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THE DEVELOPMENT OF SYSTEMIC INFLAMMATORY REACTION SYNDROME IN ACETIC ACID POISONING

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Relevance of the study. The basis of a wide variety of pathological processes, including those that are not associated with CVD or poisoning, are problems associated with endothelial dysfunction, and the functional restructuring of endotheliocytes is the starting point for the development of these very pathologies. After these physiological changes in the endotheliocyte series, microcirculation is disrupted at the capillary level, and, as laboratory and clinical studies have shown, excessive NO generation causes oxygen starvation at the cellular and tissue levels. This leads to various processes of intoxication and cell death. And although there are many works in the modern medical literature whose research is aimed at directly studying the endothelium and endotheliosis syndrome in conditions of various pathologies in the body, there are no works among them that would describe the state of the immune system, endothelium and the dynamics of pathological processes in conditions of poisoning with HC and its derivatives. According to foreign researchers, systemic inflammatory reaction syndrome is defined and characterized according to the actual state of the vascular endothelium, on which the regulation of inflammatory processes, biosynthesis and other physiological processes depend. An increase in the average level of endothelin and its content in plasma is also characteristic of cases associated with poisoning with HC and its derivatives; there is an increase in the Wilebrandt criterion, and the rate of platelet grouping and the total number of platelets drops sharply due to the fact that the body is experiencing an accumulation of hemoglobin. These processes are characteristic already by the first or second day after poisoning with HC and/or its derivatives. Oozing with oxidized lipids, cytokines initiate the production of heat shock proteins, which is generated by proteolysis; in addition, NO is produced, which cytokines also participate in the generation. Although an increased rate of cytokine production is a precedent for increased NO formation, its excess can lead to disruption and dysfunction of the microcirculation process, vasolidation, and decreased absorption of vascular walls. An analysis of scientific papers has shown that there are two main precedents in the formation of systemic inflammatory response syndrome, which include a gradual increase in LPS migration and excitation of the vascular endothelial layer, while the inflammation process is characterized by the formation of close groups of cells that emancipate inflammatory mediators, which include proteases, LC, NO, etc. Under the conditions of the protective mechanisms of cell groups, the NFKB factor is activated – its activation is caused by an increase in the activity of antiapoptotic genes and the death of cell groups. As has already been established, acute inflammation is the cause of the production of thrombin and APC hemostases, cell groups are disbanded, and inflammatory cytokines (LC) are emancipated. The critical point of thrombin activity coincides with the same point of destruction of cell groups, after which APC is generated, which ensures the stabilization of cell membranes, and changes in the integrity of cells and cell groups of the endothelial layer occur as a result of chemical reactions, including those caused by drugs that can cause signals of inflammatory This process is due to the multifaceted mechanisms of enhancing cell adhesion and stimulating neutrophils, generating oxygen, and thereby provoking the inflammatory process. Cellular and tissue groups are also able to influence the course of pathological processes.

The activity of red blood cells on a microscale depends on their deformability, their ability to aggregate, their function as connecting links between gases, and the ability to transport metabolic products, including with a particular functional and physiological state of the erythrocyte layer. In

particular, damage to erythrocyte membranes by toxins can cause the intensity of the cell grouping process, as well as a decrease in the ability of red blood cells to deform. These processes lead to disturbances in the normal circulation process. It follows that the main indicative symptom of the presence of inflammatory processes in the body is the actual restructuring of white blood cells. Neutrophils, which are one of the most relevant components of the immune system, act as the main barrier preventing the growth of the number of bacteria and other infectious agents, including viruses, fungi, while neutrophils in the shortest period of time shift to the site of infection in the body and begin to destroy pathogenic cells. Of course, such a reaction of the immune system has a positive haarcter for it, although in the case of excessive neutrophil generation and activity, pathological processes may occur, such as a mismatch of secreted protease and traumatic tissue damage during necrotic processes. Neutrophils involved in the process of immune defense, as well as other cellular agents, which, in particular, include derivatives of active leukocytes, LC may also have a destructive effect on the endothelium. It has now been established that hyperactive forms of NO compounds that are emancipated by macrophages can initiate a wide variety of pathological processes caused by xenobiotics and their effects on tissues. It also makes sense to assume that in acute poisoning, including poisoning with HC and its derivatives, decompensated acid metabolism may occur, a sharp change in the pH level at the micro and macro levels, various disturbances occur in the vital processes of cells and tissues, and the characteristic granularity of the tissue, indicating its intoxication, indicates There are two processes at the cellular level: the absorption of toxins by cells and tissues and the maturation of neutrophils. Among patients with fatal disease, exactly the processes described above were observed, already on the first day after poisoning with HC and its derivatives. Neutrophil generation in deceased patients was much more intense than in survivors of pathology. It is also important to note the fact that poisoning causes changes in the reaction of neutrophilic granutocytes (NG) under poisoning conditions, including poisoning with HC and/or its derivatives. Acute poisoning, accompanied by hypoxia and attacks of suffocation, is often accompanied by a violation of the body's anti-infective defenses, in particular, at the initial stages of intoxication (0-6 hours), a characteristic increase in the critical value of T-B lymphocyte parameters in the blood can be noted, which may become a precedent for the development of pneumonia in patients with intoxication.

Conclusion. The characteristic weakening of the immune system of this kind is due to both the action of xenobiotics and hypoxia, which directly develops under conditions of intoxication, free radical oxidation processes, which leads to endotoxicosis and increased attacks of hypoxia. Polymorphism processes in the immune system inhibit anti-infective protection at the cellular level; with impaired immunity at the humoral level, an increase in blood viscosity is observed, as well as a high concentration of bioamines, fibrogens and nitrogen oxides in it. All this is also observed in cases of poisoning with CC and its derivatives.

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