue in patients with suspicious oral cavity lesions. They reported that the overall sensitivity of methylene blue uptake in cases with suspected lesions was 90%, specificity 69%, and accuracy 79%. They also reported that the positive predictive value was 74% and the negative predictive value 87% [14].

The most commonly preferred treatment options are surgical excision or CO_2 laser therapy. In widespread lesions, photodynamic therapy may be considered. Cryotherapy is another preferred destructive method. Non-surgical treatment modalities might be considered in selected patients. Carotenoids (β-carotene, lycopene), vitamins [L-ascorbic acid (vitamin C), α-tocoferol (vitamin E), retinoic acid (vitamin A), and fenretinide], and bleomycin may be used in patients with oral leukoplakia [12].

Surgical excision should be recommended in the presence of moderate and severe epithelial dysplasia. Reported recurrence ratios after surgery treatment have been varied between 10% and 35%. Kawczyk-Krupka et al. [15] compared to efficacy of cryotherapy and photodynamic treatment and reported that complete responses were obtained in 72.9% and 89.2% of patients in groups treated by photodynamic treatment and cryotherapy, respectively. Pietruska et al. reported significant reduction (on average by 53.8%) of leukoplakia lesions sizes after photodynamic therapy. Among patients treated by topical retinoic acid, while complete response ratio was reported between 10% and 27% of patients, partial

response ratio was reported between 54% and 90% of patients. Recurrence of leukoplakia was reported as approximately 50% after withdrawing the topical retinoic acid [16].

ORAL ERYTHROPLAKIA

The term *erythroplasia* was originally used by Queyrat to describe a red, precancerous lesion of the penis [17]. The term *erythroplakia* is used for a clinically and histopathologically similar process that occurs on the oral mucosa. Similar to the definition for leukoplakia, erythroplakia is a clinical term that refers to a red patch that cannot be defined clinically or pathologically as any other condition. This definition excludes inflammatory conditions that may result in a red clinical appearance. Oral erythroplakia occurs most frequently in older men and appears as a red macule or plaque with a soft, velvety texture. The floor of mouth, lateral tongue, retromolar pad, and soft palate are the most common sites of involvement. Often the lesion is well demarcated, but some examples may gradually blend into the surrounding mucosa. Some lesions may be intermixed with white areas (erythroleukoplakia). Erythroplakia is often asymptomatic, although some patients may complain of a sore, burning sensation [18]. Prevalence of erythroplakia varies between 0.02% and 0.83%. It mainly occurs in the middle aged and the elderly. Male gender is most frequently affected. Mostly, a solitary lesion occurs over the surface of any part of the oral cavity. But the most commonly affected areas were reported as the soft palate, the floor of the mouth, and the buccal mucosa. Etiopathogenesis is not known exactly. Chewing tobacco and alcohol use are the possible etiologic factors for the development erythroplakia [19,29].

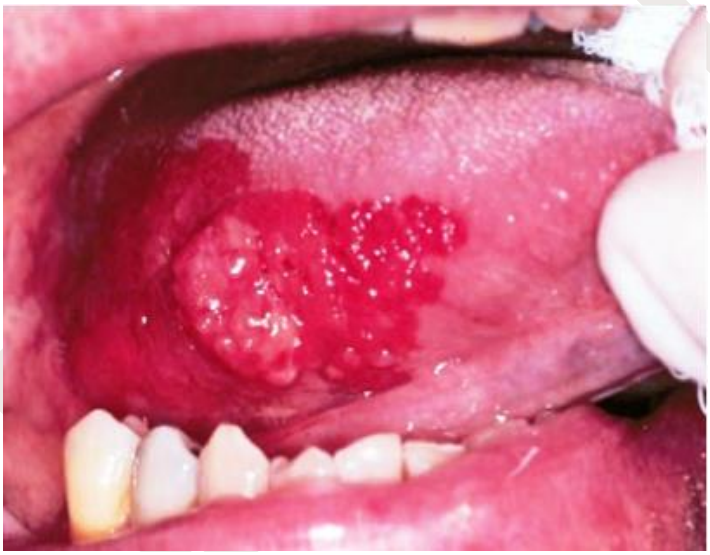


Photo by Prof.A.Katsitadze

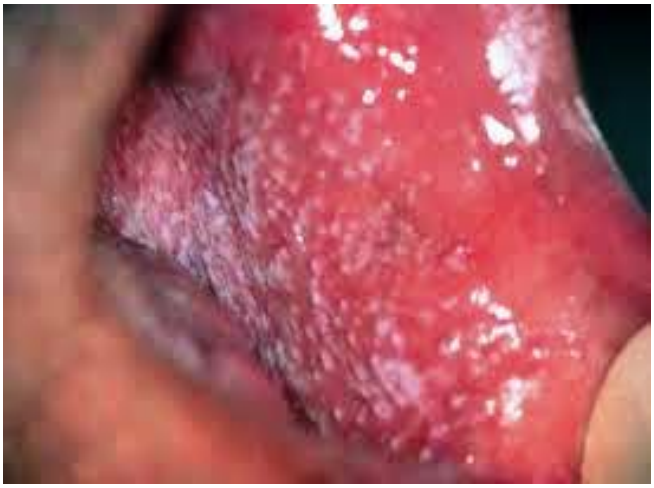
Clinically, typical lesion of oral erythroplakia is less than 1.5 cm in diameter, but it also be less than 1 cm and larger than 4 cm. Histopathologically, moderate or severe dysplasia was usually seen in lesion with erythroplakia. Malignant transformation rates are very high (vary from 14% to 50%), so it needs to be treated expeditiously.

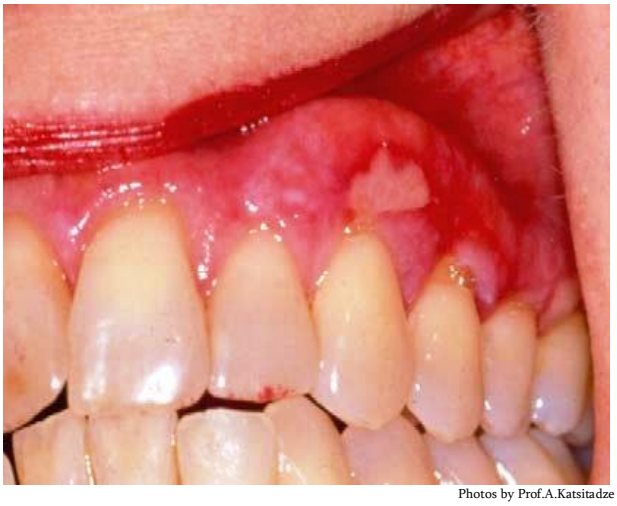
Oral erythroplakia should be diminished from any disease which clinically appears red color in oral cavity. Oral candidiasis, oral histoplasmosis, oral tuberculosis, atrophic OLP, lupus erythematosus, pemphigus, pemphigoids, amelanotic melanoma, hemangioma, telangiectasia, lingual varies, Kaposi's sarcoma, early squamous cell carcinoma, local irritation, mucositis, drug reaction, median rhomboid glossitis, and oral purpura may be confused with oral erythroplakia. Owing to the high malignant transformation rate, early effective treatment is mandatory. Surgery, either by cold knife or by laser, is the recommended therapy. Surgical excision may be used in lesions with severe epithelial dysplasia or carcinoma *in situ* [19].

ORAL LICHEN PLANUS

Lichen planus was first described by Erasmus Wilson in 1869. The disease is a chronic, autoimmune, inflammatory disease which may affect skin, oral mucosa, genital mucosa, scalp, and nails.

Prevalence of OLP varies from 0.5% to 3%. It mainly occurs among female gender and the age of onset is usually between third and sixth decade [20].





Photos by Prof.A.Katsitadze

Although it is believed that OLP is a T-cell mediated autoimmune disease, its cause is partially understood in most cases. Several factors have been proposed for the etiology including genetic background, dental materials (amalgam, metals, gold, and composite restorations), drugs (especially antimalarials, cardiovascular agents, gold salts, non-steroidal anti-inflammatory drugs, hypoglycemics), infectious agents (herpes simplex virus, Epstein-Barr virus, cytomegalovirus, herpes virus-6, hepatitis-C virus, and human papilloma virus), autoimmunity, immunodeficiency, food allergies, stress, habits, trauma, diabetes and hypertension, malignant neoplasms, and bowel disease [1,2].

Even though OLP may affect any part of the oral mucosa, most commonly affected areas are dorsum of the tongue, buccal mucosa, and gingiva. Clinically, OLP may be seen as six types including papular, reticular, plaque-like, atrophic, erosive, and bullous type. The most common type is the reticular pattern which present as fine white striae known as "*Wickham's striae*". Typically, lesions present symmetrically and bilaterally, and usually asymptomatic. Atrophic pattern presents as a red lesion. Erosive pattern is usually seen as irregular erosion or ulceration covered with a fibrinous plaque or pseudomembrane. Both atrophic and erosive pattern are generally associated with a burning sensation and pain that exacerbated by trauma and hot, spicy or acidic foods. Plaque type clinically resembles leukoplakia because of its homogenous white nature. The dorsum of the tongue and buccal mucosa are the most affected areas in the oral cavity of patients with plaque type OLP. Multifocal plaque type lesions may be seen. This subtype is more common among tobacco smokers. The papular pattern, which is rarely seen, is characterized by small, white, raised papules with fine white striation at the periphery of the lesion. Bullous pattern is the least common type of OLP that characterized by bullae formation range from a few millimeters to several centimeters in diameter [21, 31].

The first case of OLP-related oral carcinoma was reported by François Henri Hallopeau in 1910. Malignant transformation ratio has been reported in 0% to 10% of patients, according to the sample's characteristics and study design, after mean follow-up of 1.5 to 10 years. Increased malignant transformation risk occurs greater in erosive and atrophic forms and in cases of lesions of lateral border of the tongue [20].

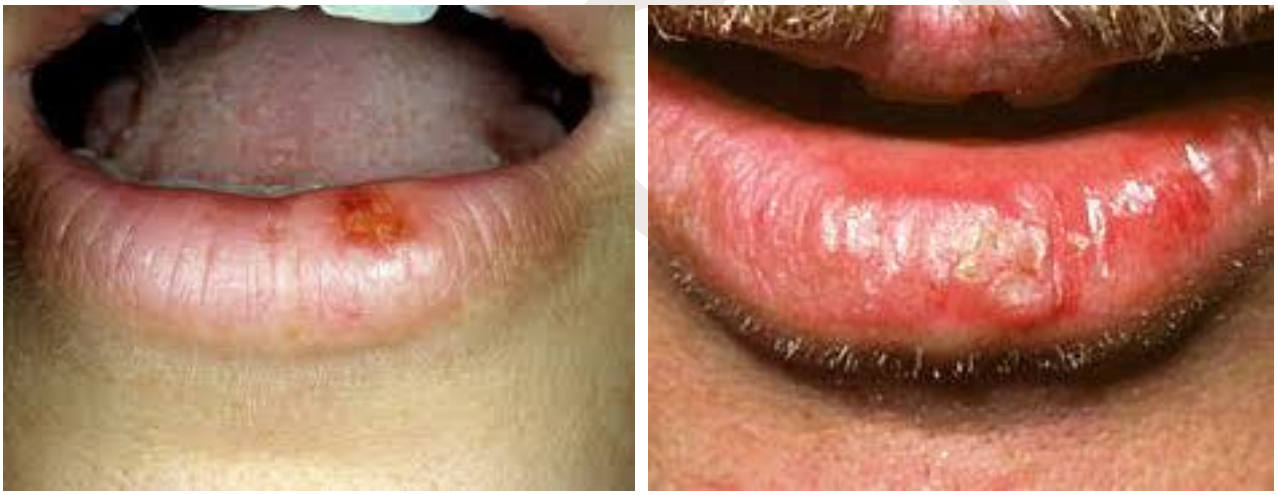
There has been considerable controversy as to whether oral lichen planus inherently harbors malignant potential. It is currently believed that the risk of malignant transformation is low. Risk factors that increase the likelihood of developing oral cancer are long-standing disease, erosive or atrophic types, tobacco use, and possibly esophageal involvement. Additionally, oncogenic subtypes of HPV, including type 16, are more common in oral lichen planus and may account, in part, for the malignancy risk. The reported rates of SCC development have varied: 0.8% of oral lichen planus in the United States, 1.9% in the United Kingdom, 0.6% in China, and 1% in the Swedish population. The majority of these cases are in situ carcinoma or with a microinvasive pat- tern. The most common site for cancer is the tongue followed by the buccal mucosa, gingiva, and, rarely, the lip [32,33,34].

If there are Wickham's striae typically, the diagnosis is easy and can be made clinically, especially reticular pattern of OLP. But erosive or atrophic pattern need to be confirmed by biopsy in order to make the correct diagnosis. Direct immunofluorescence may be useful to distinguish from some bullous diseases such as pemphigus vulgaris, benign mucous membrane pemphigoid, and linear immunoglobulin A (IgA) bullous dermatitis. IgA, IgG, IgM or C3 deposition throughout the basement membrane and irregular fibrinogen deposition in the basement membrane are the diagnostic immunofluorescence findings in OLP and positivity rate is 65.8% of the patients with OLP. Indirect immunofluorescence studies are not useful in terms of diagnosis [22, 23, 27].

Patients with reticular and other asymptomatic OLP can be followed without treatment. But if there are any symptoms and/or potential malignant risk, lesions should be treated.

ACTINIC CHEILITIS

Actinic cheilitis is a potentially malignant disease of the lip caused by exposure solar radiation. It is commonly seen the surface area of the lower lip due to the anatomic proximity. In addition to solar rays, tobacco use, lip irritation, poor oral hygiene, and ill-fitting dentures may play a role in the development of actinic cheilitis. The disease predominantly occurs in men compared to the women. While actinic cheilitis shows erythema and edema in the early stages of the disease, diffuse scaling, thickened epithelium with small greyish-white plaques (known as *leukoplakia*), inflammatory areas (known as *erythroleukoplakia*), and linear fissures may present in the late stages of the disease. Malignant transformation rate has been estimated ranging from 1.4% to 36% at an interval of 1 to 30 years [24, 25].



Photos By Prof.A.Katsitadze

Diagnosis should be confirmed by biopsy to evaluate the degree of dysplasia. Histopathologically, hyperplasia, acanthosis or atrophy of the epithelium, thickening of the keratin layer, and/or dysplasia, which may range from mild to severe, may be shown. In addition to these epithelial changes, in connective tissue, basophilic degeneration of collagen fibers, known as solar elastosis, is usually detected. In treatment, 5-fluorouracil, scalpel vermilionectomy, chemical peel, electrosurgery, cryosurgery, CO_2 laser, imiquimod, photodynamic treatment, diclofenac 0.3% gel can be preferred [26, 35].

With the development and success of screening programs for breast, cervical, and colon cancer, the potential to reduce the morbidity and mortality of oral cancer through early detection modalities is of critical importance. The approaches to the screening and detection of malignant and potentially malignant conditions have the potential to drastically alter the course of oral cavity disease but have yet to effectively reduce the overall morbidity and mortality of oral cancer. The major modalities designed to reduce this burden include oral cavity examination, supravital staining, oral cytology, chemiluminescent technique, and optical detection systems.

Because most individuals are seen more commonly by primary care physicians and general dentists than by specialists, it is important for these clinicians to perform screening examinations to identify potential premalignant oral lesions.

Table 3 summarizes the recommended components of an oral cancer examination. When a suspicious lesion is identified, a conventional biopsy using a scalpel or small biopsy forceps remains the best and most accurate means of assessing it.

Table 3. Components of an Oral cancer and premalignant lesion Examination

1	**Extraoral examination**	Inspect head and neck. Bimanually palpate lymph nodes and salivary glands
2	**Lips**	Inspect and palpate outer surfaces of lip and vermilion border. Inspect and palpate inner labial mucosa.
3	**Buccal mucosa**	Inspect and palpate inner cheek lining.
4	**Gingiva/alveolar ridge**	Inspect maxillary/mandibular gingiva and alveolar ridges on both the buccal and lingual aspects.
5	**Tongue**	Have patient protrude tongue and inspect the dorsal surface. Have patient lift tongue and inspect the ventral surface Grasping tongue with a piece of gauze and pulling it out to each side, inspect the lateral borders of the tongue from its tip back to the lingual tonsil region Palpate tongue
6	**Floor of mouth**	Inspect and palpate floor of mouth
7	**Hard palate**	Inspect hard palate
8	**Soft palate and oropharynx**	Gently depressing the patient's tongue with a mouth mirror or tongue blade, inspect the soft palate and oropharynx

CONCLUSIONS

The ability to control oral cancer and premalignant condition will depend on two cornerstones: prevention and early diagnosis. Continuing educational campaigns are needed on the local, state, and national level in order to educate the public about the risk factors and early signs/symptoms associated with these diseases. Individuals also need to be encouraged to seek regular professional oral examinations by a dentist and/or dermatologist. Finally, health care workers must be encouraged to perform oral examinations as part of their patient care regime, and to be knowledgeable about early signs of oral premalignant lesions.

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ПОТЕНЦИАЛЬНО ЗЛОКАЧЕСТВЕННЫЕ РАССТРОЙСТВА ПОЛОСТИ РТА – КЛИНИЧЕСКИЕ ОСОБЕННОСТИ

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РЕЗЮМЕ

Рак полости рта представляет значительную проблему по всем мире. Несмотря на доступность физического осмотра полости рта, многие злокачественные состояния не диагностируются до поздних этапов заболевания. Широкий спектр заболевания играет важную роль в развитии рака в полости рта, в том числе лейкоплакия, эритроплакия, оральный красный лишай, дискоидная волчанка и наследственные расстройства, как буллезный эпидермолиз.

Чтобы предотвратить злокачественную преобразование этих поражений, было разработано множественные методы скрининга и обнаружения. Раннее обнаружение рака имеет критическое значение, поскольку выживаемость заметно улучшается при определении орального поражения на ранней стадии.

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POTENTIALLY MALIGNANT DISORDERS OF THE ORAL CAVITY – CLINICAL FEATURES

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SUMMARY

Cancers of the oral cavity represent a significant worldwide health problem. Despite the general accessibility of the oral cavity during physical examination, many malignancies are not diagnosed until late stages of disease. A wide array of conditions has been implicated in the development of oral cancer, including leukoplakia, erythroplakia, palatal lesion of reverse cigar smoking, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, and hereditary disorders such as dyskeratosis congenital and epidermolysis bullosa

In order to prevent malignant transformation of these precursor lesions, multiple screening and detection techniques have been developed to address this problem. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage.

Keywords: oral cavity, malignant, leukoplakia, erythroplakia, lichen planus