



Endothelial Dysfunction in the Development of Systemic Inflammatory Reaction Syndrome in Acetic Acid Poisoning

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Abstract: Acute food poisoning of the criminal code and its derivatives incapacitates a number of organs. At the same time, the CC and its derivatives, which are the cause of this kind of dysfunction, directly affect the pathogenesis. Hypoxia attacks with poisoning of the CC and its derivatives can cause inflammatory processes throughout the body, and even in those parts of it that are not directly affected by the CC and / or its derivatives. The body's resistance to inflammatory begins to flow intensively, anti-inflammatory functions of the body, cytokines and kinins are activated; vascular resistance decreases, and blood viscosity increases, thereby increasing the likelihood of thrombus and microthrombosis. (107,108). Inflammatory processes due to poisoning with CC and / or its derivatives are associated with the launch of proteolysis in the body, which proceeds in parallel with the clotting process

At the heart of a variety of pathological processes, including those that are not associated with CVD or poisoning, there are problems associated with endothelial dysfunction, and functional restructuring of endotheliocytes is the starting point for the development of these pathologies. After these physiological changes in the endotheliocyte series, there is a violation in microcirculation at the capillary level, and, as laboratory and clinical studies have shown, excessive generation of NO causes oxygen starvation at the cellular and tissue levels. This leads to different processes of intoxication and cell death [2.4.6.8.10].

And although there are many works in the modern medical literature, the research of which is aimed at the direct study of endothelium and endotheliosis syndrome in conditions of various pathologies in the body, there are none among them works that would describe the state of the immune system, endothelium and the dynamics of pathological processes in the conditions of poisoning with CC and its derivatives. According to foreign researchers, the systemic inflammatory reaction syndrome is determined and characterized according to the actual state of the vascular endothelium, on which the regulation of inflammatory processes, biosynthesis and other physiological processes depend [1.3.5.7.9].

An increase in the average level of endothelin and its content in plasma is also characteristic of cases associated with poisoning of the criminal code and its derivatives; there is an increase in the Wilebrandt criterion, and the rate of platelet grouping and the total number of these drops sharply due to the fact that the process of accumulation of hemoglobin is observed in the body. These processes are characteristic already by the first or second days after poisoning with CC and/or its derivatives.

Oozing with oxidized lipids, cytokines initiate the process of heat shock protein production, which is generated by proteolysis in addition, NO is produced, in the generation of which cytokines also participate. And although the increased rate of cytokine production is a precedent for increased NO formation, its excess can lead to disruption and dysfunction of the microcirculation process, vasodilation, as well as a decrease in the absorption of vascular walls.

The analysis of scientific papers has shown that there are two main precedents in the formation of the systemic inflammatory reaction syndrome, which include a gradual increase in the migration of LPS and the 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 due to an increase in the activity of anti-apoptotic 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 [12.14.16.18.20].

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 the change in the unified integrity of cells and cell groups of the endothelial layer occurs as a result of chemical reactions, including those caused by drugs that can cause signals of inflammatory the process is due to the multifaceted mechanisms of enhancing cell adhesion and excitation of 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 erythrocytes on a microscale depends on their deformability, their ability to aggregate, acting as connecting links between gases, the possibility of transporting metabolic products, including with a particular functional and physiological state of the erythrocyte layer. In particular, the damage of erythrocyte membranes by toxins can cause the intensity of the cell grouping process, as well as a decrease in the ability of erythrocytes to deform. These processes lead to disturbances in the normal circulation process. It follows from this that the main indicative symptom of the presence of inflammatory processes in the body is the actual restructuring of leukocytes. Neutrophils, which are among 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 character for it, although in the case of excessive generation and activity of neutrophils, pathological processes can be observed, such as a mismatch of the released protease and traumatic tissue damage during necrotic processes [11.13.15.17.19].

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. Currently, it has been established that hyperactive forms of NO compounds that are emancipated by macrophages can initiate a variety of pathological processes that are caused by xenobiotics and their effect on tissues. It also makes sense to assume that in acute poisoning, including poisoning with CC and its derivatives, decompensated acid metabolism can occur, a sharp change in the pH level at micro and macro levels, various disturbances occur in the processes of vital activity of cells and tissues, and the characteristic granularity of the tissue indicating its intoxication indicates two processes at the cellular level: the absorption of toxins by cells and tissues and the maturation of neutrophils. Among patients with a fatal outcome of the disease, exactly the processes described above were observed, already on the first day after poisoning with CC 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 during poisoning, there are changes in the reaction of neutrophil granulocytes (NG) under poisoning conditions, including poisoning with CC and/or its derivatives.

Acute poisoning, accompanied by hypoxia and attacks of suffocation, are often accompanied by a violation of the anti-infectious protection of the body, in particular, at the initial stages of intoxication (0-6 hours), a characteristic increase in the criterion value of the parameters of T-B

lymphocytes in the blood can be noted, which can become a precedent for the development of pneumonia in patients with intoxication.

The characteristic weakening of the immune system of this kind is due to both the action of xenobiotics and hypoxia directly developing under intoxication conditions, free radical oxidation processes, which leads to endotoxicosis and increased hypoxia attacks. The processes of polymorphism in immunity inhibit anti-infective protection at the cellular level; with a violation of 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 with poisoning of the criminal code and its derivatives.

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