



Endogenous Intoxication, Activation of Hemostasis and Fibrinolysis Mechanisms in the Pathogenesis of Systemic Inflammatory Reaction Syndrome in Acetic Acid Poisoning

*Kuvatov Zoxid Xayitovich*¹

¹ *Bukhara Branch Of The Republican Scientific Center For Emergency Medical Care*

Annotation. Food poisoning, in addition to a large number of complications, the development of multiple organ failure and fatal cases, can be precedents and cause partial or complete disability, and in a certain percentage of cases, disability of patients: disability of patients to a certain extent is due to the severity and scale of the area of burn injury (burn injury) of a chemical nature. Unfortunately, in some cases, it is difficult in most cases to diagnose and predict the rate of complications in the body due to chemical burns from poisoning of the criminal code and its derivatives.

Often, the outcome of the disease can be predetermined by a set of pathological processes and dysfunctions observed in the conditions of a particular clinical picture of the course of the disease: in particular, endotoxemia caused by emancipation and activation of free radicals activates the mechanisms of hemostasis at the cellular level, causing disseminated intravascular coagulation syndrome. There is a drop in the infectious resistance of the immune system, and the processes of ensuring the vital activity of cells and tissues are subject to complications.

Characteristic of pathologies of almost any etymology is the development of intoxication syndrome at the level of the endogenous layer. This is primarily due to the activation of free radicals and their participation in fermentation processes. In addition to the above, in critical conditions of clinical etymology, the process of intoxication of the endogenous layer is observed, which, again, is due to the activation of free radicals in plasma, in the form of a cascading, multi-stage process; and although these pathological processes are often the objects of research only separately, in cases of critical clinical situations they usually occur in the form of a symptomatic group, which implies the need to study them in a comprehensive manner, due to the fact that such pathological processes cause necrotic processes that disrupt the vital activity of cells and tissues. In fact, endogenous intoxication is nothing more than a typical state of the body and/or the immune system during pathological processes in these, such as intoxication, metabolic disorders, etc., and tissue destructurization should be considered here as the primary criterion factor for the development of endotoxemia at the cellular and tissue levels, initiated by bacteria, infection of various etymologies and subsequent inflammatory processes [1.3.5.7.9.11].

The next stage in the development of endotoxemia includes a decrease in the resistance of biobarriers, accompanied by a violation of microcirculation at the capillary level, intensive activation of detoxifying immune system protection systems, which again is typical for cases of observation in the body of PN, and this is not the only criterion indicating systemic inflammatory processes.



Below are the criteria components for intoxication of the endogenous layer:

observations of substances in blood plasma with an average Mmol = 300.0-4900 Dt, and the average population of which increases due to the activity of proteases and other agents;

fat oxidation products;

composite complexes of the immune system;

Endotoxin, as a bacterial toxin;

mediators (histamine, kinins, some LC, platelets).

For the diagnosis of endotoxemia, the use of other indicative indicators is also characteristic: analysis of the chemical composition of urea, bilirubin content in the blood, cholesterol content, ALT/ACT criteria, pH type of medium, etc., although depending on the concept of intoxication of the endothelial layer, the method of clinical and/ or laboratory diagnostics appropriate in a given situation may differ, due to the fact that, purely from the point of view of the pathogenesis of endotoxemia, tissue lesions of a very different nature are observed in the treatment of CC and its derivatives. Nevertheless, measures to eliminate, CD and treat intoxication are reduced to one goal – its speedy removal.

Reasoning about the present process as a systemic inflammatory, one should pay attention to the fact that the release of the results of the process of protein activity (the release of metabolic products) does not stop as a process, and the same applies to the activity of mediators that stimulate the mechanisms of the body's resistance to toxic elements to which. first of all, the neutrophils already familiar to us belong. The titers of medium-molecular peptides increase, especially with poisoning of the CC and its derivatives by 2-3 times in relation to the standard (normal) parametric values of these, the criterion coefficient of neutrophil shift increases, the balance between the quantitative parameters of the antioxidant system and the process of fat oxidation indicates the beginning of hyperviscosity of blood [2.4.6.8.10.12].

Endotoxemia and its severity are characterized by several criteria, including laboratory ones. There are certain criteria indices, determining the values of which it is possible to determine the actual value of the index of leukocyte intoxication. for example, the universal leukocyte intoxication index. It has been proved that dysfunctions in hemostasis are in no way related to the classification of the toxicant, but are more related to the manifestations of early symptoms of DIC syndrome and that criteria indicators of activation parameters of the blood clotting process are associated with the diagnostic criterion of D-dimer. This dimer is a product of fibrin degradation, and is part of microthrombs. The percentage of D-dimer in plasma is directly proportional to the intensity of the fibrinolysis process [11.13.15.17].

As it is known from foreign sources in the CVD clinic for poisoning with CC and its derivatives, in acute forms of poisoning with it, processes of polymorphism of the thrombocytic background of hemostasis are observed, which entails an increased rate of platelet interaction due to an increased content of D-dimers. The D-dimer, in this case, serves as a criterion indicating parameter that characterizes the actual adhesive ability of both platelets and lymphocytes.

The success of CD and the treatment of exotoxic shock posed a new challenge for researchers – the need to develop methods of CD and treatment of infectious complications against the background of ES. Pneumonia is an extremely dangerous pathology for health, which is accompanied by a high degree of intoxication of the patient, and esophagitis developing as a result of an acid reaction between the esophagus and infection causes pustular and cicatricial lesions of the mucous membrane. Pronounced burn lesions of the mucous membranes of the stomach and esophagus manifest themselves in the form of a violation of the protective functions of these, there is a change in the population of microflora and the dynamics of these symptoms is characterized by the severity of the inflammatory process, and according to research by a number of foreign authors, the dissertation determined that hypoxia, or rather, its critical forms are a precedent for inflammatory reactions in the body, due to the fact that they excite the "triggers" of mediators, activate LC and kinins, lower vascular resistance and create conditions for hyperviscosity of blood. All this creates conditions for the formation of blood clots.

The sequence of inflammatory reactions, which are often also accompanied by hypoxia attacks, is largely associated with the activation of proteolysis and blood clotting. In medical practice, it often happens that



the use of correct CD algorithms in the diagnosis of patients makes it possible to predetermine possible potential pathologies at an early stage, however, it should be remembered that the use of these CD algorithms is not always possible in clinical toxicology, in particular, in poisoning with CC and its derivatives, although in any case, the possibility or impossibility of this has not yet been considered and investigated earlier in the scientific aspect. There are no finalized works, the research of which is aimed at a comprehensive analysis of the clinical picture of symptoms, CD and treatment of poisoning with CC and its derivatives [12.14.16.18.19.20].

The degree of high pathogenicity, resistance to antibiotics and anti-infective drugs of microorganisms pose great difficulties in the prevention and treatment of this pathology and force us to look for new approaches to solving this problem.

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