

# To Determine the Clinical Symptoms of the Systemic Inflammatory Response in Acute Severe Poisoning by Agents Affecting the Central Nervous System

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**Relevance.** The current stage of national healthcare is characterized by an increase in the number and severity of acute poisoning with drugs, which, according to the WHO International Classification of Diseases and Health-Related Problems of the 10th revision, are included in the T36-T50 group. According to the International Chemical Safety Program, accidental and intentional acute poisoning of medicinal origin accounts for up to 40-60% of all acute chemical pathology in the developed world. A similar pattern is observed in modern Russia, while 65-70% of acute drug poisoning is caused by drugs of psychopharmacological action (ICTC of the Ministry of Health of the Russian Federation, 2002). The most common psychotropic drugs that cause severe acute poisoning include the neuroleptic azaleptin (leponex) and the antidepressant amitriptyline, as well as combinations of psychotropic medications (Ostapenko Yu.N. et al., 2002). The severity of the condition of patients with acute poisoning is due, on the one hand, to the formation of exotoxic shock, on the other, to pronounced metabolic disorders with the development of endogenous intoxication syndrome (Kosonogoe L.F., 1995). As a result, multiple organ failure is formed, which is the direct cause of death (Luzhnikov E.A., Goldfabr Yu.S., 1995).

The development of this pathology naturally implies the occurrence of violations of the membrane functions of organs and tissues, among which the leading ones are violations of the processes of mitochondrial respiration (Glushkov S.I. et al., 2002). Metabolic disorders and hypoxia lead to an increase in the level of free radicals and the accumulation of substances that catalyze lipid peroxidation (POL), a shift in the equilibrium in the POL-AOS system towards peroxidation products (Pirozhkov C.B, 1997; Flohe L., 1982; Weiss S.J., 1986).

Currently, acute poisoning with substances affecting the central nervous system in the general structure of exogenous intoxications accounts for up to 50-60% of cases, of which up to 25% are severe and extremely severe lesions leading to the formation of critical conditions and requiring resuscitation treatment (Bonitenko E.Yu. et al., 2018; Luzhnikov E.A. et al., 2016; Bohnert A.S. et al., 2019; Muller D. et al., 2017; Taheri F. et al., 2015). The causes of widespread acute poisoning include the uncontrolled use of various medicines in everyday life, suicidal attempts, as well as the use of narcotic drugs for the purpose of drug intoxication (Rokhlina M.L., 2019; Nordstrom D. L. et al., 2017; Taheri F. et al., 2018). The severity of the condition of patients with acute severe poisoning by drugs affecting the central nervous system is due to pronounced dysfunctions of various organs and body systems due to the specific action of xenobiotics (Luzhnikov E.A. et al., 2016), as well as developing hypoxia (Livanov G.A. et al., 2014; Hashemian M. et al., 2016), leading to the development of life-threatening complications, which often determines the outcome of chemical trauma. A number of authors note that hypoxia in critical conditions causes the formation of a systemic inflammatory response consisting of increased production of pro-inflammatory mediators, activation of cytokines and kinins, increased vascular permeability, increased blood viscosity and microthrombosis (Chereshnev V.A. et al., 2019; Cavaillon J.M. et al., 2016; Ramakrishnan S. et al., 2019). Systemic inflammatory reaction and hypoxia are always associated with activation of proteolytic processes, coagulation and fibrinolytic systems (Sanotsky V.I., 2013; Alekhovich A.V., 2020).

Numerous authors have shown in their works that with the intensive and prolonged action of

inflammatory factors, numerous disorders develop both at the cellular and organ levels (Gusev E.Yu. et al., 2018; Savelyev V.S. et al., 2017; Alberti C. et al., 2015).

There are reports in the literature of widely used diagnostic algorithms for systemic inflammatory response syndrome in patients with intensive care, therapeutic, and surgical profiles, which allows early diagnosis of infectious complications and timely initiation of therapy (Zhevlakova Yu.A., 2017; Akimova V.N. et al., 2016; Ratzinger F. et al., 2015; Boehme A. K. et al., 2014). However, the possibility of their use in clinical toxicology has not been considered or investigated to date.

There are no comprehensive studies on the problem of systemic inflammation in acute poisoning with drugs affecting the central nervous system, and methods for correcting this component of the pathogenesis of acute exotoxicosis have not been sufficiently defined. The mechanisms of pharmacological action on pathological reactions that are components of the systemic inflammatory reaction syndrome, in particular, such as proteolysis and the blood coagulation system, have not been determined, which seems relevant.

There is no information about the role of systemic inflammatory reaction syndrome in the development of infectious complications in patients with acute poisoning by drugs affecting the central nervous system, which are one of the reasons for the increased duration of treatment of patients and the possible risks of death in the somatogenic phase of acute poisoning.

Of no small importance are the long periods and difficulties in treating the developed inflammatory complications. Thus, these issues require detailed study, since their solution involves the possibility of improving the effectiveness of treatment of this patient population. In connection with these arguments, the solution of the above problems through the implementation of the planned research work is timely and relevant.

An analysis of the currently available literature data allows us to define the concept of "systemic inflammatory response" as a conditional one reflecting the total (systemic) nonspecific inflammatory response of the body to various stimuli and pathological agents (injuries, burns, infections, etc.), which is manifested by the activation of all mediator systems and pathological biochemical cascades responsible for inflammation, where the cytokine network and acute-phase proteins play a leading role.

CB is part of the general adaptation syndrome, a separate phenomenon of systemic inflammation (CB) - "a typical multisyndromic, phase-specific general pathological process that develops with systemic damage and is characterized by total inflammatory reactivity of endotheliocytes, plasma and cellular factors of blood, connective tissue, and in the final stages and microcirculatory disorders in vital organs and tissues" (Chereshnev V.A., 2014). In the presence of certain clinical diagnostic criteria (according to Bone R.C., 1996), CB acquires a purely clinical concept - systemic inflammatory response syndrome (SIRS), which is considered as an important pathogenetic link in critical conditions of various etiologies of a non-infectious and infectious nature with a risk of developing multiple organ failure syndrome (Gusev E.Yu. et al., 2017; Levit D.A. et al., 2017; Savelyev V.S. et al., 2016; Bone R.C. et al., 1992). Cytokines, products of hemostasis activation, free radicals, biogenic amines, nitric oxide, platelet aggregation factor, neuroendocrine humoral factors and other mediators play an important role in the development of the body's CVD to damaging factors of various nature (Chereshnev V.A. et al., 2012; Gusev E.Yu. et al., 2014; Olson N. et al., 2011; Neri M. et al., 2013; Bernstein H.G. et al., 2014; Boehme A. K. et al., 2014).

**Conclusion.** There is practically no complete information in the literature on the comprehensive assessment and diagnostic capabilities of CVD in patients with acute poisoning by neurotropic agents. The possible connection between acute poisoning, purulent infection and the outcomes of acute poisoning with substances of neurotropic action is not considered. In this regard, the subject of this study will be the diagnosis of CVD and the assessment of its role in the pathogenesis of acute poisoning with substances of neurotropic action, as well as the development of a pathogenetically sound method of its treatment.

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