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THE ROLE OF VASCULAR ENDOTHELIUM IN THE DEVELOPMENT OF PERI-IMPLANTITIS IN PATIENTS WITH PERIODONTITIS WITH COMBINED PATHOLOGY OF THE CARDIOVASCULAR SYSTEM

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Abstract

The paper assesses the character of the functional state of the vascular wall in patients with chronic periodontitis combined with pathology of the cardiovascular system in the development of peri-implantitis. The study involved 74 patients with chronic generalized periodontitis, combined disease of the cardiovascular system and partial secondary adentia (absence of 1-2 teeth, duration of dentition defect existence up to 1 year), before dental implantation. A decrease in the anticoagulant and fibrinolytic activity of the vascular wall, which correlates with the severity of the clinical manifestations of pathology, was revealed. Complex treatment of patients with combined pathology provides a significant restoration of the anticoagulant and fibrinolytic activity of the vascular wall and a more pronounced positive dynamics of the clinical picture of the disease.

Keywords: periodontitis, cardiovascular pathology, endothelial dysfunction, homocysteine, endothelin.

Introduction

Currently, periodontal disease is a complex problem, which is primarily associated with the high prevalence and intensity of tissue damage around the tooth. The greatest prevalence among inflammatory periodontal diseases is generalized periodontitis, which is a dystrophic-inflammatory process resulting from the combined effects of various exo- and endogenous factors. According to WHO (2014), about 80% of the world's population suffers from periodontal diseases, which are the main cause of tooth loss. in people over the age of 30, the resulting periodontal pocket provides nutrients to the bacteria and improves the growth of anaerobic gram-negative bacteria involved in the destruction of periodontal tissue. Parodontopathogenic microflora in the oral cavity activates the complement system, stimulates the production of prostaglandins, leukotrienes, interleukins. The increased migration of macrophages and lymphocytes into the lesion focus causes

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immunological reactions with the participation of cytokines. The increased content of prostaglandins, cytokines interleukin-6 (IL-6), interleukin-ip (IL-ip), tumor necrosis factor-a (TNF-a) in the gingival fluid provokes processes of bone tissue destruction by activating osteoclastic resorption.

Dental manipulations are accompanied by bleeding, which leads to the possibility of penetration of microbial flora into the general bloodstream. Therefore, dental manipulations and surgical interventions are a risk factor for the development of transient bacteremia and, as a result, a systemic infectious disease. Based on this, the American Cardiovascular Association (AHA) recommends antibiotic prophylaxis before dental procedures involving significant bleeding (periodontal surgery, scaling, and professional hygiene) in patient populations at high risk of developing infective endocarditis. In addition, at present, in accordance with the European guidelines for the treatment of infective endocarditis, antibiotic prophylaxis is carried out during dental procedures associated with tooth extraction or procedures on the periapical zones of the teeth. However, bacteremia is observed even in the absence of dental interventions, during personal hygiene, especially in patients with significant inflammation of the periodontal tissues. The AHA states that "patients at risk of infective endocarditis (IE) should establish and maintain satisfactory oral health to reduce potential sources of bacterial dissemination."

There is also an opinion that oral microorganisms and their endotoxins can injure the endothelial vascular wall. There is increasing evidence that periodontal pathogenic microflora can directly contribute to the pathogenesis of atherosclerosis by increasing the amount of proinflammatory cytokines entering the bloodstream.

Silvestre FJ (2011), in their studies have shown thatStreptococcussanguis и Porphyromonagingivalisinduce platelet aggregation and activation through the expression of collagen-like proteins. These data are consistent with our research and results. Forner L соавторов (2006), which in carotid artery samples revealed the presence of P*Gingivalis*в100%examined samples.In another experimental animal study Lallaetal.(2003)demonstrated that infection P. gingivalisпривелото an increase in the number of adhesion molecules of vascular cells 1 (VCAM-1), serum IL-6 and tissue factor levels. It was shown on an experimental model that P. gingivalis in high concentration can induce apoptotic death of vascular endothelial cells. In the pathogenesis of generalized periodontitis (GP), microcirculation disorders, accompanied by an increase in vascular tissue permeability, which are caused by

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changes in the processes of activation and aggregation of platelets and thromboresistance, play a significant role. Vascular endothelium.

The above confirms the relevance of studying the relationship between periodontal diseases and the structural and functional state of the vascular wall, in order to optimize the prevention and treatment of inflammatory periodontal diseases and reduce cardiovascular risk. In our opinion, the changes occurring when chronic generalized periodontitis (CGP), are able to aggravate hemorheological changes characteristic of atherosclerosis. The presented data indicate the ability of periodontal pathogenic microflora to cause adhesion and aggregation of platelets, which may contribute to the development of endothelial dysfunction. To date, there is indisputable evidence that the endothelium is a neuroendocrine system that performs secretory, hemostatic, vasotonic functions, and also participates in the processes of inflammation and remodeling of the vascular wall. Based on the foregoing, the purpose of this study was to assess the nature of the functional state of the vascular wall in patients with chronic periodontitis combined with pathology of the cardiovascular system in the development of peri-implantitis.

Material and research methods

Clinical and laboratory studies were carried out in 74 patients, of which: I -group (14 patients) with chronic generalized periodontitis of moderate severity, group II (26 patients) with chronic generalized periodontitis with associated diseases of the cardiovascular system and III- group (34 patients) with chronic generalized periodontitis with associated diseases of the cardiovascular system with partial secondary adentia (the absence of teeth) with (the absence of 1-2 teeth, the duration of the dentition defect is up to 1 year), before dental implantation. In addition, the II-group of patients received a traditional treatment-and-prophylactic complex before dental implantation, the III-group of patients received the treatment-and-prophylactic complex developed by us aimed at the prevention and treatment of chronic inflammatory and destructive processes in the periodontium, correction of the hemostasis system parameters and the prevention of cardiovascular pathology ... This decision was made jointly with doctors - cardiologists. At the same time, to reduce the risk of cardiovascular catastrophes, a lipid-lowering drug was used - atorvastatin (KRKA, Slovenia), Clexane 0.4 ml SC, an anaerobicidal agent metronidazole in

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combination with chlorhexidine in the form of a gel. To prevent the processes of destruction and resorption of bone structures in the area of the implant, an additional course of glucosamine sulfate and chondroitin sodium sulfate was prescribed (Theraflex, Sagmel, Inc., USA). The drug was administered orally according to the following scheme: 2 capsules per day containing 1500 mg of glucosamine sulfate and 1200 mg of chondroitin sulfate, regardless of food intake, with a small amount of water. The recommended duration of the course of treatment was up to 3 months, but the patients were examined before dental implantation, at 14 months after traditional and complex therapy.

This approach to the complex treatment of patients with combined pathology was carried out 14 days before dental implantation in order to eliminate foci of chronic inflammation in the peri-implantation tissues, endothelial dysfunction and prevent the development of destructive processes in the alveolar bone in the peri-implantation area. To provide a drug effect on all components of the pain reaction (sensory, psychoemotional, vegetative and motor), we used combined anesthesia, which included local anesthesia and sedative sedation by oral administration of Relanium solution at the rate of 0.03 mg / 1 kg of the patient's body weight. All patients were referred for a consultation with a cardiologist to obtain an opinion on the possibility of performing the surgical stage of dental implantation. When examining patients at the preoperative stage, general clinical, radiological, functional and laboratory examination methods were used. Also, in this work, we used a computed tomograph located in the radiology department of the private medical company “DIOR”, “HiSpeedDX-IPlus” manufactured by General Electric with the software “DentoScan” and the following technical parameters. 14 days after the complex therapy, together with doctors - cardiologists and the conclusion of a doctor of radiation diagnostics, it was decided that it was possible to perform intraosseous dental implantation in patients with cardiovascular pathology. In all patients, before and after therapy, the level of individual hygiene and the condition of the periodontal tissues were assessed. Oral hygiene was determined by the Green-Vermillion (OHI-S) method (Simplified Oral Hygiene Index). The depth of the periodontal pocket (PC) and the loss of the periodontal attachment (PZP) were measured. Gum bleeding was assessed using the gum bleeding index according to H.R. Muhleman. Подвижность зубов определяли по шкале Miller (в модификации T.J.Fleszar).To identify the developed forms of periodontal pathology, the periodontal index was used (PI,

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Rüssel, 1956); the degree of gingival recession by classification P.D. Miller (1985). To determine the qualitative and quantitative composition of microorganisms in periodontal pockets, the method of polymerase chain reaction in "real time" was used. To identify possible atherosclerotic vascular lesions of the circulatory system and disorders of the blood coagulation system, at the stage of planning the operation of intraosseous dental implantation, we studied the hemostasis system. Blood sampling was carried out in the morning, on an empty stomach, by gravity into a plastic test tube. Blood for the study was taken from the cubital vein twice: in the amount of 10 ml before the cuff test (3-5 minutes clamping of the shoulder vessels using a cuff from a sphygmotonometer) and 5 ml after the cuff test. 5 ml of blood obtained before the cuff samples were subjected to centrifugation (3000 rpm) for 10 minutes in order to obtain serum for studying the content of homocysteine and endothelin -1. Serum samples were quickly frozen and stored at minus 20 ° C in well-closed tubes. 5 ml of blood obtained before the cuff test and 5 ml of blood obtained after the cuff test were stabilized with a 3.8% sodium citrate solution in a ratio of 9: 1. The studies were carried out on platelet-poor plasma, which was obtained by double centrifugation: first at 1000 rpm (7 minutes), then at 3000 rpm (15 minutes). Centrifugation was performed immediately after blood sampling, and plasma sampling for research was performed immediately after centrifugation. Plasma samples were analyzed no later than 3 hours after blood sampling.To determine the anticoagulant activity of the vascular wall endothelium, the level of antithrombin III (AT III) activity in the blood was determined before and after the cuff test. The ratio of AT III activity before and after the cuff test characterizes its release by endothelial cells. Normally, after the cuff test, anticoagulants are released into the blood (the activity of AT III increases). To determine the activity of AT III, we used a set of "Human" company. To determine the fibrinolytic activity of the endothelium of the vascular wall, the rate of Hageman-dependent fibrinolysis of blood plasma was determined before and after the cuff test (3-5 minutes of clamping of the shoulder vessels using a cuff). The ratio of the rate of Hageman-dependent fibrinolysis after and before the cuff test characterizes the release of tissue plasminogen activator (t-PA) and plasminogen activator inhibitor (PAI-1) by endothelial cells. Normally, after the cuff test, t-PA is released into the blood and the production of PAI-1 decreases, which leads to an increase in the rate of Hageman-dependent fibrinolysis. Hageman-dependent fibrinolysis was determined using a kit from Renam (Russia). Determination of the level of endothelin I and

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homocysteine in blood serum was carried out by the enzyme-linked immunosorbent assay using a kit from Human. Laboratory studies were performed on an enzyme-linked immunosorbent analyzer (Human). The results were carried out using the methods of parametric and nonparametric statistics. Descriptive statistics methods consisted in estimating the arithmetic mean (M), the mean error of the mean (w) - for features with a continuous distribution, as well as the frequency of occurrence of features with a discrete value. To assess the intergroup differences in the mean values of characteristics with a continuous distribution, the Student's t test was used.

Research results and their discussion

The anticoagulant and fibrinolytic activity of the vascular wall is based on the creation of short-term (3 min) local ischemia caused by the imposition of a sphygmomanometer cuff on the subject's shoulder and creating a pressure in it that exceeds the systolic indicator by 10 mm Hg, which leads to the release of prostacyclin from the vascular endothelium of healthy people into the blood , nitric oxide, antithrombin 111, tissue plasminogen activator [Baluda VP, Deyanov I, Baluda MV, Kirichuk VF et al, 1992].

We found that in patients with chronic generalized periodontitis in combination with cardiovascular pathology, thromboresistance of the vascular endothelium was reduced, which is expressed by a decrease in the anticoagulant activity index of the vascular wall to 1.17 ± 0.09 uel. units in group II patients, and up to 1,14± 0,07 units in the III-group of patients with combined pathology and a drop in the index of fibrinolytic activity of the vessel wall to 0.57 ± 0.05 uel. units in the II group of patients and up to 0.55 ± 0.06 conventional units. in III - group of patients. Consequently, in patients with chronic generalized periodontitis in combination with cardiovascular pathology, thromboresistance of the vascular endothelium was reduced. This is evidenced by a statistically significant decrease in the decrease in the index of anticoagulant activity and the index of fibrinolytic activity of the vascular endothelium. The decrease in the anticoagulant activity of the vascular endothelium in the examined individuals is manifested by inhibition of the release of antithrombin III by the endothelium of the vascular wall. It is known that thrombomodulin binding thrombin causes changes in the conformation of its active center, as a result of which the rate of its inactivation by antithrombin III increases. On the other hand, it was found that a number of inflammatory cytokines, in particular interleukin 1, as well as

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tumor necrosis factor, cause a decrease in the anticoagulant activity of the vascular endothelium.

Table

Thromboresistance of the vascular endothelium in patients with Chronic generalized periodontitis with combined cardiovascular pathology in preparation for dental implantation (before therapy)

Indicators	I-group n=14	II-group n=26	III-group n=34
Antithrombin-III to cuff test in%	88,24±6,32	74,32±5,48	70,25± 4,57*
Antithrombin-III after cuff test in%	109,17± 8,04	87,13 ± 5,21	80,13± 4,68*
Anticoagulant index endothelial activity usl.	1,23± 0,06	1,17±0,04	1,14± 0,03*
XIIa-dependent fibrinolysis before cuff test sec.	614,61 ± 13,7	690,78±12,53	752,98±12,51*
XIIa-dependent fibrinolysis after cuff test sec	389,41± 11,9	403,11±12,56	421,07±13,28*
Index of fibrinolytic activity of endothelium conventional units	0,63± 0,04	0,57± 0,03*	0,55± 0,03*
Homocysteine concentration μMol / L	8,81± 0,74	15,67± 1,12*	17,23±1,57*
1 Concentration of endothelin -1 blood plasma μMol / L	1,64± 0,13	4,67± 0,32*	5,86± 0,41*

Note: * - reliability of differences relative to the comparison group P <0.05

In this regard, it is most likely that in patients with CGP combined cardiovascular pathology, a decrease in the anticoagulant activity of the endothelium of the vascular wall is mediated by the action of immune mechanisms implemented in a long-existing focus of inflammation, i.e., at the site of the extracted tooth and periodontal pocket. The observed inhibition of the fibrinolytic activity of the vascular

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endothelium may be associated with a decrease in the release of tissue plasminogen activator t-PA.

Homocysteine is a cytotoxic amino acid and its high content leads to hyperhomocysteinemia and, consequently, toxic effects on endothelial cells. In our studies, an increase in the level of homocysteine in patients of group II by 1.8 times was noted, while in group III of patients it exceeded the initial level by 1.96 times relative to the indicators of the comparison group. As a result of the damaging effect of homocysteine, the production of a number of regulatory substances produced by the endothelium, in particular, a decrease in the synthesis of nitric oxide and prostacyclin and an increase in the formation of thromboxanes. It is known that homocysteine reduces the anticoagulant activity of the endothelium of the vascular wall, due to the degradation of thrombomodulin, a decrease in the expression of antithrombin III-heparin complexes on the surface of endothelial cells and significantly reduces the activity of the protein C. In addition, homocysteine causes a decrease in plasminogen activation by stimulating a thrombin-activated inhibitor fibrinolysis - TAFI (thrombinactivatablefibrinolysisinhibitor). Importantly, homocysteine also increases the expression of the plasminogen activator inhibitor-1 (PAI-1) gene, which inhibits fibrinolysis.

As you know, despite the short life span, endothelin-I is a powerful vasoconstrictor, causes significant changes in hemodynamics: a decrease in heart rate and stroke volume of the heart, an increase in vascular resistance, and promotes remodeling of the vascular bed. It is the differences in the physiological and pathological roles of endothelin I that determine its diagnostic significance as a marker of damage and dysfunction of endothelial cells. The main stimulators of endothelin I production by the endothelium of the vascular wall are reactive oxygen species, inflammatory cytokines such as IL-1, IL-6, and TNF. In our studies, a potential mechanism of the detected increase in the concentration of endothelin I in CGP with combined cardiovascular pathology may be the induction of oxidative stress in the periodontal tissues. Probably, an increase in the concentration of endothelin I in the blood serum in patients with the combined form of the disease should be considered as a reaction to systemic manifestations of the inflammatory process.

From the presented data, it can be concluded that periodontitis leads to higher systemic disorders of endothelial cells. The revealed changes in the studied parameters can enhance the inflammatory activity not only in the periodontal tissues,

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but also potentially increase the risk of cardiovascular diseases. It is possible that the revealed changes occurring in the periodontal tissues, in particular, the periodontal pathogenic microflora and the process of atherogenesis, may contribute to the development and progression of blood vessel thrombosis. Therefore, for the timely prevention of cardiovascular diseases and destructive changes in the periodontal tissues, it is necessary to use pathogenetically substantiated correction of periodontal tissue diseases and other immuno-inflammatory diseases of a systemic nature.

In the future, the effectiveness of traditional and complex treatment was assessed for indicators of thromboresistance of the vascular endothelium in patients with CGP with combined cardiovascular pathology in preparation for dental implantation. As it turned out, in the group of patients with CGP concomitant cardiovascular pathology, after the conventional treatment for 10-14 days, an improvement in the hygienic state of the oral cavity, a decrease in gingival bleeding, the intensity of pain the activity of the vascular wall after the traditional treatment remained less than the control values. The data obtained indicate the insufficient effectiveness of traditional methods of treating CGP with combined pathology of the cardiovascular system, which dictates the need to search and test new pathogenetically substantiated methods for correcting the revealed disorders of various links of the hemostasis system, which significantly improve the clinical picture of the disease.

In connection with the above, a series of clinical and laboratory studies were carried out in patients with CGP with combined cardiovascular pathology to test the use of systemic drugs in complex therapy; atorvastatin, clexane, anaerobicidal agent and chondroprotector. This approach to complex treatment of patients with combined pathology was carried out 14 days before dental implantation, in order to eliminate foci of chronic inflammation in the peri-implantation tissues, endothelial dysfunction and prevent the development of destructive processes in the alveolar bone in the peri-implantation area. The use of clexane in complex treatment improved the antithrombogenic properties of the vascular wall. This was evidenced by an increase in the indices of anticoagulant and fibrinolytic activity of the vascular wall in comparison with the values in the group of patients who did not receive complex therapy.

The lack of proper effectiveness of traditional methods of therapy for this combined pathology, as well as the changes in the indicators of the hemostasis system that we established, determined the need to use clexane, an anticoagulant of

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systemic action, in the complex treatment of CGP with combined pathology of the cardiovascular system. The drug was administered according to the above scheme. Evaluation of the effectiveness of this treatment was carried out in terms of the index of anticoagulant activity and fibrinolytic activity.

Table

Thromboresistance of the vascular endothelium in patients with CGP with combined cardiovascular pathology in preparation for dental implantation (after therapy)

Indicators	I- группа n=14	II-группа n=26	III-группа n=34
Antithrombin -III to cuff test in%	88,24± 6,32	76,52 ±5,83	86,67±6,13
Antithrombin-III after cuff test in%	109,17±8,04	90,13± 8,02	105,79± 7,63
Antithrombin-III after cuff test in%.	1,24 ± 0,11	1,18 ± 0,08	1,22± 0,07
XIIa-dependent fibrinolysis before cuff test sec.	614,61 ± 13,7	662,18± 14,14	621,38± 13,76
XIIa-dependent fibrinolysis after cuff test sec	389,41 ± 11,9	401,43± 10,24	391,25 ± 11,72
Fibrinolytic index endothelial activity usl.	0,63±0,04	0,61 ±0,03	0,63±0,03
Homocysteine concentration μMol / L	8,81 ±0,74	13,57 ±0,69	9,42 ±0,64
Plasma endothelin -1 concentration μMol / L	1,64±0,13	3,89± 0,27	2,01± 0,17

Note: * - reliability of differences relative to the comparison group P <0.05

Also, after the course of therapy with the inclusion of complex therapy, an improvement in the clinical picture of the disease and a decrease in the indicators of the main indices characterizing the state of the periodontal tissues were found: the periodontal index, the papillary-marginal-alveolar index and the index of oral hygiene in comparison with group II of patients who did not receive this therapy.

Thus, summarizing the results obtained in general, it should be concluded that one of the leading pathogenetic factors of disorders of local ("in the oral cavity") and systemic microhemodynamics in the combined pathology of periodontal diseases are disorders of the anticoagulant and fibrinolytic activity of the vascular wall, correlating with endothelial dysfunction in the background high values of

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homocysteine and endothelin-I, as well as the degree and severity of the clinical manifestations of pathology.

The data obtained in this work indicate the insufficient effectiveness of traditional methods of treating CGP with combined pathology of the cardiovascular system, inflammatory - destructive processes in the periodontal tissues, which do not allow preventing violations of the thromboresistance of the vascular endothelium and microcirculatory disorders.

Deepening the existing understanding of the pathogenesis of periodontal diseases and the cardiovascular system at the molecular-cellular, organ and systemic levels made it possible to formulate new principles of pathogenetic therapy of CGP with a combined disease of the cardiovascular system using anti-inflammatory, anticoagulant therapy, as well as destructive changes in the bone tissue of the periodontium, which opens up new possibilities diagnostics and prevention of complications after dental implantation.

CONCLUSIONS

1. One of the leading mechanisms of the development of microcirculatory disorders in patients with chronic generalized periodontitis and a combined disease of the cardiovascular system is a decrease in the anticoagulant and fibrinolytic activity of the vascular wall, which correlates with the severity of the clinical manifestations of the pathology.
2. Complex treatment of patients with combined pathology provides a significant restoration of the anticoagulant and fibrinolytic activity of the vascular wall and a more pronounced positive dynamics of the clinical picture of the disease.

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