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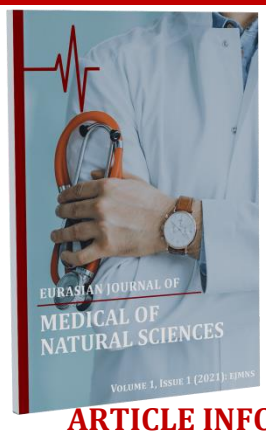

ETIOPATHOGENETIC ASPECTS OF DENTAL DISEASES IN PATIENTS WITH BRONCHIAL ASTHMA (LITERATURE REVIEW)

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ABSTRACT

Insufficient assessment of common somatic diseases can lead to complications during the treatment of patients and lead to inefficiency of dental care. The connection between General somatic diseases and the condition of the oral cavity is made through various types of homeostases (metabolic, immunological, etc.), the violation of which leads to various diseases of the whole organism and the oral cavity

In recent years, more and more attention has been paid to improving the diagnosis and treatment of additional somatic diseases .

Bronchial asthma is a serious problem in all countries of the world, including the Republic of Uzbekistan, which is also associated with a clear increase in the frequency of occurrence, the number of complications, exacerbation of the disease.

Multifactorial is characteristic of the etiology and pathogenesis of inflammatory diseases in the oral cavity, the main place in which is occupied by the microbial factor [2]. A decrease in local and general resistance leads to an increase in the concentration of microorganisms, their toxins and the products of tissue breakdown in the foci of infection [8]. Thus, the damaging effects of microorganisms are manifested either when their quantity and virulence are high and simple mechanisms are not able to neutralize them, or when the activity of local and systemic immunity decreases [3].

The main role in the alternation of Parodont tissues is played by microbe enzymes such as collagenase, elastase, fibrinolysin, chondroitinsulfatase and others [1].

Violation of microcirculation in the area of periapical tissues leads to the development of odontogenic foci chronicling as well as fibrosis changes [1], which, along with an increase in vascular-tissue permeability, is an important pathogenetic condition in the development of diseases of an inflammatory nature [5].

Microcirculation disorders not only play a key role in the pathogenesis of the inflammatory disease parodont, but are also associated with their degree of severity [5]. In

inflammation, a decrease in the indicators of vascular tone, peripheral resistance indices, as well as vascular elasticity is noted in the micro-circulatory flow of the parodont [6].

In order to detect disturbances in the microcirculation flow of the parodont, the method of dopplerography of the parodont vessels is used [9]. On the basis of this method lies the Doppler effect, which consists in calculating the change in the frequency of the signal returning from the moving object with respect to the proportional value to the speed of the reflecting motion.

In dental carash, an active factor - a protein component-has been identified, which affects the vascular permeability of parodont tissues, creating conditions for the formation of the autoimmune component of alterasia [10].

Leukocyte migration is significantly increased as a result of increased permeability of the Parodont vessels as well as the development of acute vasculitis of microbial Genesis, which is noted during the development of gingivitis, which may be one of the reasons for increased lysozyme activity [8].

The processes of carash and stone formation cannot be explained only by the presence of microbial inflammation. Both quality and quantity of oral fluid play an important role in carash and rock formation [10]. As a natural liquid biological environment, saliva plays a huge role in the life activity of teeth and parodont, in the support of homeostasis in the oral cavity. The neutralizing and mineralizing properties of saliva are largely associated with the state of the sour-alkaline balance, the objective indicator of which is considered pH. Hydrogen indicator in mixed saliva - the homeostasis of mineral components in enamel, as well as the head natural straightener of dynamic equilibrium in the process of exchange in the "enamel-saliva" system. The rate of enamel demineralization largely depends on pH: the lower it is, the faster the demineralization process goes.

Thus, a decrease in salivary detachment as well as changes in salivary composition lead to various diseases in the oral cavity. Local as well as systemic immune disorders play a major role in Pyak occurrence as well as chronicling.

I.N. According to Antonova (2020), Pyak found that low concentrations of lysozyme, slgA, and IgG in the oral fluid of existing patients are negative in the prognosis, and that professional hygiene measures in the oral cavity of such patients are less effective.

In recent years, many studies have been carried out, as a result of which the participation of cytokines in the pathogenesis of parodont inflammatory diseases has been proven. It is known that inflammatory diseases of parodont are associated with an increased production of inflammatory cytokines, including interferons.

The idea of an autoimmune component in the pathognesis of Parodont disease was expressed by researchers in the mid-60s [14], when the assumption that parodont osteocytes were damaged by autoantibodies was confirmed.

In later published work, it was the parodont tissue complex that was considered as a target for immune attack [8]. T. Safarov found a significant correlation of parodontal index levels to serum milk antigen with autoantibodies levels in a 1985 study of parodont disorders that harmonize with ulcerative disease.

Fundamental research determining the role and place of autoimmune in periodont pathology L.Yu. It was Orekhova's [12] work. In SUP pathogenesis, reliable evidence has been

obtained on the importance of AT levels to gum tissue as well as its high predictive importance.

An additional contact between dental Carache and periodont tissue can be found in this causes the occurrence of autoimmune processes in tissues that are accompanied by alternative changes [7].

Currently, it is established that in the blood of patients with inflammatory diseases of the parodont, a parodont-specific antigen, antitana to this antigen, circularizing immune complexes (CICs), as well as a number of non-specialized displacements can be observed, testifying to the possibility of developing an autoimmune process during chronic inflammatory formation [9]. A close relationship has been found between autoimmune shifts and the clinical specifics of the disease, as well as its prognosis [9.].

The effect of chemical elements on the condition of parodont tissues in patients with bronchial asthma

In recent times, the study of mineral content in various biological environments in periodont diseases is of particular interest, since information about the qualitative and quantitative composition of micro - and Macroelements in them fully forms the perceptions of the pathogenesis of changes occurring in the oral cavity in periodont diseases [7]. According to a number of authors, the change in the amount of saliva microelements increases with an increase in the degree of damage to the parodont [16]. Structural changes in the parodont are associated with a deficiency of macro - and microelements, which are responsible for many processes in Cell Metabolism. The lack of macro - and microelements in different biological environments leads to an increase in the susceptibility of parodont tissues to infections, a slowdown in reparative activity, metabolic and structural changes. This occurs due to the fact that the life activity and activity of immune-component cells, their participation in immunobiological processes, are closely related to mineral exchange in the body [15]. The state of transmembrane effects in cells depends on the activity of calcium-magnesium and sodium-potassium channels, the activity of complex enzyme systems is determined by the metabolism of chemical elements.

The realization of many immune functions in the body is associated with magnesium. The synthesis of immunoglobulins, the participation of magnesium in the role of a cofactor in the control of the complement system, has been proven. The formation of antibodies in magnesium deficiency, the tendency to autoimmune reactions increases. Magnesium is involved in the processes of mineralization of bone tissue. The lack of magnesium in the body increases the abnormality of atherosclerosis rashes and leads to calcinosis in atherosclerosis-altered vessels [7].

Magnesium is part of 325 cell enzymes that control the formation of ATF, the migration and separation of phosphate groups (sour phosphotase, pyrophosphotase, Atphase) [2.]. Due to its interaction with ATF, magnesium is a prerequisite participant in all reactions in energy metabolism as well as a necessary component of transport systems [6]. Magnesium acts as an activator of more than 300 enzymes. They include ATFs and agonists that transport ions across cell membranes, and adenylacyclaase, which catalyzes the formation of tsatf in receptor interactions [8]. Magnesium thinns the degranulation of the main cells and provides soft muscle relaxation in the bronchi and vessels [10]. Lack of magnesium is of particular

importance when fatigue develops in the respiratory muscles, which plays an important role in the pathogenesis of lung diseases. The biological role of magnesium is determined by its ability to compete with calcium at attachment sites in membranes and proteins [6].

Calcium is a universal intracellular Messenger. By combining with various protein structures of the cell and changing their conformation, calcium ions will be able to trigger or modulate almost all vital functions of the cell. In patients with bronchial-obstructive syndrome, the amount of its entry into the cells increases significantly, without major changes in the volume of calcium excretion. The presence of calcium in large quantities inside the cell activates the contraction of the smooth muscles of the bronchi, the secretion of the main cells in the lungs, the formation of tracheobronchial secretions, as well as an increase in the tone of the wandering nerve. Increased intracellular calcium concentration leads to the formation of bronchial hyperreactivity and the development of ba [19]. An increase in intracellular calcium in patients with Ba can be associated with a strong reaction of lipid peroxide oxidation [20]. Excess lipoperioxidation products disrupt the phospholipid membrane bivalve structure and lead to the formation of hydrophilic areas in it, which ensures high diffusion of the Ion and leads to an increase in its intracellular concentration [7].

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