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Immunological Aspects of Pathogenesis of Periodontal Pathology in Patients with Chronic Pyelonephritis

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Abstract Chronic pyelonephritis often accompanies dental patients. Due to impaired myocardial contractility, as a result of various pathologies of the urinary tract system, an imbalance of vasoconstrictor and vasodilating neurohumoral systems occurs, which leads to insufficient blood supply to organs and tissues, including periodontal tissues, fluid retention occurs in the body, and the mucous membrane is no exception lining of the oral cavity. As a result of developing dystrophic and degenerative changes in the periodontium, periodontal tissues become inflamed, a secondary infection occurs, which leads to the activation of the body's immune response. Both in urinary tract insufficiency and in patients with periodontal pathology, an increase in the levels of cytokines TNF-α and IL-1β is observed. These findings indicate the need to optimize the diagnosis, prevention and treatment of dental diseases in patients with CHF.

Keywords Urinary tract, Generalized periodontitis, Oral mucosa, Periodontal condition in CHF, Cytokines TNF-α and IL-1β

1. Relevance

The principle of body integrity underlies the improvement of the approach to assessing dental health. A comprehensive analysis of the combined pathology of the oral cavity and internal organs allows us to gain an understanding of the essence of the origin of many periodontal diseases dependent on the somatic state. In the last decade, pathology of the cardiovascular system has taken first place in terms of incidence. Cardiovascular diseases are the leading causes of disability and mortality both in our country and abroad. One of the most common complications of these diseases is chronic heart failure (CHF), which is very common among dental patients. Epidemiological data show that among people aged 50-59 years the incidence of heart failure is 1-2%, and among people aged 80-89 years it is 10%. In the USA, among hospitalized patients with cardiovascular diseases over 65 years of age, 50% are patients with heart failure, and 80% are over 75 years of age. In Uzbekistan in 2013, newly diagnosed patients with cardiovascular pathology were 532,015, which was 2225. 5 per 100,000 population. According to official data, in the same year, 89,734 people died from cardiovascular pathology, which is 61.6% of the total mortality population (Institute of Health and Medical Statistics, 2013). The most common causes of chronic heart failure in recent years have become coronary heart disease (CHD) and myocardial infarction (MI), which occur in 60-70% of inpatients and are associated primarily with impaired systolic function of the left ventricle (LV). Among other causes of the development of CHF, dilated cardiomyopathy and rheumatic heart defects should also be noted. In older age groups (over 60 years), arterial hypertension (AH) and a hypertensive heart, associated primarily with the development of diastolic disorders, play a leading role in the development of heart failure, along with ischemic heart disease.

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2. The Purpose of the Study

This is also facilitated by an age-related decrease in the muscle element and increased deposition of fibrous tissue in the myocardium of the elderly. The third most important cause in older age groups is type 2 diabetes mellitus (DM), which, together with hypertension, determines the ever-increasing number of patients with CHF.

3. Research Materials and Methods

The pathogenesis of all of the above pathologies is a deterioration in the ability of the myocardium to fill, an imbalance of the vasoconstrictor and vasodilating neurohumoral systems occurs, as a result of which the blood supply becomes inadequate organs and tissues, including periodontal tissues, fluid retention occurs in the body, and the oral mucosa is no exception.

4. Results and Analyzes

As a result of venous stagnation, it may be pale or bluish,

and in some cases the blood vessels dilate. This is especially clearly visible in the sublingual area. The fragility and permeability of vascular walls increases. The mucous membrane becomes vulnerable, even minor injuries cause bleeding. This symptom manifests itself most clearly in the gum area: the slightest pressure when chewing or brushing teeth leads to bleeding. In the presence of carious teeth, habitual biting of the cheeks and other traumatic effects, painful erosions may appear on the mucous membrane, increasing in size, not healing for a long time, and difficult to treat. Pathological processes are especially difficult when teeth are poorly cleaned of plaque.

Patients with CHF are also characterized by sensitivity disorders: pain, taste, tactile. Unpleasant sensations appear in the form of burning and tingling. They begin at the tip of the tongue, and then spread to the entire tongue, lips, and palate, acquiring the persistent, painful nature of glossodynia. Acute cardiovascular failure is accompanied by the formation of cracks and erosions in the oral mucosa. In addition, CHF leads to the rapid development of degenerative phenomena in the bone part of the periodontium, which causes disruption of the attachment of teeth and often leads to the development of secondary adentia due to periodontal disease. Due to destructive processes in periodontal tissues, patients often experience the development of pathological occlusion. Often, patients with chronic heart failure have a reduced chewing optimum, defined by WHO - the presence of at least 20 intact teeth. With fewer teeth, chewing function is impaired, and patients are forced to limit their food intake and food choice. This leads to insufficient supply of nutrients to the body and even more rapid progression of heart failure. Secondary adentia also leads to disruption of the aesthetic and speech functions of the mouth. Many patients with secondary adentia are forced to limit themselves in communication and lead a more secluded, less active lifestyle. At the same time, a decrease in physical activity and communication function is also a risk factor for the progression of chronic heart failure. Violation of chewing and aesthetic function in patients with chronic heart failure can lead to the development of depression and decreased physical activity. Such conditions, in turn, further worsen the prognosis of the underlying disease. In patients with coronary artery disease, almost all clinical manifestations of dental pathology occur 1.5-2.0 times more often than in patients who do not have this pathology.

Some sources have shown that inflammatory periodontal diseases are an independent risk factor for coronary heart disease. There is some evidence that increased inflammatory activity in the periodontium as a key element contributes to the development of plaque atheroma, which causes cardiovascular disease, or that the mechanism could be cross-reactivity between antibodies against periodontal microorganisms and the formation of shock proteins in endothelial cells. These proteins are associated with atheroma plaque formation. On the other hand, it has been found that bacteremia of periodontal origin can have a direct effect. Numerous studies related to the study of the composition of biofilm in patients with periodontitis have shown that periodontal diseases are associated with a higher content of anaerobic gram-negative microorganisms, such as Prevotella, Leptotrichia, Veillonella, Porfiromonas, Treponema. These microorganisms destroy periodontal tissue directly through pathogenic products such as endotoxins, collagenases, causing macrophage stimulation, the release of inflammatory mediators, which play an important role in atherogenesis, affecting the accumulation of platelets, which can subsequently lead to thrombus formation. Both in heart failure and in generalized periodontal lesions, we can observe an imbalance of inflammation-cytokine indicators. Thus, in chronic heart failure, interleukin 1β (IL1β), IL-6, tumor necrosis factor (TNF-α), TGF-beta1 (TGF β1), chemokines (MCP-1, MIP β1), angiogenic growth factor are important vascular endothelium - (VEGF). According to some studies, there is an increase in the blood levels of IL-1β and TNF-α, which directly depends on the stage of CHF.

IL-1β,-6 and TNF-α increase apoptosis of cardiomyocytes and increase the activity of metalloproteinases, increasing the excretion of inhibitors, leading to remodulation of the heart muscle [Li Y.Y., Feng Y.O., Kadokami T., 2010]. The activity of the cytokine system is overwhelming Among patients with CHF, they lead to hypertrophy of cardiomyocytes and cardiac fibrosis, and 9 Journal of biomedicine and practice 1 (2016) also enhance the effect of angiotensin II (ATII) on fibrosis processes in the liver and heart [Hitora H.Chen J, Betz U.A., 2009]. The development of the pathological process in patients with periodontitis is accompanied by an imbalance of cytokines, clearly correlating with the severity of the pathology, with a significant increase in the level of pro-inflammatory cytokines - the greatest damaging effect in periodontal diseases is characteristic of interleukin-1β (IL-1β) and TNF-α and a less pronounced increase or even decrease the content of interleukin-4 (IL-4) and interleukin-10 (IL-10) as anti-inflammatory cytokines that restrain the destructive-inflammatory process in the periodontium and suppress osteoporosis. The highest level in the gingival fluid is determined by the content of TNF-α and exceeds that in practically healthy individuals by more than 7 times. Along with this, the content of the anti-inflammatory cytokine IL-4 in the gingival fluid in chronic periodontitis is lower than in practically healthy individuals. The content of IL-10 in gingival fluid also decreases.

Parakhonsky also observed a 10-fold increase relative to the control level of TNF-α and a 2.7-fold decrease in the level of IL-4. TNF-α is released from immunocompetent cells during the inflammatory process. The functions of TNF-α are diverse and range from participation in inflammatory processes to the regulation of apoptosis. TNF-α plays an important role in the initiation and coordination of intercellular interactions, contributing to the development of the immune system's response to the introduction of an infectious agent. Thus, a direct relationship has been established between the presence of Porphyromonas gingivalis in the oral cavity of individuals with periodontal pathology and the expression of TNF-a in its tissues. Its main source is

activated macrophages. The increase in TNF-α during inflammatory and destructive processes of periodontal tissue is protective in relation to the microflora that penetrates into its tissue. It is known that TNF-α has an inhibitory effect on the growth of staphylococci and has the ability to neutralize bacterial toxins in gram-negative infections, however, in addition to the protective function against the invasion of microorganisms, TNF-α also plays a destructive role in relation to tissue structures. TNF-α stimulates the production of proinflammatory cytokines, prostaglandins and leukotrienes, increases the expression of intercellular and vascular adhesion molecules-1 (ICAM-1 and VCAM-1), involved in the migration of lymphocytes to the pathological focus, proliferation of fibroblasts and synoviocytes, stimulates the formation of matrix metalloproteinases (enzymes that destroy connective tissue tissue) and inhibits the synthesis of their inhibitors, activates osteoclasts. TNF-α is considered as the main mediator that determines the development and progression of inflammation in periodontal tissues.

An increase in its content in oral, periodontal fluids or periodontal tissues during inflammation has been shown by many researchers. There is evidence that in periodontal pathology, TNF-α can be detected in the periodontal fluid even before clinically significant manifestations of the disease and thus serve as its indicator. TNF-α plays a key role in the pathogenesis of inflammatory-induced bone loss in periodontal diseases. Failure or insufficient power of counter-regulatory systems provokes excessive release of cytokines. IL-4 is able to block spontaneous and induced production of IL-1, IL-8 and TNF-α, as well as induce the expression of adhesion molecules on macrophage cells and promote their entry into apoptosis. When IL-4 is deficient, there is no local counteraction and the immune response is no longer counter-regulated. Interleukin-10 (IL-10) is produced by T cells. IL-10 suppresses the production of all proinflammatory cytokines and the proliferative response of T cells to antigens. Thus, from the above, some conclusions can be drawn. A decrease in the pumping function of the myocardium as a result of impaired myocardial contractility leads to impaired microcirculation, venous stagnation in periodontal tissues with the subsequent development of destructive processes, as a result of which patients often develop a pathological bite, the chewing optimum decreases, which in turn leads to a lower supply of nutrients to the body , reduces the quality of life, patients try to retire, do not go anywhere, which in turn leads to physical inactivity.

5. Conclusions

All this further worsens the course of heart failure and its prognosis. In addition, a common pathogenesis has been identified in the immunological status of patients with CHF and periodontal pathology. Both in heart failure and in patients with periodontal pathology, an increase in the levels of cytokines TNF-α and IL-1β is observed. Considering the mutual aggravating influence of foci of chronic infection in the oral cavity and cardiovascular diseases, as well as the dependence of the state of dental health on the severity and genesis of CHF, complex treatment of such patients should be carried out with the participation of a dentist, taking into account the standards of therapy for CHF and the algorithm for providing dental care. help. These findings indicate the need to optimize the diagnosis, prevention and treatment of dental diseases in patients with CHF, with the possible addition of anti-cytokine therapy in the future.

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