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Poisoning with Ethanol and Alcohol Surrogates

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Abstract Acute poisoning with ethyl alcohol and its surrogates is characterized by clinical signs of poisoning, dependent primarily on the concentration (dose) of consumed poison and its physical-chemical composition. Forensic diagnostics should be based on the results of clinical pattern and forensic chemical blood and urine tests of the victim.

Keywords Forensic expertise, Acute and chronic alcohol intoxication, Thanatogenesis, Morphological alterations

According to WHO, a quarter of all poisoning cases is taken by alcohol poisoning (WHO, 2016). More than 60% of all lethal poisoning cases are also included into that group. Basic reasons of lethal poisoning are considered to be consumption of a great amount of alcohol, especially fasting, chronic alcoholism, specific lifestyle, and family alcoholism [1,3]. Ethanol penetrates tissue membranes easily, fast absorbed in stomach (20%) and small intestine (80%). In average 1.5 hours its concentration in blood reaches a maximal level. Ethanol low doses act as a selective depressant on CNS, while its high doses act as general depressant; it has psychotropic (narcotic) effect, which is accompanied by suppression of excitement processes in CNS due to neuron metabolism, disorders in mediator system functioning, and inhibition of oxygen utilization [2].

A significant role in the pathogenesis of poisoning is played by metabolic toxicosis and acidosis (accumulation of ethanol biotransformation products). Basic endogenous product is poisonous acetaldehyde formed in all variations of oxidative degradation of ethyl alcohol expressed intoxication develops when acetaldehyde dehydrogenase cannot convert acetaldehyde completely into acetate convert it to acetate. Acetaldehyde disrupts epinephrine and other catecholamines cerebral and peripheral cycle, it effects cardiovascular system, liver, and kidneys [4].

Main reasons of lethal outcomes of ethanol poisoning are acute cardiac failure, liver cirrhosis, renal failure, and total intoxication.

The objective of the study was design of clinical and morphological assessment criteria for acute ethyl alcohol and its surrogates poisoning in hot climate.

Materials for study were conclusions of forensic expertise of ethyl alcohol and its surrogates acute poisoning cases from 2010 to 2017, and results of our own autopsies (82 corpses) of people, who died due to alcohol poisoning and 76 cases of alcohol and its surrogates poisoning in alive people, treated at the Republican Scientific Emergency Medical Center (RSEMC). We performed the analysis od all clinical and laboratory research methods used in ethanol alcohol and its surrogates poisoning cases in RSEMC, and applied common morphological tests in lethal cases of this persons.

Results of the study: Alcohol poisoning develops from stage to stage. Clinical progression depends on the dose. Acute poisoning should be suggested when health status of a person, who consumed a great dose of alcohol, suddenly deteriorates with development of consciousness impairments, loss of ability to walk, perception disorders, occurrence of stupor and coma. On the basis of the data of clinical pattern and taken from victims treated at RSEMC and results of forensic expertise we designed a table of acute ethanol poisoning in the conditions of our country.

Symptoms of alcoholic coma are not specific and represent a variation of narcotic coma. It is characterized by obturative-aspirative disorders (tongue retraction, hypersalivation, bronchorrhea, vomiting mass aspiration), stridor, tachypus, acrocyanosis, cervical veins swelling, probability of appearance of large bubbly wheezing in lungs, pupils widening. Death can occur due to respiratory center paralysis, occlusion of respiratory ways by vomiting mass.

In autopsy of corpses of people died because of alcohol poisoning we did not find any specific alterations. Often there was cyanosis and swelling of face and eye-lid edema. Blood was thickened. In 93% there were tardes spots on cardiac and pulmonary surface. There was notable plethora and edema of meanings, brain, and lungs. Bladder was overfilled with water in 87% of the cases. Autopsy of cavities was always accompanied by alcohol smell.

Ethanol surrogates are its substitutes. These include all other alcohols (methyl, buthyl, propyl, etc.) and ethylene glycol, dichloroethan, and substances often used for intoxication.

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Table 1. Dynamic clinical pattern of acute ethanol poisoning dependent on ethyl alcohol concentration (dose) in blood

Alcohol concentration in blood, % mass/ volume	Alcohol effect stage	Clinical manifestations
0.01 – 0.05	Sobriety	There is no clear effect. For a usual observer behavior is normal. Slight changes are revealed by special tests.
0.03 – 0.12	Euphoria	Mild euphoria, sociability, talkativeness. Increase self-confidence, weakening of inhibition reactions. Weakening of attention, judiciousness, self-control, loss of subtle operations and manipulations capability.
0.09 – 0.25	Excitement	Emotional instability, weakening of inhibitory reactions. Loss of distribution. Weakening of memory and cognition. Weakening of sensory response, increase of reaction time. Mild disorder of coordination of movements.
0.18 – 0.30	Confusion	Disorientation, confusion, dizziness. Intensive emotions (fear, anger, sadness, etc.). Sensory dysfunction (dyplonia), color, shape, motion, and size perception disorders. Rise of pain threshold. Disorder of equilibrium, expressed disorder of coordination of movements, staggering gait, slurred speech.
0.27 – 0.40	Stupor	Apathy, total inertia, approaching paralysis. Notable weakening of reactions to any stimuli. Loss of coordination of movements, inability to stay and walk. Vomiting, urinary and fecal incontinence. Blurred consciousness, deep sleeping and stupor.
0.35 – 0.45	Coma	Complete loss of consciousness; anesthesia. Reflex suppression or absence. Decrease in body temperature. Urinary and fecal incontinence. Circulatory and respiratory disorders.
0.50 and more	Death	Possibility of lethal outcome. Death due to respiratory muscles paralysis.

The portion of methyl poisonings in our study was equal to 3.5% of the total number of deaths due to acute poisoning. Methyl alcohol is easily absorbed to blood. Initially it has weak narcotic effect with further suppression of oxidative processes in tissues and development of oxygen starvation. Lethal dose in these cases was from 40 to 100 mL of consumed poison.

In the clinical progression we distinguish the following doses:

1. narcotic, manifested by poisoning symptoms;
2. toxic, causing renal and cardiac disorders;
3. CNS damage manifested primarily by loss of vision acuity.

Morphological pattern was not expressed. Presence of sweet-sugary smell coming from brain and open cavities and reddish gray cadaveric spots are worth paying attention.

Poisoning by ethylene glycol in our study was registered in two cases. Ethylene glycol is included into the composition of break fluid and it is used as anti-combiner. In the cases studied in our research ethylene glycol was used for suicide. In human body it is decomposed to very toxic products of glycol and oxalic acid. As a result there is damage of CNS, sudden excitement, clamps, followed by loss of consciousness, respiratory and cardiac system disorders. In relation to the formation of insoluble oxalic acid salts, which cause occlusion renal channels, there is development of acute renal failure.

The most severe disorders were observed in cases of poisoning with dichloroethane (6 cases). Dichloroethans are widely used as solvents for dry cleaning, for adhesion and other purposes. It effects almost all organs, and primarily, cardiovascular system, liver, and kidneys. It causes fast development of cardiac failure, which hardly treated, with development of unconsciousness with expressed convulsions.

Conclusion: Acute poisoning with ethyl alcohol and its surrogates is characterized by clinical signs of poisoning, dependent primarily on the concentration (dose) of consumed poison and its physical-chemical composition. Forensic histological and macroscopic patterns of these kinds of poisoning have no specific features. Forensic diagnostics should be based on the results of clinical and forensic chemical blood and urinary tests.

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