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EXPERT ASSESSMENT OF THE RISK OF DEATH AND SEVERITY OF CHEMICAL INJURY IN ACETIC ACID POISONING AGAINST THE BACKGROUND OF ALCOHOLIC INTOXICATION

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Abstract

The article provides a quantitative assessment of the risk of death of victims from acetic acid poisoning against the background of various degrees of alcohol intoxication. The justifications for determining the expert criterion for the severity of chemical injury in combined poisoning are given.

Keywords: *risk of death, probit analysis, hemolysis, free hemoglobin, alcohol intoxication.*

Relevance of the problem:

In the structure of violent death, acute poisoning occupies a leading place in forensic medical practice. With the development of the chemical industry and the increase in the areas of application of chemicals, especially in everyday life, every year there is a tendency to an increase in acute poisoning with caustic poisons, in particular, acetic acid (Bakirov A. B., 1990).

Forensic medical diagnosis of acetic acid poisoning, as a rule, does not cause acute difficulties. However, to date, quantitative criteria have not been developed to substantiate the main cause of death in these poisonings against the background of alcohol intoxication. Meanwhile, it should be noted that in most cases (86.8%) acute poisoning of acetic acid occurred against the background of alcohol intoxication. This is due to the fact that to date there are no scientifically based criteria for assessing the risk of death and the severity of chemical injury in such combined poisoning.

In this regard, the **purpose of this study** was to develop and substantiate an objective criterion for assessing the causes of death and the severity of chemical trauma in these intoxications.

We consider it necessary that such a criterion meet the following conditions:

— it should be recorded in all cases of acute acetic acid poisoning;
— it must have a significant impact on the entire pathological process;
— the degree of its severity should correspond to the severity of poisoning;
— it must be acceptable both in the expert assessment of the severity and in the assessment of fatal poisoning;
— It must be quantifiable.

It is known that the triggering factors for the pathogenesis of these poisonings are: resorption of hydrogen (H+) acid ions into the vascular bed and hemolysis of erythrocytes in the changed (acidic) environment of the blood against the background of a chemical burn of the gastrointestinal tract. It is these factors, first of all, that should be considered as objective criteria for assessing the severity and risk of death in these poisonings.

It should be noted that the above main criteria for assessing the risk of death and the severity of chemical trauma in these poisonings are closely intertwined and interrelated. It remains to single out the main criterion.

Materials and methods of research

The material for this study was the clinical and sectional data of 140 victims of acute acetic acid poisoning, who were treated at the Republican Scientific Center for Emergency Medical Care. Among them, in 90 cases (64.3%) the victims were in various degrees of alcoholic intoxication. Examinations of corpses were carried out according to the generally accepted method no later than 24 hours from the moment of death. The outcome was observed in 60 patients (67.6%). Among them, 43 (71.7%) were men and 17 (28.3%) were women, aged 15 to 63 years (average 35.6±6.3 years).

In the work, toxicological, forensic chemical and histological methods of research, as well as multivariate methods of statistical analysis, such as probit analysis "poison concentration – effect" and nonlinear registration analysis, were used.

Results and discussion

As mentioned above, first of all, it was necessary to determine the main forensic criterion for assessing the severity of chemical trauma in acetic acid poisoning against the background of alcohol intoxication.

It was of particular interest to establish how much the pH of the blood and the level of free hemoglobin change in accordance with the increase in the length of the chemical burn of the gastrointestinal tract.

We studied this ratio with a favorable outcome and in cases of fatal poisoning in these intoxications and determined a certain pattern: with an increase in the length of a chemical burn, the gastrointestinal tract pH of the blood decreases, and the level of hemoglobinemia increases. In turn, with a favorable outcome of poisoning, against the background of the same type of burn area, acidemia and hemolysis were always much smaller.

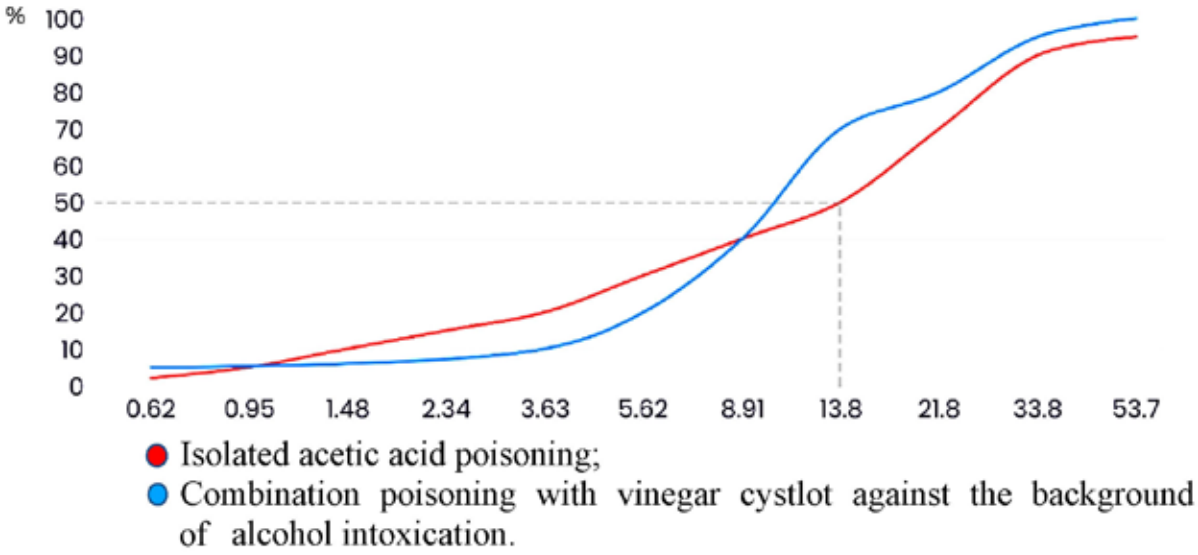
We see the explanation for this fact in the fact that against the background of the use of massive alkalizing therapy for these poisonings, there is a rapid neutralization of H^+ acid ions and the restoration of blood pH to normal values. Thus, a situation is created when the root cause of pathological disorders (resorption of H^+ acetic acid ions into the vascular bed) is less traumatic for the body than its consequences (intravascular hemolysis). This is also due to the fact that hemoglobinemia has a number of additional damaging effects on homeostasis and hemostasis (violation of the rheological properties of the blood, influence on its coagulation system, etc.). Therefore, the severity of acetic acid poisoning is most objectively indicated by the depth and extent of the chemical burn of the gastrointestinal tract, as well as the level of intravascular hemolysis. Since hemolysis is easier to quantify, it was chosen as the main expert criterion of severity in these poisonings.

Until now, in forensic medical and clinical toxicology, the degree of toxicity in poisoning with caustic acids was carried out according to the level of free hemoglobin in the blood. However, these data are not always reliable, since in most cases of poisoning occur against the background of alcohol intoxication. Currently, there are no specific scientifically based recommendations for assessing the severity of chemical injury.

Of all the possible methodological approaches to solving this problem, we have chosen the method of "probit analysis". It is based on the well-known mathematical the-

ory of hitting "targets" and consists in the graphical construction of the relationship "concentration of free hemoglobin – effect" (Figure 1).

Figure 1. *Probit-graph of the relationship "concentration of free hemoglobin – effect" in acetic acid poisoning against the background of alcohol intoxication on the abscess axis – hemolysis level (mg/ml); on the ordinate axis is the percentage of risk of death*



On the graph, this dependence is displayed in the form of a curved line with a characteristic S-shape. Its lower horizontal level shows to what extent the fatal outcome has not yet been observed for an increase in hemoglobinemia. Then the curve of the graph gradually rises upwards. Its steepness or angle of inclination is an objective indicator of the degree of toxicity of chemicals.

At the top of the graph, the curve reverts to a horizontal position. This means that a further increase in hemolysis is no longer accompanied by an increased risk of death. Within this asibole, in most cases (from 80 to 100%), death occurs. Against the background of alcohol intoxication in acetic acid poisoning, the risk of sweeping increases. For example, if in isolated poisoning with acetic acid alone, the DM of 50 is equal to 13.8 mg/ml, then in poisoning combined with alcohol, the DM of 50 decreases to 8.91 mg/ml. However, it should be noted that alcohol intoxication here was severe (from 2.5 to 3.0‰ per mille). At lower concentrations of ethyl alcohol in the blood (0.5–1.5‰ ppm). We observed the opposite effect, i.e. alcohol in moderation reduces the risk of death (Table 1).

Table 1. *Results of toxicometry of the relationship "concentration of poison – effect" in combined poisoning*

Type of poisoning	Toxicometry parameters				
	SD05	SD25	SD50	SD75	SD95
Isolated poisoning (acetic acid)	1.48	5.62	13.8	33.8	53.7
Combined poisoning (acetic acid + + alcohol mild intoxication	2.36	7.84	21.8	33.4	62.8
Combined poisoning (acetic acid + + alcohol severe intoxication	0.97	3.86	6.83	12.92	23.62

As follows from the table, the effect of alcohol intoxication on the course of the pathological process and the outcome of acetic acid poisoning is ambiguous. At low

concentrations in the blood in the range of 0.5–1.5% per mille, alcohol has a beneficial effect on the course of chemical disease and the risk of death is much lower than in isolated poisoning with acetic acid alone. Thus, at these concentrations of alcohol in the blood, the risk of death decreases, as evidenced by the value of DM 50, which increases from 13.8 mg/ml (in isolated poisoning) to 21.8 mg/ml (in combination with alcohol). Whereas in severe alcohol intoxication (from 2.5 to 3.5‰ ppm), the course of the pathological process becomes more severe in comparison with isolated acetic acid poisoning and the risk of death increases sharply, DM 50 decreases from 13.8 mg/ml to 6.83 mg/ml ($P<0.001$).

Thus, with the help of this graph, it is possible to quantify the risk of death at different levels of acetic acid intoxication against the background of alcohol intoxication.

The results of these studies indicate that the concomitant intake of moderate doses of alcohol can neutralize the toxic effect of acetic acid, i.e. free hemoglobin. This "protective" effect of ethinol is apparently explained by the fact that ethyl alcohol induces metabolic enzymes of the liver in small quantities, thereby accelerating the biotransformation of kinobiotics (free hemoglobin), reducing their biological activity and toxicity (4, 5).

The second phenomenon of the effect of alcohol is that its concomitant intake reduces the risk of death mainly in the zone of critical concentrations of free hemoglobin, but reduces its protective effect in the zone of content close to the level of biological irreversibility (CD75-CD100). At the same time, the toxic effect of alcohol in concentrations over 2.5‰ ppm in the blood increases significantly. This appears to be due to the fact that the increased blood alcohol concentration contributes to the increased toxic effect of free hemoglobin, i.e. both toxic substances act as synergists, increasing each other's toxicity, increasing the risk of death.

Findings

1. The severity of chemical disease in acetic acid poisoning is most objectively evidenced by the depth and extent of the chemical burn of the gastrointestinal tract and the level of intravascular hemolysis.

2. The level of intravascular hemolysis was chosen as the main expert criterion for assessing the severity of chemical trauma in acetic acid poisoning against the background of alcoholic intoxication.

3. The effect of alcohol intoxication in acute acetic acid poisoning is ambiguous: alcohol in small concentrations has a beneficial effect on the outcome of poisoning, improving the biotransformation of free hemoglobin, and in large concentrations it sharply increases its toxic effect.

4. The method of probit analysis of the relationship "poison concentration – effect" allows to give a quantitative assessment of the main cause of death in these combined poisonings.

References

Bakirov A. B. Poisoning acetic acid // forensic medical examination. 1990. T33, – 31. – 56 p.
Goncharova L. N., Ivanova N. V. Mathematical forecasting of the course and outcomes of acetic acid poisoning // therapeutic archive, 2003. – T. 48. в 7. – P. 100–109.
Iskandarov A. I. et al. Toxicometric assessment of acute poisoning with cauterizing poisons. – T. 2016. – P. 47–49.
Luzhnikov E. A. Clinical toxicology. – Moscow, Med, 1982. – 368 p.
Mutalipov M. M. Ispol'zovanie probit-grafika v sudebnoy meditsine (The use of probit-graphics in forensic medicine). – Alma-Ata, 2002. – No. 1(2). – P. 34–35.
Principles and methods for assessing the toxicity of chemical substances, WHO – M. Med, 1983. 2.1. – 312 p.
Boyce S. H., Simpson K. A. Hydrochloric acid inhalation, who needs admission. J. Accid Emerg. Med., 1996. – Now/13 (6). – P. 422–424.
Pivovarov G. N. Erroneous diagnosis of myocardial infarction in acid poisoning. Lik Sprava. 2000. Sep (6). – P. 83–84.

Su M., Nelson L. Eur massive necrosis of the gastrointestional tract after ingestion of hydrochloric acid. J. Surg. Oct. 2015. – 167 (10). – 798 p.

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