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CRITERIA FOR THE ASSESSMENT OF THE STATE OF IMMUNOLOGICAL INDICATORS IN CONNECTION WITH IMMUNIZATION OF VASCULAR TISSUE HOMOANTIGEN

Abstract: *On the experimental model of autoimmune vascular lesions, immunological shifts have been studied by posing a number of simple, convenient, effective methods for analyzing immune shifts arising from the introduction of a homologous vascular antigen to experimental animals. Numerous clinical and experimental studies have shown the importance of autoimmune changes in the pathogenesis of a number of diseases of internal organs. However, the issue of the role of immunological reactivity in the development of vascular pathology (athero-arteriosclerosis) has not been sufficiently studied, despite the fact that there are a considerable number of works on this issue indicating the presence of an autoimmune reaction to vascular tissues in arteriosclerosis.*

The study of immunological changes in the development of vascular lesions caused by their damage by homologous tissue antigens is relevant. At the same time, however, it is necessary to take into account the peculiarities of organ-specific antigens, their isolation in the body and the absence of their access and circulation,

which also makes it possible to autosensitize. This circumstance undoubtedly has an important practical significance in modeling organ pathology, since it allows one to deepen the understanding of the pathophysiological mechanisms in each specific case.
Key words: homo antigen, autoimmune, atherosclerosis, allergic alteration.

MATERIAL AND METHODS

Autoimmune changes were performed on experimental models of athero-arteriosclerosis, obtained by exposing the animal (dogs) and vascular wall damage to homologous tissue antigens. For studying autoimmune shifts generated on the contact models eksprimentalnyh athero-arteriosklerozamy complex used immunological tests:. Passive haemagglutination toned on erythrocytes and leucocytes specific alteration direct degranulation of basophils neutrophils allergic alterations aimed at the detection of antibodies and detection of the degree of sensitization [1, p.48].

Studies of cellular reactions were carried out taking into account that they are not inferior in terms of the sensitivity of the Prausnitz-Kustner reaction, and in terms of simplicity, they can serve as a criterion for detecting autosensitization of the organism.

Studies conducted on 10 dogs. The introduction of tissue homoantigen was made five times with an interval of 2 days at the rate of 0.5 ml per 1 kg of animal weight. Experiments were made after the last administration of homoantigen, and later on the 15th, 30th, 45th, 75th, 90th day in the dynamics of the development of functional changes. [2, p.30].

All the results of the research are developed by the methods of mathematical (variation) statistics. For the analysis and evaluation of the results of PCA, logarithm was performed with their subsequent statistical processing [1, p. 53].

RESULTS OF RESEARCH.

Immunization of animals (dogs) with homoantigens of the vascular wall promoted vigorous production of specific (circulating) antibodies (D 0.001), which began with a period of homoantigen stimulation and continued during the 15-45th day of research after the last injection of antigen.

Later on, on the 75th day of the study, there was a slight decrease in the production of circulating antibodies, which increased again from the 90th day (P 0.001) [3, p.16].

Changes in cell reactions coincide with the level of antibody titer. Thus, the indicators of leukocytolysis and neutrophil damage (PPN) sharply increase after homoantigen stimulation (P 0.001). Starting from the 15th day after the last administration of homoantigen, the leukocytolysis index and neutrophil damage (PPN) tends to decrease slightly. This decline in leukocytolysis continues for 45 days, and neutrophil damage indicators for the 75th day of the study (P <0.001).

From the 60th day of the study, the indicator of leukocytolysis begins to rise again and remains at this level until the 90th day of the study of neutrophils (PPN) / occurs from the 90th day of the study (P 0.001).

DISCUSSION OF THE OBTAINED DATA

It can be assumed that enhanced production of antibodies (circulating) in the early stages of the development of experimental atherosclerosis may be due to the nonspecific sensitizing action of a foreign protein that disrupts immunological reactivity, since fixed normal autoantibodies specific for a given organ are present in each cell and the tissue is created when the tissue is destroyed for admission to the bloodstream of these antibodies. Another option is possible that immunization of animals with homologous antigens causes early changes in the vascular wall and contributes to more vigorous production of specific autoantibodies.

It should be noted that with the development of experimental atherosclerosis, the resulting autoimmune complex is fixed in the vascular wall and with the participation of complement has a damaging effect on the tissue, with the release of structural antigens with auto-antigenicity (secondary). In accordance with this, the immunological processes associated with the reaction to the antigens of the vascular wall develop. At the same time, the constituent components are - lipoproteins as an antigen and - as an autoantibody [4, p.42].

Based on the idea of the “secondary” nature of antibodies induced by structurally altered autologous proteins and polysaccharides entering the blood, it can be assumed

that more vigorous production of specific autoantibodies is associated with an inadequately enhanced response of the immune system.

Based on modern ideas that the main role in the development of the pathological process is played not by the blood, but fixed in the walls of the autoantibody, it can be assumed that the decrease in the production of antibodies (circulating) in the late stages (75th day) of experimental atherosclerosis is due to their fixation cells of various organs. In addition, a decrease in the blood content of the anti-vascular antibodies may be associated with the total depressant effect of the auto-and homo-vascular antigen on immunocompetent tissues. The possibility of reducing the titer of circulating autoantibodies by binding them with the introduced antigen is also not excluded. Currently, there is enough data to suggest that in the mechanism of allergic alteration of leukocytes (leukocytolysis) and granulocyte damage (PPN) with delayed allergy, humoral factors play a significant role (react)

The interaction of the allergen with the antibody on the cell surface or very close to it actively involves leukocytes due to their functional characteristics in the process of inactivating the immune complex through absorption and intracellular digestion. During this exposure, leukocytes undergo both morphological and enzymatic alteration, and finally, they themselves become a victim of the lytic action of the released enzymes. Thus, the alteration of leukocytes under the action of the immune complex is completed by leukocytolysis.

Giving a general assessment of these reactions, one should note the specificity with respect to the corresponding organ antigen in the absence of nosological specificity. With its help, you can identify and assess the degree of autosensitization of the body. In the process of sensitization with vascular homoantigen, along with the production of antibodies, the formation of circulating and adsorbed immune complexes (antigen-antibody) occurs with the participation of complement. As a result of the adsorption of this immune complex on cells, a number of biologically active amines are released, which have a damaging effect on the tissue. One of these moments, reflecting the appearance of GNT, accompanied by the formation of biologically active substances such as histamine, is the reaction of basophil degranulation. [5, p.40].

Studies have shown that the index of degranulation increases Homo-antigen stimulation (P 0.001). This increase continues on the 30th day after the last administration of anitigen (P 0.001). On the 45th day, there are signs of a slight decrease in the degranulation index, which, starting from the 75th day of the study, begins to rise again. This increase continues until the end of the study (P 0.001) (Table 1). The mechanism of the allergic reaction of mast cells is not their damage, but an increase in their function (AM Iminova, 1971). The antigen-antibody reaction that takes place on the membranes of mast cells leads to their degranulation, releasing the biologically active substances they contain.

Thus, the conducted studies show that immunization with an antigen of homologous vascular tissue, causing alteration of vascular tissue or a profound disruption of the protein metabolism of this organ, may also cause a change in the antigenic structure of the wall proteins. At the same time, specific autoantigens that have arisen stimulate the production of anti-vascular autoantibodies. In the future, the formation of immune complexes, which are fixed on the surface of blood vessels and cellular elements of the blood, in turn, causes the degranulation of basophils and the alteration of neutrophils with subsequent damage to the vascular wall [6, p. 68].

FINDINGS:

Experimental studies show:

1. Experimental athero-arteriosclerosis, obtained by introducing the homoantigen of the vascular wall, is based on genesis conducive to its further development.

2. Antibodies to vascular tissues appear, which arise during the development of the experimental athero-arteriosclerotic process.

3. Introduction of the homoantigen of the vascular wall causes a pronounced strong sensitization of the organism.

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