Medical Care for Patients with Acute Acetic Acid Poisoning with Exotoxic Shock

Quvatov Z. S., Abdurahmonov M. M.
Bukhara branch of RNCEMP, Bukhara, Uzbekistan

ABSTRACT

Relevance: Due to the wide availability of acetic acid, acute poisoning with acetic acid is one of the most common etiological factors of household poisoning throughout Uzbekistan, and their specific gravity is quite large from 8.5 to 14.7%, and the mortality rate reaches 19 - 22.5%.

The aim of the study was to identify early markers of the development of systemic inflammatory response syndrome and sepsis manifestations in patients with severe acute acetic acid poisoning.

Materials and methods: The study was conducted in 124 patients with acute acetic acid poisoning. As indicators of the activity of inflammation, the following were studied: the content of IL-6, IL - 10 and procalcitonin (PCT) in blood serum.

Results: The increase of IL-6 content in the blood of patients to the level of 54-214 pg/ml at admission was determined. With the development of sepsis, an increased concentration of IL-6 (up to 115 pg/ml) was observed up to the fifth day. The IL-10 content in the blood of patients with subsequent sepsis upon admission was significantly higher (56-113 pg/ml) than in the blood of patients without sepsis. The concentration of IL-10 correlated with the development of sepsis (r=0.8) and the frequency of deaths (r=0.5). The content of PCT increased starting from the third day in patients with sepsis to a level exceeding the diagnostic value for sepsis (2 ng/ml).

KEYWORDS: *acetic acid poisoning, interleukins, procalciotonin, systemic inflammatory response, sepsis.*

They cause the development of tissue hypoxia associated with violations of the acid-base state and the direct depressing effect of high doses of drugs on respiratory enzymes. CC has a local cauterizing effect by the type of coagulation necrosis and a pronounced resorptive hemato-, nephro- and hepatotoxic effect due to hemolysis of erythrocytes, the development of toxic coagulopathy, the syndrome of scattered intravascular coagulation. The cauterizing effect is most pronounced in the respiratory tract and in the gastrointestinal tract. Necrotizing is not only the mucous membrane - the process can spread to the entire thickness of the submucosal and muscular layers. When taking CC, burn disease of chemical etiology develops due to the local destructive effect on tissues and its resorptive effect.

As a result of massive plasma loss, there is a decrease in the volume of circulating blood, and at the same time a decrease in all indicators reflecting central hemodynamics: cardiac output, shock and minute heart volumes, central venous pressure and blood flow velocity, which forms the development of acute circulatory insufficiency, which is classified as exotoxic shock (ES) by hypovolemic type. One of the main characteristic signs of the development of systemic inflammation is an increase in the concentration of inflammatory and anti-inflammatory cytokines in the blood.

Treatment included infusion therapy, relief of pain syndrome and ESH, which includes the

administration of narcotic drugs (morphine, promedol, omnopone), neuroleptanalgesia, cholinolytics (papaverine, atropine, platyphylline) subcutaneously 3-5 times a day, forced diuresis with mandatory blood alkalinization (up to 800-1500 ml of 4% sodium bicarbonate intravenously to relieve hemolysis), administration of colloidal and crystalloid solutions in a ratio of 1:3 (in severe cases up to 8-12 liters) with simultaneous administration of 4-12 ml of torsid under the control of CVD, as well as antibacterial and symptomatic therapy. The development of systemic inflammatory response syndrome (SERS), sepsis was determined by the presence of signs presented in the recommendations of the International Guidelines on Severe Sepsis and Septic Shock (The Third International Consensus Definitions for Sepsis and Septic Shock - Sepsis-3 (2016)). The severity of multiple organ failure associated with sepsis was assessed according to the SOFA scale (1994) adopted by the European Society of Intensive Care. The degree of hypoxia in the victims was assessed by the level of RaO2 in arterial blood. As indicators of the activity of inflammation, the following were studied: the content of IL-6, IL - 10 and procalcitonin (PCT) in blood serum. The studies were performed upon admission of patients to the hospital, 24 hours later, on the 3rd and 5th days after poisoning. Upon admission and on the 3rd day after the incident, the patients underwent a bacteriological examination of the material from the pharyngeal mucosa, urine and blood in order to identify pathogenic and conditionally pathogenic microflora.

In acute poisoning with acetic acid, the clinical picture in all patients was characterized by depression of consciousness to the level of coma I-II degree (6.1 ± 0.8 points on the Glasgow coma scale). Comatose state with acetic acid poisoning was accompanied by the development of acute respiratory failure of mixed genesis. The central mechanisms of respiratory depression were combined with obturation disorders as a result of bronchorrhea and hypersalivation.

On the 3rd-7th day sepsis was diagnosed in 35 patients (40.7%) - in 9 patients (10.5%), which was confirmed by the results of bacteriological analysis. The results of clinical and laboratory studies in patients with acute poisoning showed that all patients had 2 or more clinical (tachycardia 122±9.7 beats/min, tachypnea 30±3.7 times/min, hyperthermia 39.7±0.8 S0) and laboratory (leukocytosis 18.4±1.8-109/l, pCO2 33.2±1.1 mmHg) of signs of CVD, and in patients with subsequent development of severe sepsis, a greater number of signs of CVD (3.4± 0.5) were observed compared with the rest of the patients. By day 5, in patients without septic complications, the number of signs of CVD decreased from 2.0 ±0.5 to 0.8± 0.5 ($p<0.05$), while in patients with severe sepsis, a significant increase in signs of CVD and organ dysfunction was observed.

An increase in the IL-6 content in the blood of patients to the level of 54-214 pg/ml was observed upon admission; after 1 day, the cytokine level in patients with CVD decreased to normal values, whereas in severe sepsis, an increased concentration of IL-6 (up to 115 pg/ml) was observed up to 5 days. The concentration of IL-6 in the blood on day 3 significantly correlated with the development of severe sepsis ($r = 0.45$). The IL-10 content in the blood of patients with subsequent development of severe sepsis upon admission was significantly higher (56-113 pg/ml) than in the blood of patients without severe sepsis. The concentration of IL-10 correlated with the development of severe sepsis ($r=0.8$) and the frequency of deaths ($g = 0.5$). The content of PCT increased starting from the third day in patients with sepsis and severe sepsis to a level exceeding the diagnostic value for sepsis (2 ng/ml) [8]. The level of PCT on the 3rd day after the incident in patients was significantly correlated with the development of severe sepsis ($r = 0.6$).

Conclusions.

1. In patients with ESH with severe acute acetic acid poisoning, the development of CVD caused by hypoxia ($pO2 = 68.5 \pm 6.5$ mm Hg) was observed already upon admission to the hospital.
2. By 3-5 days after admission, 14% of patients had the development of a septic condition, the

laboratory predictor of which on day 1 was a high blood content of IL-10 (15 times or more).

3. An increase in the blood content of IL-6 (more than 115 pg/ml) and procalcitonin (more than 2 ng/ml) on the 3rd day after toxic exposure can be considered as early diagnostic criteria for the progression of sepsis.

LITERATURE.

1. Akalaev R.N., Stopnitsky A.A. Algorithm of application of substrate metabolic drugs at the early stage of acute poisoning with acetic acid. Emergency medical care 2014; 2: 9-13.
2. Ketlinsky S.A., Simbirtsev A.S. Cytokines / St. Petersburg: LLC "Foliant Publishing House", 2008. - 552 p.
3. Kutsenko S.A. Fundamentals of toxicology. / S.A. Kutsenko. - St. Petersburg: Foliant, 2004. - 670 p
4. Marupov A.M., Stopnitsky A.A., Shoabsarov A.A. Intensive therapy of patients with acute poisoning with acetic acid. Bulletin of Extra Medicine 2008; 1: 78-83.
5. Provado, A.V. Morphofunctional changes in parenchymal organs in acute poisoning with acetic acid in various stages of stress reaction : abstract. dis. ... Candidate of Medical Sciences / A.V. Provado. — Irkutsk, 2007. — 22 p.
6. Stopnitskiy A.A., Akalaev R.N. Intensive therapy of patients with acetic acid poisoning complicated by the development of exotoxic shock. General reanimatol 2014; 10 (2): 18-21.
7. Urazaev, T. H. Clinical and laboratory tests in assessing the condition of patients with acute acetic acid poisoning : abstract of the Candidate of Medical Sciences / T. H. Urazaev. — Yekaterinburg, 2004. — 22 p.
8. Becker K.L., SniderR., Nylen E.S. Procalcitonin in sepsis and systemic I. - 2010. - Vol. 159. - P. 253-264.