**FUNCTIONAL STATE OF VESSEL ENDOTHELIAL CELLS IN HYPERTROPHIC GINGIVITIS IN ADOLESCENTS**

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**Introduction.** Endothelium - the inner lining of blood vessels - takes an active part in the regulation of vascular tone, producing various biologically active substances (BAS). Biologically active substances that act on the endothelium are produced by platelets, leukocytes, mast cells or are activated in the blood plasma, some of the substances are synthesized in the endothelium itself and act on the endotheliocytes either after they are brought into the bloodstream or paracrine (5,11,12). The effect on the endotheliocytes of biologically active substances is associated with the presence of specific receptors on endotheliocytes, the stimulation of which causes vasodilation or vasocostriction.

With endothelial dysfunction (ED), the functional state of the inner lining of the vessels is disrupted, which leads to the production of an overestimated amount of nitric oxide (NO). Large amounts of NO in the blood can form peroxynitrite, which activates the process of free radical oxidation of proteins and lipids. Therefore, one of the causes of impaired regional blood circulation and microcirculation is endothelial dysfunction, which can lead to vasospasm, increased thrombosis and increased white blood cell adhesion to endothelium. Nitric oxide takes part in the regulation of almost all the functions of the endothelium (regulation of vascular tone, vascular thrombotic resistance), and in addition, it is the factor most sensitive to damage (9.16).

Endothelial dysfunction has another, no less important aspect - hemostatic disorders. While the endothelium is intact, not damaged, it synthesizes mainly anticoagulation factors, which are also vasodilators. In addition, endothelium adsorbs numerous anticoagulants from blood plasma. The combination of anticoagulants and vasodilators on endothelium under physiological conditions is the basis for adequate blood flow, especially in microcirculation vessels.

With prolonged damage to the endothelium, according to many researchers, it begins to play a key role in the pathogenesis of a number of systemic pathologies, in particular in pathology of the dentition. This is explained by the switching of endothelial activity to the synthesis of oxidants, vasoconstrictors, aggregates, and thrombogenic factors (8,12,13). And platelets are the main cells that ensure the normal course of hemostasis, the main function of platelets is their participation in blood coagulation processes. Thus, under physiological conditions, the holistic endothelium is the main anticoagulant factor.

Based on the foregoing, we studied the anticoagulant and fibrinolytic properties of the vascular wall endothelium, as well as the determination of endothelial dysfunction markers - endothelin-1 in adolescents with hypertrophic gingivitis (HG).

**Material and research methods.**

Due to the fact that the true period of puberty, according to age morphology, physiology and biochemistry, is 13–16 years for boys and 12–15 years for girls, during the study we selected groups of adolescents from 13 to 15 years old. A clinical study was conducted in 65 children, which reflected almost the entire population of the studied age. Next, a comparative clinical and functional study was carried out, with the identification of groups with HG and an intact periodontal period, the definition of objective examination criteria, the status of hypertrophy.

The antithrombogenic activity of the vascular wall endothelium was determined by a cuff test [1,3]. Antithrombin III activity was determined using a Solar-CGL2120 coagulometer (Belarus), using reagents from “Tekhnologiya Standart” (Barnaul, Russia). Based on the obtained data, the indices of antiplatelet, anticoagulant and fibrinolytic activity of the vascular wall endothelium were calculated [1,3].

The content of endothelin-1 (endothelin-1, Et-1) was carried out on an immunoassay analyzer "MindrayMR-96 A".

Statistical processing of the results was carried out using computer programs Statistica 6.0. Statistical analysis of quantitative traits was performed using Student's criterion. P values <0.05 were considered highly significant and reliable.

**The results obtained and their discussion**

The data obtained indicate that in the examined patients with HG, the anticoagulant activity of the vascular wall endothelium is inhibited. Moreover, in patients with HG, there is a statistically significant decrease in the activity of antithrombin III in the blood before and after the occlusion test, as well as a decrease in the index of anticoagulant activity of the endothelium of the vascular wall, compared with clinically healthy donor volunteers. Thus, the activity of antithrombin III in the blood before and after a cuff test is statistically significantly reduced (Figure 1).

**Fig. 1. Indicators of anticoagulant activity of vascular endothelium (AT-III) in adolescents with hypertrophic gingivitis**

This indicates that in patients with HG the release of antithrombin III by vascular endothelium is impaired. Therefore, in the examined patients, the anticoagulant activity of the endothelium of the vascular wall is impaired, which is manifested by a decrease in the endothelial secretion of antithrombin III.

When assessing the fibrinolytic activity of the vascular wall endothelium, it was found that in patients, there is a statistically significant reduction in time, compared with clinically healthy donor volunteers, of Hageman-dependent fibrinolysis before and, especially, after a cuff test, which reflects a decrease in the release of tissue plasminogen activator and / or increased production of plasminogen activator inhibitor.

Differences in the index of fibrinolytic activity of the vascular wall endothelium in the examined individuals are not expressed equally and the time of Hageman-dependent fibrinolysis before and after the cuff test is statistically significantly greater in individuals with pathology, which indicates a more pronounced inhibition of fibrinolytic activity and a more significant imbalance in the release of tissue plasminogen activator is associated and its inhibitor endotheliocytes (Fig. 2).

**Fig. 2. Indicators of fibrinolytic activity of vascular endothelium (XIII-dependent fibrinolysis before and after a cuff test) in the examined patients**

Therefore, in the examined adolescents, the fibrinolytic activity of the vascular endothelium is higher than that of healthy individuals.

Thus, as a result of the studies, it was found that in patients with HG changes in the fibrinolytic activity of the vascular wall endothelium occur, which are manifested by a decrease in the induced release of tissue plasminogen activator and / or an increase in the release of its inhibitor. The data obtained allow us to conclude that in patients with HG there are significant violations of the thromboresistance of the vascular wall, which are manifested by a change in the anticoagulant and fibrinolytic properties of the endothelium. In this case, disorders of both anticoagulant and fibrinolytic activity of vascular endothelium prevail compared to healthy individuals (Fig. 2).

**Fig. 3. Indices of endothelin-1 (mmol / ml) in children with HG**

To assess endothelial dysfunction, a study was made of the concentration in the blood serum of endothelin I in the examined patients. As a result of the studies, it was found that with this pathology, a statistically significant change occurs compared with clinically healthy volunteer donors (Fig. 3). Moreover, in the examined patients, the concentration of endothelin I in the blood serum is statistically significantly higher in comparison with healthy individuals. Thus, it was found that in adolescents with HG, in contrast to healthy individuals, there is an increase in the content of endothelin-I in the blood serum. The data obtained allow us to conclude that in patients there are significant violations of the thromboresistance of the vascular wall, which are manifested by a change in the anticoagulant and fibrinolytic properties of the endothelium, which leads to increased synthesis of endothelin-1.

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