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DETERMINATION OF MORTALITY IN CASE OF ACETIC ACID POISONING

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Resume

This article presents the main types of complications of acetic essence poisoning that lead to death on the first day, as well as statistical data on the incidence of acetic acid poisoning in the Department of toxicology, literary data on the pathogenesis of acute poisoning, the development of chemical burns depending on the degree of hemolysis of erythrocytes and the main methods of treatment. Materials and methods: reports on the activities of the Department of Toxicology from 2000 to 2014, literature data Purpose: to reveal the most common causes of death of patients with acetic essence poisoning, to determine the mortality rate in recent years.

Key words

exogenous intoxication, mortality, specialized toxicological care, chemical trauma, high mortality, acetic acid, hemoglobinuria nephrosis, acute renal failure, gastrointestinal bleeding, erythrocyte hemolysis.

Relevance

According to the Department of Toxicology in 2001-2014, patients with acetic acid poisoning accounted for an average of 6% to 15% of the total number of patients with acute exogenous poisoning. This is due to the high availability and constant use of acetic acid in the household. It is worth noting that in the countries of the European Union and America, cauterizing poisons occupy an insignificant small place in the overall structure of acute poisoning: 0.4% - 0.5% of the total number of toxicological patients

Acetic acid is a colorless liquid with a characteristic pungent smell. Highly volatile compound. Soluble in water, heavier than water. Vapors are easily ignited in case of fire and form explosive mixtures. Glacial acetic acid contains 96% acid, diluted acetic acid-40-90%, table vinegar-3-8%. In Uzbekistan, a 70% solution of acetic acid is most widely used in the food industry and everyday life.

Acetic acid has a local cauterizing effect on the type of coagulation necrosis and a pronounced resorptive-hemato -, nephro-and hepatotoxic effect due to hemolysis of red blood cells, the development of toxic coagulopathy.

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The cauterizing effect is most pronounced in the area of the gastrointestinal tract and respiratory tract. The most affected areas of the digestive tract are the oral cavity, pharynx, esophagus in the thoracic region and its lower third, stomach in the region of the bottom, small curvature, cardinal and antral parts.

Necrotizing is not only the mucous membrane – the process can spread to the entire thickness of the submucosal and muscle layers. Endoscopically, there are three degrees of gastrointestinal burns:

Grade 1 - hyperemia and edema of the mucous membrane;

grade 2 – damage to the submucosal layer, necrosis up to the muscle layer;

Grade 3 - damage to all layers, this phase is characterized by early profuse bleeding and perforation of the esophagus in the early stages.

Tissue damage is caused by disruption of cell membranes as a result of the dissolution of lipids, which make up their main structural unit. The formation of acid radicals stimulates lipid peroxidation (LPO) of membranes and worsens the process of cell destruction.

Destruction of the cell membranes of the gastrointestinal mucosa and the cell membranes of the vascular wall leads to a progressive decrease in the mass of circulating blood due to the loss of its liquid part and to the development of absolute hypovolemia, which is the main link of exotic shock in this pathology in 47% of cases, the mortality rate is 70% on the first day. Sharp hyperemia of the burned mucous membrane of the stomach and intestines contributes to the rapid penetration of acetic acid into the bloodstream. The consequence of resorption is hemolysis of red blood cells. The undissociated acetic acid molecule is the main hemolytic agent, which leads to another complication - the development of hemoglobinuric nephrosis, which, with inadequate treatment, leads to the development of acute renal failure, with a mortality rate of 40%. And the third in the first day is early bleeding, the mortality rate is 67% of all cases.

It is necessary to elaborate on the mechanism of hemolysis of red blood cells. In the process of hemolysis, there are 3 stages::

The first stage is the contact of the surface of the red blood cell and hemolysin (acetic acid molecules), which suppresses the selective permeability and active transport of substances through the shell and penetrates into the cell;

The second stage is the destruction of the internal structure of the red blood cell. The low-molecular fraction leaves the red blood cell along the osmotic concentration gradient, and large protein fractions leave the red blood cell along the osmotic concentration gradient.

molecules released from ordered structures are trapped inside the cell. As a result, the contents of the cell become hypertonic in relation to the medium, and

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water begins to flow inside, the shell stretches until the mechanical resistance of the shell is overcome by osmotic pressure from the inside;

- The third stage is the rupture of the cell wall and ejection from the osmotic pressure between the red blood cell and the environment.

Transport of free hemoglobin through the renal tubules under conditions of intravascular hemolysis, microcirculation disorders and thrombosis in small vessels of the kidneys, causes damage to the basement membrane up to rupture of the distal tubules, manifested by the pathomorphological picture of acute hemoglobinuric nephrosis. According to our observations, there is a direct proportional dependence of patient mortality on the level of blood hemolysis.

Basic treatment measures for acetic acid poisoning

At the pre-hospital stage, in the emergency room, in the intensive care unit

1 Stabilization of the patient's condition

- pain relief (administration of narcotic or non-narcotic drugs)
- relief of bronchospasm (atropine)
- decongestant therapy (prednisone, diphenhydramine, eufillin, furosemide)

2 Gastric lavage through a probe lubricated with oil 8-10 liters of cold water (the presence of blood is not a contraindication for gastric lavage)

3 intensive care

- forced diuresis (6-8 liters with blood alkalinization, introduction of sodium bicarbonate 4% - 800-1000 before hemolysis is stopped, under the control of CSF)
- introduction of colloidal and crystalloid solutions 1: 3
- administration of heparin with an average severity of 5-10 thousand rubles. No. 4 per day, with severe 10-30k. under the control of a coagulogram
- hormone therapy of 60-90 mg of prednisone 2 times a day, with a severe degree of 120-150 mg for 3-4 days, then the dose is reduced to 30 mg for 15-18 days.
-antibacterial therapy
- local burn treatment (almagel, oil).
- treatment of a VDP burn using a nebulizer for 2 minutes 4 times a day with moderate severity, with severe-tracheostomy.

Conclusions: The most common causes of death of patients with acetic essence poisoning are: Exotoxic shock, early gastrointestinal bleeding, as well as the development of acute renal failure against the background of severe hemoglobinuric nephrosis at a later stage.

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