

Changes in Kidney Microstructure in Acetic Acid Poisoning

Muhammadiyah. F. R

Bukhara State Medical Institute

Summary: Characteristic features of hemoglobinuric nephrosis in accidental and suicidal poisoning with acetic acid. But the degree of damage to the kidney parenchyma is most clearly manifested if there is acute or chronic stress before poisoning.

Keywords: kidney, dystrophy, necrosis, stress, acetic acid, poisoning.

Relevance

Specific features of hemoglobinuric nephrosis in accidental and suicidal poisoning with acetic acid are revealed. But the degree of damage to the kidney parenchyma is more pronounced if it was acute or chronic before poisoning.

Stress - It is known that poisoning of various etiologies is accompanied by stress [1,5], but despite being toxic, the role of the stress factor in the pathogenesis of poisoning is very modest. Stress has its own characteristics. In cases of accidental poisoning, the body is not stressed before taking the toxic substance. In cases of suicide, poisoning also occurs in the stage of anxiety in a state of passion (demonstration of suicide) or in the background of a long and intense stress effect that causes the patient to become depressed and prompts him to commit suicide. . Many studies have established that stress, despite its protective-adaptive nature, can have a pronounced harmful effect on internal organs, being one of the links in the pathogenesis of various diseases. Stress changes of internal organs before poisoning should probably affect the clinical appearance of poisoning, its outcome and development complications.

POISONING WITH ACETIC ACID (ESSENCE).

Pathogenesis.

1. When acetic acid gets into the vascular cavity and inside the cell, it causes sub- and decompensated acidosis. The decay products of tissues as a result of chemical burns increase acidosis.
2. Resorption of acetic acid and entry into the cavity of erythrocytes increases osmotic pressure, causes fluid to enter erythrocytes, their swelling and rupture, and release of free hemoglobin into plasma.
3. Free hemoglobin blocks nephron tubules in the kidney, its acidic compounds - crystals of hematin hydrochloric acid - have a mechanical effect on the tubules. A chemical burn causes local necrosis in the nephron, destruction of the basal membrane of the tubules causes the development of nephrosis with acute hemoglobinuria.
4. As a result of tissue destruction due to burns, hemolysis of erythrocytes, thromboplastin (tissue and cell) is released into the cavity, and phase I of DVS syndrome - hypercoagulation occurs.
5. The rheological properties of the mine are disturbed.

6. Hyperkalemia develops.
7. Under the influence of intravascular hemolysis, exotoxic shock and microcirculation disorders, as well as toxic coagulopathy, there are spots of necrosis in the liver and disturbances in its main function.

Clinic. The patient smells of vinegar essence. Signs of chemical burns are detected on the skin of the face, lips, and mouth. Complaints include pain in the mouth, redness and pain in the stomach. Swallowing is impaired. A mixed return is observed. Symptoms of UNE develop when acetic acid vapors enter the upper respiratory tract. Objective tachycardia, first hyper- and then hypotonia, decrease in MVB is observed.

Many aspects of acetic acid poisoning are well-studied. The severity of acetic acid poisoning is determined by the degree of damage to internal organs, which is associated with the specific effect of acetic acid (hemoglobinuric nephrosis against the background of intravascular hemolysis of erythrocytes) and exotoxic shock. The most important morphofunctional changes are observed in parenchymal organs (lungs, liver, spleen, kidneys), whose damage mainly determines the clinical appearance and severity of the disease in the acute period of poisoning [5, 7]. It is known that suicidal types of poisoning are preceded by a stressful, traumatic situation [2, 4, 9, and 10]. Analysis of the literature showed that the effect of stress on the nature of the poisoning process has not been studied in practice [8, 9, and 10]. The different reactivity of the body associated with different stages of the stress reaction in which poisoning occurs and the duration of stress before poisoning should affect not only the clinical appearance of poisoning, but also the adequacy of the body's response to the treatment used. . All this indicates the need for further research aimed at studying the effect of stress on parenchymal organs during acute poisoning with acetic acid and searching for new effective methods of correcting this pathology with drugs [3, 6]. The purpose of the study: to determine the correlation of morphofunctional changes in the liver in acute poisoning with acetic acid at different stages of the stress reaction and to correct the identified disorders using a complex of drugs with cytoprotective, antioxidant and stress-limiting effects.

TOXICOLOGICAL SIGNIFICANCE OF ACETIC ACID.

Acetic acid is widely used in the chemical industry in the synthesis of complex ethers, indigo and other dyes, in the production of cellulose acetate, acetone, and in medicine in the synthesis of drugs such as aspirin, phenacetin, and vanillin. Acetic acid and vinegar essence are used in the food industry and for domestic consumption. Acetic acid is used a lot, so there are frequent cases of poisoning from it. Poisoning usually occurs due to drunken people drinking this acid in order to prolong the "high" or some people with the intention of killing themselves. Such accidents are especially caused by vinegar essence and concentrated acetic acid at home. It is caused by careless storage. Because young children can drink it as water without knowing it.

Concentrated acetic acid contains 96%, vinegar essence 40-80%, and food vinegar 3-8% SN3SOON. All these are dangerous for health if care is not taken. If concentrated acetic acid gets on the skin, it will burn and cause swelling and ulceration. The lethal amount of acetic acid is 2-15 g, which is equivalent to 10-20 ml of vinegar essence or 200-300 ml of table vinegar.

The permissible concentration in the air is equal to 0.005 mg/l. If concentrated acetic acid is ingested, it burns the esophageal tract, bloody vomiting, diarrhea, hemolytic anemia, hemoglobinuria, anuria, and uremia occurs. Poisoning with acetic acid is easier than poisoning with sulfuric and hydrochloric acid. When poisoned with vinegar acid vapors, it burns the respiratory tract, and there are many cases of bronchopneumonia, catarrhal bronchitis, and injuries to the digestive system. When poisoned with acetic acid, the heartbeat slows down, the body temperature rises (in some cases it reaches 39 C0), blood composition and kidney function are disturbed, the patient's mouth and respiratory tract smell of vinegar. Pathoanatomical examination of the body that died as a result of poisoning revealed necrosis in the liver and nephrosis in the kidney, in addition to the smell of acetic acid. All of these are important for

conducting a forensic chemical examination. Metabolism. In the body, acetic acid is metabolized and forms acetaldehyde, ethyl alcohol and partly SO₂. Due to its volatility, it is separated from the composition of various objects by driving it with steam. When there is a large amount of acetic acid in the object, a characteristic acetic acid smell comes from the distillate. A small amount of acetic acid is formed in the human body as a result of biochemical activity.

When extracting from the object, driving it allows to determine the free unbound acetic acid. To determine the salts formed in the body, the medium of the object is acidified by adding 10% sulfuric acid solution until it is 2.5-3.0. In both cases, in order to prevent the acetic acid from escaping, the distillate is pumped into a container containing 0.1N alkali solution until it is completely separated. The distillate is brought to a certain volume and divided into two equal parts. A portion is left for quantitative analysis. The other part is used to determine the acetate ion. For this, it is steamed in a water bath until dry.

List of references

1. Baraba V.A., Brahman I.I., Golotin V.G. and others. Oxidation and stress. - St. Petersburg, 1992. - 149 p.
2. Golikov S.N., Sanotske I.V., in TI. Common toxic mechanisms. - L., 1986. - 280 p.
3. Darovsky B.P. Cortical kidney necrosis Siberian medical journal, 2009, No. 5 Ethylene glycol poisoning // Medical business. - 1969. No. 2.-S.62-66.
4. Zimina L.n. Morphological liver changes and kidneys with ethylene glycol // Archives of pathology. -1977.-No. 2.-P.5-58.
5. Izatuln V.G., Shashkova O.N., OVOva Yu.S. and others. Features of acute poisoning against the background of emotional stress // morphological statements. - 2004. No. 1-2. - S.42.
6. Luzhnenko Yu.N., Sukhodolova G.N. etc. Urgent conditions for acute poisoning (diagnosis, clinic, treatment). - M., 2001. - 220 p.
7. Iliev Y.T., Mitrev I.N., Ansonova S.G. Psychopathology and adult self-poisoning due to psychosocial causes in Plovdiv region, Bulgaria // Folia Med (Plovdiv). - 2000. Vol. 42. No. 3. p. 30-33.
8. Kichendo M.A., Friend J.M., Halberst B., et al. COBRid major depression with subsequent stress disorder and risk for suicide // AM J Psychiatry. - 2003. - VOL. 160. No. 3.-pc50-582.
9. H.M. Prague. Stress and suicide we are equipped with Explore this problem // crisis. - 2004. - VOL. 25. No. 2. p. 80-85.
10. Smith M.T., Perlis M.L., Hayhntwaite J.A. The Meaning of Suicidal Chekik Musculoskeletal Pain Outpatients: An Interpretation of the Role of Sleep Sleep and Pain Intensity // Clinic. J pain. - 2004. - VOL. 20. - No. 2. -P.111-118.