World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-19, February 2023
ISSN: 2749-3644

THE EFFECT OF TOBACCO SMOKING ON THE ORGANS AND TISSUES OF THE ORAL CAVITY

Karshieva Dilovar Rustamovna
Bukhara State Medical Institute named after Abu Ali ibn Sino, Uzbekistan

Article history:

Received: December 11th 2022
Accepted: January 11th 2023
Published: February 20th 2023

Abstract:

A person who smokes one pack of cigarettes a day makes more than 70,000 puffs a year, while the constituent components of tobacco smoke can affect the human body directly through the mucous membranes of the oral cavity, airways and respiratory sections of the lungs, as well as indirectly - being absorbed into the blood . At the same time, the organs and tissues of the oral cavity are the site of the primary contact of the smoker's body with the components of tobacco smoke. The resulting changes in the oral mucosa and minor salivary glands can be the first early symptoms for diagnosing diseases caused by smoking. The nature, mechanism of the damaging effect of tobacco smoke and the resulting changes in the oral mucosa, MSF and oral fluid in smokers are of theoretical and practical interest.

Keywords: Tobacco, oral cavity, sick children, leukoplakia, cytological examination.

In the process of smoking, the organs and tissues of the oral cavity are exposed to irritating, thermal, toxic and carcinogenic effects. The intensity of exposure is determined by many factors, including individual morphological and functional features of the oral mucosa, the duration and intensity of smoking.

The physical and chemical injury caused by tobacco smoke depends on the type and quality of tobacco, its growing conditions (the use of mineral fertilizers, pesticides) and drying. In the mechanism of the damaging effect on the tissues of the oral cavity, temperature indicators are of great importance. The glowing end of a cigarette has a temperature of about 300°C. Tobacco smoke passing from the smoldering cone of a cigarette through a layer of tobacco stuffing to the mouthpiece end has time to cool, but not enough to equal the temperature of the oral cavity. The temperature of inhaled tobacco smoke varies between 50° and 60°C. The temperature of the air entering the oral cavity during smoking is 35° - 40°C below the temperature of tobacco smoke. Such significant temperature fluctuations occur from 10 to 20 times during the smoking of one cigarette.

Tobacco companies, faced with a decline in cigarette sales in developed countries at present, are promoting the habit of chewing tobacco by releasing tobacco tablets for oral use. Currently, there is a significant increase in the consumption of smokeless tobacco by adolescents and young men, considering that this way of using tobacco relieves stress and causes relaxation without harming the body. However, smokeless tobacco, due to its high nicotine content, is as harmful as smoking. Oral tobacco use causes an increase in the incidence of diseases of the organs and tissues of the oral cavity with precancerous lesions and cancer.

THE EFFECT OF SMOKING ON THE MUCOUS MEMBRANE OF THE ORAL CAVITY

Among diseases of the oral mucosa in tobacco smokers, chronic catarrhal stomatitis, cheilitis and various forms of leukoplakia are more often diagnosed, which can occur against the background of chronic catarrhal stomatitis or cheilitis.

Chronic stomatitis and cheilitis of smokers has a peculiar clinical and morphological picture. At the beginning of the disease, the mucous membrane partially loses its luster, becomes dull, bluish-red in color, then areas with a whitish tint appear, which, according to the authors, is associated with the appearance of foci of keratinization of the integumentary epithelium. At the same time, tissue swelling, characteristic of serous stomatitis and cheilitis of other origin, is absent. With a long-term existence of the process, the mucous membrane acquires a bluish-brown appearance, becomes as if compacted, and in advanced cases, even its focal atrophy may appear.

The conducted studies suggest that tobacco smoke has a pronounced damaging effect on the epithelium of the oral mucosa. It leads to accelerated death and desquamation of cells of the surface layer of the epithelium, the appearance of keratinization foci and cells of the basal growth layer in it, as well as to the development of fibrosis of the submucosal layer. All this contributes to the functional restructuring and weakening of the histohematic barrier of the oral mucosa, which creates optimal conditions for the introduction and reproduction of pathogenic microflora and its penetration into the underlying tissues.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-19, February 2023
ISSN: 2749-3644

Leukoplakia, according to most researchers, should be considered as a precancerous process. According to domestic and foreign authors, it is detected in 22-38% of cigarette smokers, in 13.8% of cigar smokers and in 28.3% of pipe smokers. There was no statistically significant difference between the effect of smoking cigarettes with or without a filter on the development of leukoplakia. Leukoplakia is 4 times more common in men than in women, in 59.7 - 64.3% of patients aged 40 to 70 years. Leukoplakia in smoking patients is mainly detected in areas of the oral mucosa and the red border of the lips, which, during smoking, are constantly irritated by tobacco components smoke. Most authors indicate that the process is more often localized on the mucous membrane of the cheeks - 48.4%, lips - 25.7%, on the tongue - 9.0%, in the sky - 5.9%, in the area of the bottom of the oral cavity - 2 .1% and in other areas - 8.9%. On the mucous membrane of the cheeks, the lesions in most cases are located in the corners of the mouth - 65.2%. The foci of leukoplakia can be localized in one or more areas of the oral cavity.

At present, unfortunately, there is no generally accepted clinical and morphological classification of leukoplakia. The classifications previously proposed and used in the clinic do not always satisfy the growing demands of modern practical and theoretical medicine and are not without drawbacks, in particular: they do not take into account the etiopathogenetic mechanisms of the development of leukoplakia. Most authors distinguish the following forms of the disease: flat (simple) leukoplakia, verrucous leukoplakia, which can be represented by a plaque variant of the structure or warty, erosive-ulcerative leukoplakia and Tappeiner's leukoplakia. Such a classification principle does not allow us to talk about the etiology and pathogenesis of the disease, compare statistical data obtained by different authors, does not give a comprehensive idea of this pathology in general, as well as its possible outcome and prognosis.

As shown by studies of domestic and foreign authors, among all clinical and morphological forms of leukoplakia, the flat form of the disease is determined in 42.6 - 48.3%) of observations. It is characterized by the presence of sharply defined grayish-white spots, of various sizes and shapes, which do not rise above the surrounding mucous membrane and are not removed mechanically. On microscopic examination in places lesions show moderate hyperkeratosis, parakeratosis, and acanthosis. In the underlying tissue, there is edema, congestive plethora, and the presence of perivascular polymorphocellular infiltrates.

The verrucous form of the disease is detected in 34.5–39.0% of cases, including the warty variant in 35.4%, and the plaque variant in 3.6%. Onn are sharply defined plaque or warty keratinization foci of round or irregular shape, grayish-white, slightly dense, tuberous, protruding above the surface of the mucous membrane, sometimes located against the background of flat leukoplakia. Histological examination in these formations is determined by pronounced hyperkeratosis without loosening of the stratum corneum, parakeratosis and acanthosis with unevenly expressed epidermal outgrowths. The outgrowths of the spiny layer are thickened, and in some groups of cells intracellular edema is determined. In the submucosal layer, edema, congestive plethora of dilated vessels are revealed, around which focal lymphocytic infiltrates are observed.

Among all clinical and morphological forms of the disease, erosive-ulcerative leukoplakia is diagnosed in 10.8–22.9% of cases. Most modern clinicians and morphologists consider this form of damage to the oral mucosa as the result of a complication of simple, plaque or warty variants of leukoplakia. It is characterized by the presence of single or multiple erosions or ulcers of various shapes and sizes, usually occurring against the background of keratinized lesions oral cavity. At the same time, cracks can also occur. Morphological examination reveals an epithelial defect, along the edges of which hyperkeratosis, parakeratosis and acanthosis are found with elongation of epithelial outgrowths. In the underlying tissue, pronounced inflammatory changes are noted, accompanied by hyperemia, edema and the appearance of diffuse inflammation filtrates consisting of lymphocytes with an admixture of plasmocytes and tissue basophils.

Some authors have noted that in leukoplakia, the content of DNA in the nuclei of cells of the integumentary epithelium is not changed even with the onset of malignancy, and the content of RNA in the cytoplasm of cells decreases, especially with malignancy of leukoplakia. Other researchers indicate that RNA and DNA accumulate in epithelial cells in squamous and verrucous forms of leukoplakia.

It has been established that in leukoplakia, the glycogen content in epithelial cells is directly dependent on the nature of keratinization, and not on its clinical and morphological form. If there is parakeratosis-type keratinization, then its content in cells is increased, and in orthokeratosis-type keratinization, it is reduced. It is also known that when the process is malignant, the content of glycogen in epithelial cells decreases. This can be used to diagnose early stages of dysplasia and incipient malignant growth. Depending on the type of

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-19, February 2023
ISSN: 2749-3644

hyperkeratosis that develops with leukoplakia, there were differences in the content of sulfhydryl and disulfide groups. With orthokeratosis, the amount of disulfide compounds increases, and the content of sulfhydryl compounds decreases. In parakeratosis, the content of sulfhydryl groups is increased, while disulfide groups are negligible. In the lesions, a decrease in the production of glycoproteins and by epithelial cells is revealed, especially if the process has a tendency to malignancy. There are no data in the literature on quantitative and qualitative changes in the composition of these compounds in the foci of leukoplakia.

With leukoplakia, regardless of its clinical and morphological form, there is an increase in the activity of such enzymes as lactate dehydrogenase, glucose-6-phosphate dehydrogenase and succinate dehydrogenase. The latter play an important role in the processes of glycolysis and in the pentose cycle. The same changes were noted in malignant tumors.

It is known that smoking cessation can lead to regression of lesions in leukoplakia. Some researchers note that if the factors that caused the occurrence of leukoplakia are not eliminated after radical surgical treatment, the disease can recur in 34.4% of patients. In patients with leukoplakia who continue to smoke, malignancy of the process is possible with the development of cancer "in situ" or invasive squamous cell carcinoma.

The transition of leukoplakia to cancer is preceded by dysplasia of the integumentary epithelium. Dysplasia is understood as controlled and reversible disorders of differentiation of the epithelium (or non-epithelial tissue) of a precancerous nature as a result of the proliferation of cambial elements (undifferentiated progenitor cells, stem cells) with the development of their atypia, loss of polarity and disturbance of the histostructure without basal invasion! membranes. In accordance with the degree of proliferation of epithelial cells and the severity of cellular and structural (tissue) atypia, there are three degrees of dysplasia: I - weak (small, mild), II - moderate (medium) and III - severe (significant, pronounced). At the same time, cellular atypia of the epithelium and violations of the histoarchitectonics of the tissue increase from degree to degree. It is known that changes in I and II degrees of dysplasia are most often reversible. Changes in grade III dysplasia are much less likely to reverse development and are considered precancerous. Sometimes it is difficult to distinguish changes in severe dysplasia from cancer in situ, when tumor growth occurs within the epithelial layer, without transition to the underlying tissue and with the preservation of the basement membrane.

Many authors cite in their works a different percentage of possible malignancy of leukoplakia from 10% to 50%, in most cases in non-smokers, more often in women over the age of 60. Such a spread of indicators can be explained by the fact that these authors analyzed the cent of the glamorization is not the entire group of leukoplakia as a whole, but its individual variants. It is known that different variants of leukoplakia have different degrees of potency for malignancy. Therefore, according to A.L. Mashkilleison and E.V. Borovsky, it would be more correct to determine the percentage of malignancy separately for each form of leukoplakia and thereby determine its place in a number of precancerous processes. They found that leukoplakia transforms into cancer only in 11.9% of cases: in the flat form of the disease, malignancy was noted in 4.2% of patients, in the verrucous form - in 20.0%, and in the erosive-ulcerative form - in 20.0%). Thus, the authors have shown that verrucous and erosive-ulcerative forms of leukoplakia have the greatest potential for malignancy. At the same time, malignancy of the process often occurs within a period of one to five years after the onset of the disease.

Studies by many authors have revealed a correlation between tobacco smoking and the incidence of oral cancer. According to them, cancer of the tongue occurs in 87% of smokers, the floor of the mouth in 94%, and the oropharynx in 99%. This indicates the selective sensitivity of different parts of the oral and pharyngeal mucosa to carcinogenic components of tobacco smoke.

Tappeiner's leukoplakia is detected in 21.3% - 31.6% of smokers, and in "malicious" tobacco smokers in almost 100% of cases. It occurs on the mucous membrane of the hard palate and the anterior soft palate, which in the process of smoking are in direct contact with tobacco smoke. At the same time, against the background of edematous hyperemic mucosa, sometimes with a cyanotic tint, compacted areas of a grayish-white color with a wrinkled, folded surface are visible. In these areas, especially in the posterior half of the palate, red dots are visible, which are gaping orifices of the excretory ducts of the ICF. With a pronounced process, these red dots can be located on top of small hemispherical nodules. Histologically, this form of leukoplakia is characterized by is characterized by hyperkeratosis, which is not accompanied by acanthosis and stromal reaction.

Visually visible depressions in the form of red dots represent dilated openings of the excretory ducts of the salivary glands. At a short distance from the ectatic mouth, there is a sharp narrowing of the excretory duct, below which it is sharply expanded and passes into the

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-19, February 2023
ISSN: 2749-3644

retention cyst of the salivary gland, which is clinically manifested by a nodule. The red rim around the excretory duct is due to desquamation in this area of the epithelium. Tappeiner's leukoplakia is a reversible process, since smoking cessation leads to its disappearance.

In tobacco smokers, all forms of leukoplakia or their simultaneous combination are detected in the same patient.

Currently, the so-called "hairy" leukoplakia (hairy, hairy, villous, oral hairy leukoplakia) of oral cavity is isolated, which is considered a pathognomonic sign of HIV infection and is caused by the Epstein-Barr virus. According to the literature, it is most often localized on the lateral surface of the tongue, less often on the buccal mucosa. The lesions have the appearance of warty growths of the integumentary epithelium of white color, on the surface of which thin whitish hairs are determined, as if adhering. They are painless, but create a feeling of inconvenience and disturb patients with their unusual appearance. Lesions, unlike mild leukoplakia, are not removed with a spatula. Hairy leukoplakia is characterized by focal hyperplasia of the integumentary epithelium of the oral cavity, papillomatosis, hyperkeratosis, moderately pronounced acanthosis, and ballooning epithelial dystrophy. On the surface of the integumentary epithelium, threads of pseudomycelium of a yeast-like fungus of the genus Candida are detected. In the submucosal layer, there is a mild lymphoplasmacytic infiltration.

According to the literature, B.M. Pashkov's mild leukoplakia can be attributed to congenital forms of the disease, in which the familial nature of this disease is noted and cases of its detection in newborns or in early childhood and reaching its maximum severity by adolescence. It is known that congenital malformations of organs and tissues can manifest themselves both prenatally and at various time intervals in the postnatal period. This is very important for understanding the pathogenesis of mild leukoplakia, when it does not occur immediately after the birth of a child, but some time later, in the process of its further growth, eventually causing the appearance and formation of lesions on the oral mucosa. This is also consistent with the data that in the oral cavity in embryos and young children a large number of light cells are determined, similar in structure to the light cells in mild leukoplakia. It is somewhat more common in women, as a rule, without subjective sensations, less often patients complain of roughness of the mucous membrane, peeling, feeling of thickened "extra" tissue. The lesions have the form of narrow whitish strips of soft consistency that rise above the surrounding mucosal mucosa. They are covered with soft, easily removed with a spatula, scales. After removing the scales, a pinkish-white surface remains in this place without any signs of inflammation and erosion. Lesions are more often localized on the mucous membrane of the cheeks, lips, tongue, and less often they can occupy a significant part of the oral cavity. In morphological examination, mild leukoplakia is characterized by focal hyperplasia of the integumentary epithelium, pronounced parakeratosis, moderate acanthosis, the presence of light, as if empty, cells with pycnomorphic nuclei, and the absence of inflammatory infiltration in the subepithelial layer. Unlike other types of leukokeratosis, mild leukoplakia is not malignant.

The association of smoking with the development of candidiasis of the oral mucosa has also been noted. Cases of acute candidiasis after heavy tobacco use. Experiments have confirmed that smoking contributes to the growth of candidal flora, which, apparently, is associated with impaired function of the immunocompetent tissue. It has been established that fungi of the genus Candida are capable of producing carcinogenic nitrosamines. This is confirmed by the frequent combination of candidal infection with precancerous processes and malignant tumors of the oral mucosa.

It is known that long-term smoking leads to the development of secondary immunosuppression. In this case, a deficiency of humoral and cellular immunity occurs, possibly as a result of a defect in the population of both B- and T-lymphocytes or their precursors. It has been established that in smokers there is an increase in the number and activity of the suppressor subpopulation of T-lymphocytes. Tobacco smoking also leads to a decrease in nonspecific resistance of the body, which is manifested by a decrease in the phagocytic activity of polymorphonuclear leukocytes and macrophages, an increase in microbial contamination of the oral mucosa, an increase in the content of opportunistic microorganisms and a change in the ratio between aerobic and anaerobic microorganisms.

Smoking plays a role in the development of tongue diseases, primarily rhomboid glossitis. In chronic smokers, there are also changes in the color of the tongue, hypertrophy of the filiform papillae, and focal desquamation of its integumentary epithelium. At the same time, these objective changes are often accompanied by a number of subjective signs in the form of a perversion or decrease in taste sensations, especially when eating sweet and salty foods.

World Bulletin of Public Health (WBPH)
Available Online at: https://www.scholarexpress.net
Volume-19, February 2023
ISSN: 2749-3644

REFERENCES

1. Almas K., Maroof F., Mcallister C, Freeman R. Smoking behaviour and knowledge in high school students in Riyadh and Belfast. *II* Odontostomat. Trop. - 2002. - Vol. 25. - № 98. - P. 40 - 44.
2. Bendick C, Scheifele C, Reichart P.A. Oral manifestations in 101 Cambodians wit HIV and AIDS. *II* J. Oral. Pathol. Med. - 2002. - Vol. 31. - № 1. - P. 1 -4.
3. Casiglia J., Woo S.B. A comprehensive review of oral cancer. *II* Gen. Dent. -2001. - Vol. 49. - № 1. - P. 72 - 82.
4. Faddy M.J., Gullinan M.P., Palmer J.E. et al. Ante-dependence modeling in a longitudinal study of periodontal disease: The effect of age, gender, and smoking status. *II* J. Periodontol. - 2000. - Vol. 71. - № 3. - P. 454 - 459.
5. Sharipova Gulnihol Idiyevna. DISCUSSION OF RESULTS OF PERSONAL STUDIES IN THE USE OFMIL THERAPY IN THE TREATMENT OF TRAUMA TO THE ORAL MUCOSA// European Journal of Molecular medicinevolume 2, No.2, March 2022 Published by ejournals PVT LTDDOI prefix: 10.52325Issued Bimonthly Requirements for the authors.
6. Sharipova Gulnihol Idiyevna. THE EFFECTIVENESS OF THE USE OF MAGNETIC-INFRARED-LASER THERAPY IN TRAUMATIC INJURIES OF ORAL TISSUES IN PRESCHOOL CHILDREN//Academic leadership. ISSN 1533-7812 Vol:21Issue 1
7. Данилевский Н.Ф., Леонтьев В.К., Несин А.Ф., Рахний Ж.И. Заболевания слизистой оболочки полости рта. - М.: Издательство ОАО "Стоматология", 2001.-272 с.
8. Кулик И.В., Миргородская Л.В. ВИЧ-инфекция. Проявления в полости рта. Журнал: Институт стоматологии. - 2001. - № 2. - С. 36 - 40.
9. Курение как фактор заболеваний пародонта, кариеса и потери зубов. // Стоматологическое обозрение. - 2003. - № 2. - С. 3 - 4.
10. Трубачева И.А., Перминова О.А., Шатров СВ. Популяционные аспекты табакокурения у взрослого населения Томска // III Международная конференция по восстановительной медицине (реабилиталогия). — М.: Злато-граф, 2000. - С. 426 - 427.