From Clinical Manifestations To Treatment: Understanding The Pathogenesis, Diagnostic Evaluation, Prognostic Considerations, And Management Of Acetic Acid Poisoning

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ABSTRACT

Ingestion of acetic acid can lead to a severe medical crisis characterized by tissue damage, red blood cell destruction, poisoning effects, and potential organ failure. Manifestations of this poisoning encompass intense pain, bloody vomit, breathing difficulties, and shock. A diagnosis is established through a combination of patient symptoms, laboratory analysis, and endoscopic examination.

Management of acetic acid poisoning involves immediate detoxification, pain management, intravenous therapies, and ensuring the stability of essential bodily functions. Potential severe consequences include narrowing of the esophagus, kidney failure, and fatality. Preventing these incidents hinges on regulating the storage of hazardous substances and educating the public. This article will delve into the key clinical features and diagnostic markers associated with acetic acid poisoning.

Keywords: burns, hypersalivation , hemolysis, acute renal failure, strictures

Introduction. The severity of acetic acid poisoning is largely determined by the extent of internal organ damage [2]. This correlation arises from the specific pathophysiological effects of acetic acid and the resultant systemic shock response. Acetic acid is a potent corrosive that, upon absorption, causes widespread intravascular hemolysis of erythrocytes. The massive release of hemoglobin into the circulation can overwhelm the binding capacity of plasma proteins, leading to a high level of free hemoglobin in the blood. The kidneys attempt to filter this free hemoglobin, which precipitates in renal tubules and induces hemoglobinuric nephrosis – an acute toxic injury to the kidneys characterized by hemoglobin in the urine. In addition, rapid systemic absorption of the acetic acid molecule exacerbates its hemato-, nephro-, and hepatotoxic effects. Together, these processes contribute to significant internal organ injury in severe cases of poisoning. Concurrently, the development of exotoxic shock – a form of shock triggered by an exogenous toxic substance – is a major factor in life-threatening acetic acid poisoning. This shock state arises from a combination of factors: the intense pain caused by the caustic injury (severe pain syndrome), the extensive chemical burns of the gastrointestinal mucous membranes, and early-onset gastrointestinal bleeding (particularly from the stomach). These injuries lead to fluid losses, hematologic disturbances, and intense sympathetic nervous

system activation, collectively precipitating circulatory collapse if not promptly managed.

The corrosive effect of the acid primarily damages the mucous membranes of the oral cavity, pharynx, and esophagus, leading to pain and superficial inflammation at these sites. Liver and kidney function show only minimal deviations from normal in mild poisoning, reflecting the relatively limited absorption of acid and lesser systemic impact. The inflammatory changes observed in the affected mucosa are typically catarrhal-fibrinous in nature, meaning the inflammation is largely superficial with mucous exudation and a thin fibrinous coating on the injured mucosal surfaces. Notably, patients with mild acetic acid poisoning do not develop shock, since the degree of tissue injury and pain, while significant, is not enough to trigger the profound hemodynamic compromise seen in more severe poisonings. It should also be noted that inhalation of acetic acid vapors – which can occur during exposure to concentrated acetic acid – predominantly causes irritation of the upper respiratory tract. Such vapor exposure leads to pronounced edema and hyperemia of the mucous membranes lining the upper airway, manifesting as swelling and redness of the nasal passages, oropharynx, and larynx. These respiratory effects are generally localized and resolve with removal from exposure, and they typically accompany mild exposure scenarios where systemic involvement is minimal.

In case of mild poisoning, as determined by Luzhnikov E.A. and Kostomarova L.G., the concentration of free hemoglobin in the blood does not exceed 5 g/l (500 mg%). The disease manifests itself mainly by damage to the mucous membranes of the oral cavity, esophagus and pharynx, with minimal deviations in the functioning of the liver and kidneys. The inflammatory process is catarrhal-fibrinous in nature, without the development of shock. Inhalation of vapors of the substance causes edema and hyperemia of the mucous membranes of the upper respiratory tract.

For moderate poisoning, there are esophageal and al burns, injuries to the mouth, pharynx, stomach. Hemolysis is 5-10 g / l. Nephro and hepatopathy of moderate degree. The inflammatory nature is catarrhal-serous. It is caused by the phenomena of exotoxic shock. Inhalation penetration leads to the formation of significant edema and bronchospasm. Small necrotic foci are formed on the mucous membrane.

Severe degree of concentration of hemolyzed hemoglobin>10 g/liter. Manifested by liver and kidney failure. Burns extend to deep parts of the digestive tract. Tissue changes of ulcerative-necrotic type. Severe exotoxic shock . Inhalation of acid is accompanied by irritation of the nasopharynx, trachea, bronchi, lungs. [1]. In foreign sources, there are no cases of patients surviving after severe poisoning with acetic acid, when the concentration of free hemoglobin in the blood exceeded 10 g/l.

Acetic acid has the ability to cause local coagulation necrosis and have a pronounced resorptive effect on hematological, renal and hepatic functions. These effects are due to hemolysis of erythrocytes, the development of toxic coagulopathy , and also the syndrome of disseminated intravascular coagulation [3-16]. Tissue destruction, known as tissue lysis, occurs due to disruption of the integrity of cell membranes. This process is caused by the dissolution of lipids, which are a key component of these membranes. The formation of reactive acid radicals initiates a chain reaction of lipid peroxidation, which accelerates cell destruction. In the gastrointestinal tract, the oral cavity, pharynx, esophagus, as well as the fundus, antrum , cardiac regions of the stomach, and the lesser curvature are most at risk. Necrosis affects not only the superficial layer of the mucosa, but also deeper tissues, including the submucosa and muscular layer [10, 11]. Destruction of cell membranes in the gastrointestinal tract lining and vessels causes plasma leakage, which reduces the total circulating blood volume and provokes hypovolemia . Such blood loss is an invariable factor in the development of exotoxic shock in poisoning [7,9].

Acute hyperemia of the damaged mucous membrane of the stomach and intestines promotes rapid absorption and penetration of acetic acid into the bloodstream. The acid causes serious disturbances of the acid-base balance

with subsequent subcompensated metabolic acidosis. These disturbances are mostly associated with endogenous factors - underoxidized metabolic products that form during chemical damage to the gastrointestinal tract and its various complications. Acidosis is caused by several mechanisms:

- direct entry of acetic acid into the blood leads to an increase in the concentration of hydrogen ions (H^+), which reduces the pH of the blood.
- loss of bicarbonates (HCO_3^-), necessary to maintain acid-base balance, increases acidosis.
- lactic acidosis develops as a result of hypoxia and activation of anaerobic glycolysis, which leads to the accumulation of lactic acid (lactate) in the blood.

The result of absorption is hemolysis of erythrocytes. The undissociated acetic acid molecule plays a major role in the hemolytic process [4 - 6]. In the presence of acetic acid, hemoglobin disintegrates into globin and heme , and is oxidized to hemin. Hemoglobin, especially hemin compounds, accelerate the decomposition of hydroperoxides with the formation of free radicals capable of activating new chain oxidation reactions [11].

Hemolysis of erythrocytes plays an important role in the onset of toxic coagulopathy syndrome . Tissue breakdown by burns and erythrocyte breakdown lead to the release of a large amount of thromboplastic material and the onset of the first stage of toxic coagulopathy - the hypercoagulation stage [4].

Transport of free hemoglobin through the renal tubules under conditions of intravascular hemolysis, impaired microcirculation and thrombus formation in small renal vessels leads to damage to the basement membrane, sometimes to rupture of the distal tubules, which is reflected in the pathomorphological signs of acute hemoglobinuric nephrosis [4 - 6]. Observations indicate a direct proportional dependence of the level of blood hemolysis on patient mortality. The impact of two main pathological processes - intravascular hemolysis and exotoxic shock with severe microcirculation disorders, as well as toxic coagulopathy - leads to liver damage in the form of focal necrosis (infarctions) with disruption of its basic functions [4]. Exotoxic shock is the main cause of death in acetic acid poisoning in the first 1-3 days [3, 9].

The development of central hemodynamic disorders and exotoxic shock is caused by a number of factors, primarily absolute hypovolemia caused by plasma loss due to burns and bleeding. [7, 9]. In cases of acetic acid poisoning, one of the important causes of exotoxic shock may be acute transcapillary metabolism disorders associated with the release of cytokines and damage to the vascular epithelium, on the one hand, and Starling equilibrium disorders, on the other [9].

According to the latest data, patients with acetic acid poisoning may develop acute lung injury syndrome, characterized by alveolar hypoxia, arterial and venous hypoxemia, intrapulmonary shunting of blood and impaired oxygen transport function of the blood, which leads to a decrease in oxygen flow and an increase in its utilization at the cellular level. The degree of respiratory dysfunction is proportional to the severity of intoxication, expressed by the level of free hemoglobin in the blood plasma [8, 9].

Thus, contact with acetic acid causes a chemical burn disease caused by the local destructive effect on tissues and the resorptive effect as a hemolytic agent. At present, the main classification of acute acetic acid poisoning remains the system proposed by E.A. Luzhnikov and Yu.S. Goldfarb in 1989, according to which 3 degrees of poisoning severity are distinguished [3, 4]:

Mild poisoning, in which the burn is limited to the mucous membrane of the mouth, pharynx, esophagus and manifests itself as catarrhal-serous inflammation. In this case, mild nephropathy and minor liver dysfunction are observed.

Moderate poisoning, in which the burn affects the mucous membrane of the mouth, pharynx, esophagus and stomach and is manifested by catarrhal-serous or catarrhal-fibrinous inflammation. In this case, exotoxic shock (in the compensated phase), hemolysis, hemoglobinuria at a level of 5-10 g / l, as well as moderate toxic nephropathy and mild or moderate toxic liver dystrophy are observed.

Severe poisoning, in which the burn covers the esophagus, stomach, small intestine and is

manifested by ulcerative-necrotic inflammation. In this case, burns of the upper respiratory tract, exotoxic shock, hemolysis, hemoglobinemia over 10 g/l, acute hemoglobinuric nephrosis and severe toxic nephropathy, as well as early and late complications (endotoxicosis) are also observed [3,4].

Diagnosis of acute acetic acid poisoning includes clinical, laboratory and instrumental methods at various stages of poisoning.

acid poisoning is based on the following clinical symptoms:

- The appearance of pain, mainly in the upper part of the neck, with a description of a burning, stabbing nature. The pain intensifies when swallowing. There is also a sharp pain in the esophagus and epigastric region. Vomiting with the smell of vinegar, in severe poisoning, bleeding is possible.
- Bleeding.
- Drooling.
- A burning sensation in the throat.
- Difficulty swallowing .
- Taste disturbance.
- Cough and difficulty breathing due to burns of the throat and respiratory tract.

Conclusion.

Acetic acid poisoning is a serious medical condition that requires a comprehensive and careful approach to treatment. The study examined various aspects of the clinical picture, pathophysiology, and diagnostic methods for investigating this type of poisoning.

It has been established that acute poisoning with acetic acid is accompanied by a variety of clinical manifestations, ranging from pain in the epigastric region, vomiting, anorexia, hyperemia of the oral mucosa and ending with multiple organ failure.

During the analysis of existing diagnostic methods and clinical manifestations of acetic acid poisoning, it was found that early detection and adequate medical intervention significantly affect the outcome of the disease. The effectiveness of infusion therapy using drugs such as reamberin , succinasol and cytoflavin allows neutralizing the toxic effects of acetic acid and restoring biochemical processes in the body . In case of massive blood loss, the use of fresh frozen plasma and red blood cell mass allowed restoring the BCC and preventing the development of circulatory and hemic hypoxia. In case of renal infarction, the use of hemodialysis solved the problem of acute renal failure.

An important aspect of treating acetic acid poisoning is also the issue of the patient's nutrition. An individually selected diet, including complete proteins and essential vitamins, helps restore the body and reduce complications.

Thus, the optimal intensive care strategy for acetic acid poisoning includes early diagnosis, adequate infusion therapy, balanced nutrition, and comprehensive patient monitoring. Attentive medical care and timely intervention contribute to a significant improvement in prognosis and a decrease in mortality among patients with this pathology.

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