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Immune System Dysfunctions in the Development of Systemic Inflammatory Response Syndrome, Multiple Organ Failure, and Inflammatory Complications from Acetic Acid Poisoning

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Relevance of the study. The syndrome of systemic inflammatory reaction, as already described in two paragraphs earlier, is a reactive mechanism of the body to pathogenic effects on it from the outside, including in conditions of poisoning with HC and its derivatives. The nature of CVD is such that in most cases it is accompanied by multiple organ failure (MN), characterized by symptoms already familiar to us such as hypoxia and tachycardia. In case of CVD, laboratory examination is of primary importance, in which it is necessary to identify the actual value of the criteria of LC, amines, N2O, platelet aggregation coefficient and other mediators: in the absence of control over these criteria, the issue of assessing the actual state of health of the patient becomes complex, and if they get out of control during treatment, it is possible There is a serious threat of disruption and dysfunction of multiple organ microcirculation, leading to inflammatory purulent, infectious processes. Judging by studies aimed at studying the nature of CVD, the initial stages of the inflammatory response in the body in CVD are characterized primarily by the generation of inflammatory LCS, which include interleukin-1-beta, interleukins-6 and -8, respectively, and TNF-alpha, and all these cytokines are generated at the cellular level, in the system mononuclear phagocytes; as a rule, the concentration of these in plasma increases significantly with CVR. It is easy to guess and draw the appropriate conclusions that the listed LC are the initiators of a chain of inflammatory reactions in the body, although at the same time, they are the ones that withstand and regulate the inflammatory process as a whole. It should be re-noted that the sharp dynamics (both positive and negative) in the change in the total amount of cytokines in plasma (interleukins-1-beta, interleukins-6, -10, -8) are critical parameters indicating inflammatory processes in the body.

As is known, cytokines serve as transporting agents involved in the processes of blood adhesion, in the processes of cell migration and cell groups, and also serve as criteria parameters indicating a number of physiological parameters, in particular, the adhesion processes between platelets and leukocytes are regulated directly by leukocytes. It should be noted that LC serve as indicating clinical parameters that reflect the functional state of a number of physiological parameters, including from the point of view of toxicology. The chain sequence of reactions due to poisoning with HC and its derivatives is characterized by a sharp increase in the generation of such LC as interleukin-1-beta, interleukins-6 and -10, respectively; at the same time, it is necessary to carefully and regularly monitor the concentration of these in plasma, due to the fact that pathological changes (including and those that lead to death) are interrelated with changes in the concentrations of these same LC in plasma, in particular, excessive generation of them can disable the protective functions of the body and cause its vulnerability to pathogenic influencing factors, which is highly likely to lead to a critical condition for the body. The determination of immune indication criteria and parameters characterizing the body's response to inflammatory processes is carried out in the laboratory. These criteria will make it possible to give an objective assessment of the mechanisms occurring in the body under conditions of CVD, as well as to characterize the symptomatic components of it, whether hypoxia, hypercoagulation or inflammatory processes. The final results of the research aimed at the object of this dissertation over the past years have confirmed the fact that the nature of CVD can be

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predetermined at the genetic level. This means that depending on a person's genotype, which is known to be individual in nature, it is possible to determine possible predictive criteria for pro-inflammatory and anti-inflammatory drugs, as well as the possible nature of the body's inflammatory response under certain pathogenic conditions. The complicating signs of poisoning with CC and its derivatives are considered to be a high rate of negative dynamics of the sober thinking of the poisoned, the observation of ODN and other aspiratory dysfunctions. Obviously, such symptoms imply the need for an immediate connection of the patient to a ventilator. Also, the complicating signs of poisoning with HC and its derivatives include an attack of impaired myocardial assimilation. In addition to the signs described above, other symptomatic signs of acute poisoning with HC and its derivatives include acute catarrhal erosive bronchitis, which begins to develop already at the initial stage of the disease (day 1 of poisoning with HC or its derivatives), and an increase in the concentration of xenobiotics in plasma may set a precedent for the development of pneumonia. The process of signal transmission to the cell nucleus described in the previous paragraph is a cascade of chemical reactions, one of the stages of which is the activation of the key RVR (inflammatory response regulator), which is the cytoplasmic protein NFKB, which, being transported to the cell nucleus, is active through the generation of peptide molecular compounds, which, in particular, include some interleukins such as, for example, IL1, as well as TNF-alpha. Membrane adhesion is disrupted by this process, because In this case, SPN is observed, that is, the process of LC generation is characterized by the flow during SPN.

According to some scientific assumptions in the field of CVD diagnosis, possible chemical microtrauma at the cellular level, which is characterized by the manifestation of pathological processes such as hypercytokinemia and weakening of the immune system, can lead to various infectious pathologies, including acute forms of sepsis, PD, and even possible death. By itself, the syndrome of systemic inflammatory reaction proceeds in several stages due to hypercytokinemia, weakened immunity, etc. As the analysis of the literature in the field of CVD by foreign authors has shown, the formation of CVD includes such processes as increased generation of various inflammatory mediators, cytokine excitation, increased vascular absorption, and the formation of blood clots in blood vessels. The critical diagnostic assessment of CVD and the direct diagnosis of it should be justified and conditioned by a number of symptomatic parameters, which include biochemical criteria, indicative parameters, indications of instrumental examination and examination of infectious foci. Determining the critical value of cytokine content in plasma is one of the main biochemical laboratory tests necessary for performing in conditions of CD and treatment of both CVD and poisoning with HC and its derivatives, and it is also important to determine the level of NO-syntheses as additional indicative criteria reflecting hemostatic characteristics. The onset of the inflammatory process, which is initiated when the standard concentration of a number of interleukins (interleukin-8, -18 and -6, respectively) increases in the blood, occurs in the first 4 hours after poisoning with HC and / or its derivatives, and the clinical picture of pathology, due to its complexity and complexity, is compiled and studied in the form of components, within each of which One or another functional change in the body caused by CVD is being investigated and examined. These components of pathology research include the identification of such symptomatic and pathological manifestations of CVD in case of poisoning with HC and its derivatives as endotoxicosis, hypercoagulation, the development of a blood clot in blood vessels and a general weakening of the immune system. A subspecies of liposaccharide structure, endotoxin, is considered to be a group of key use cases in which CVR is initiated. Endotoxin, which includes a toxic agent, group "A" lipid, is the main element that causes CVD, when released, mediator groups (LC, etc.) emancipate the vast majority of mediator groups in the body, thereby radicalizing them, and as has been shown by the practice of CVD research under similar conditions, when this very emancipation and radicalization of mediator groups, pathologies of metabolic disorders occur in the body, in particular, lactate and amine groups, ketones, LC, lipids. In clinical studies of such issues, it is advisable to conduct autotoxemia, which determines the proteolytic enzymes contained in cells, substances that inhibit cell growth. Such an examination plays an important role in CD sepsis and other necrotic processes in

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CVD. Being integral signs of the entire pathogenesis, the above compounds are analyzed in clinical and laboratory studies as criteria for additional alteration, and in addition, they can significantly affect the metabolic process at the cellular and tissue levels, resulting in the risk of deposition of toxic products of cellular activity in the body, which obviously has a negative character.

Conclusion. Modern theoretical medical reference books describe the algorithmic process of CD in CVD conditions from a therapeutic and surgical point of view at the earliest stages of the disease, which is very optimistic, for the same reason as indicated in the previous paragraph. – early CD and initiated treatment of CVD can significantly simplify the treatment process, although the possibility of using these same methods of early CD and treatment of CVD in case of poisoning with HC and its derivatives has not been studied before, there is no well-founded information base characterizing the inflammatory processes of cellular and tissue levels that occur in the body during poisoning with HC and its derivatives. A specialized study of CVD will make it possible to develop criteria-based measures for assessing and predicting the dynamics of the disease, its timely diagnosis and treatment, as well as propose methods of highly effective immunomodulatory therapy.

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