
# Clinical Characteristics and Diagnostic Criteria for Acetic Acid Poisoning

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**ABSTRACT**

In recent times, incidents of acetic acid poisoning have witnessed a surge, primarily attributed to its easy accessibility. Suicide attempts, wherein acetic acid is frequently employed, constitute a significant factor. Additionally, the issue of alcohol dependence and misuse has become a pressing concern within the population. Government initiatives are directed towards addressing this problem by exerting pressure on producers to raise the prices of alcoholic beverages. Owing to the grip of alcohol addiction, individuals seek alternatives, often resorting to the use of acetic acid. This article will delve into the clinical features and diagnostic criteria associated with acetic acid poisoning.

**Keywords:** acetic acid, hemolysis, acute renal failure, strictures, endoscopy.

**Introduction.** The extent of acetic acid poisoning primarily hinges on the harm inflicted on internal organs [2]. This is attributed to the specific impact of acetic acid, resulting in hemoglobinuric nephrosis alongside intravascular hemolysis of erythrocytes and active absorption of the acetic acid molecule. It also leads to the onset of exotoxic shock, brought about by factors like pain, widespread chemical burns on the mucous membranes of the gastrointestinal tract, and early gastric bleeding.

As per Luzhnikov E.A. and Kostomarova L.G.'s classification, in cases of mild poisoning, the level of free hemoglobin in the blood remains below 5 g/l (equivalent to 500 mg%) and the general symptoms involve damage to the oral cavity, esophagus, and pharynx. There are minor alterations in the liver and kidneys, characterized by an inflammatory nature of catarrhal-fibrinous. Shock is not observed, and inhalation of vapors leads to swelling and redness of the upper respiratory tract's mucous membranes.

In instances of moderate severity of poisoning, there are burns in the esophagus and oral region, causing injuries to the mouth, pharynx, and stomach. Hemolysis increases to 5-10 g/l. There is a presence of nephro and moderate hepatopathy, with an inflammatory nature described as catarrhal-serous. This is instigated by the effects of exotoxic shock. Inhalation of the acid results in significant edema and bronchospasm, with small necrotic foci forming on the mucous membrane.

In cases of severe poisoning, hemolysis leads to a concentration of hemoglobin exceeding 10 g/liter. This manifests as liver and kidney failure, with burns extending to the deeper regions of the digestive tract. Tissue changes exhibit an ulcerative-necrotic nature, accompanied by severe exotoxic shock. Inhalation of the acid causes irritation of the nasopharynx, trachea, bronchi, and lungs [1].

According to foreign sources, there have been no reported instances of patient survival after severe poisoning with acetic acid when the concentration of free hemoglobin in the blood exceeded 10 g/l.

Acetic acid possesses the capacity to induce localized coagulative necrosis and exerts a marked resorptive impact on hematological, renal, and hepatic functions. These effects are a result of erythrocyte hemolysis, leading to the development of toxic coagulopathy, as well as disseminated intravascular coagulation syndrome [3-8].

Tissue breakdown occurs due to the breakdown of cell membranes brought about by lipid dissolution, which forms the fundamental structural component of these tissues. The formation of acid radicals amplifies lipid peroxidation within membranes, intensifying destructive processes within cells. Among the regions of the gastrointestinal tract, the oral cavity, pharynx, esophagus, fundus, antrum, and cardiac sections of the stomach, along with the lesser curvature, are most profoundly affected. Necrosis impacts not only the mucosa but also the submucosa and muscle layer [10, 11]. The destruction of the gastrointestinal mucosa membrane and vascular wall leads to a reduction in blood volume due to plasmorrhagia, resulting in hypovolemia. This stands as a consistent component of exotoxic shock during poisoning [7, 9].

The damaged mucous membrane of the stomach and intestines undergoes acute hyperemia, facilitating the rapid absorption and entry of acetic acid into the bloodstream. This acid induces significant disruptions in the amino acid hormone receptors, leading to subcompensated metabolic acidosis. Many of these disturbances are linked to endogenous factors, specifically under-oxidized metabolic byproducts formed during chemical damage to the gastrointestinal tract and its ensuing complications.

The absorption process results in the hemolysis of red blood cells, with the undissociated acetic acid molecule playing a pivotal role in this hemolytic process [4-6]. In the presence of acetic acid, hemoglobin breaks down into globin and heme, subsequently oxidizing to hemin. Particularly heme compounds within hemoglobin accelerate the breakdown of hydroperoxides, generating free radicals that can trigger new oxidation chain reactions [11].

Red blood cell hemolysis plays a crucial role in the initiation of toxic coagulopathy syndrome. The tissue breakdown from burns, along with the rupture of red blood cells, leads to the release of a substantial amount of thromboplastic material, marking the commencement of the hypercoagulation stage in the first phase of toxic coagulopathy [4].

In the kidneys, damage to the basement membrane may extend to the point of distal tubule rupture, reflected in the pathomorphological signs of acute hemoglobinuric nephrosis [4-6]. Observations indicate a direct correlation between the level of blood hemolysis and patient mortality. The impact of the two main pathological processes - intravascular hemolysis and exotoxic shock with severe microcirculation disorders, as well as toxic coagulopathy - culminates in liver damage, characterized by focal necrosis (infarction) and a disruption of its core functions [4]. Exotoxic shock stands as the primary cause of death within the first 1-3 days following acetic acid poisoning [3, 9].

The onset of central hemodynamic disorders and exotoxic shock is attributed to various factors, primarily absolute hypovolemia resulting from plasma loss due to burns and bleeding [7, 9]. In cases of acetic acid poisoning, one significant factor contributing to exotoxic shock may be acute disruptions in transcapillary metabolism linked to the release of cytokines and damage to vascular epithelium, alongside disturbances in Starling equilibrium [9].

Recent data indicates that patients experiencing acetic acid poisoning may develop acute lung injury syndrome, characterized by alveolar hypoxia, arterial and venous hypoxemia, intrapulmonary blood shunting, and impaired oxygen transport function of the blood. This leads to a reduction in oxygen flow and an increase in its cellular utilization. The severity of respiratory dysfunction corresponds directly to the degree of intoxication, as

indicated by the level of free hemoglobin in the blood plasma [8, 9].

Therefore, contact with acetic acid results in a chemically induced burn disease, characterized by a local destructive impact on tissue and a resorptive effect as a hemolytic agent. The primary classification for acute acetic acid poisoning remains the system proposed by E.A. Luzhnikov and Yu.S. Goldfarb in 1989, which delineates three degrees of severity [3, 4]:

Mild poisoning, characterized by limited burns to the mucous membrane of the mouth, pharynx, and esophagus, with manifestations of catarrhal-serous inflammation. Mild nephropathy and minor liver dysfunction may also be observed.

Moderate poisoning, in which burns extend to the mucous membrane of the mouth, pharynx, esophagus, and stomach, presenting with catarrhal-serous or catarrhal-fibrinous inflammation. This phase may involve exotoxic shock (in the compensated phase), hemolysis, hemoglobinuria at a level of 5-10 g/l, as well as moderate toxic nephropathy and mild to moderate toxic liver dystrophy.

Severe poisoning, where burns encompass the esophagus, stomach, and small intestine, with manifestations of ulcerative necrotic inflammation. This stage includes burns of the upper respiratory tract, exotoxic shock, hemolysis, hemoglobinemia exceeding 10 g/l, acute hemoglobinuric nephrosis, severe toxic nephropathy, and early and late complications (endotoxicosis) [3, 4].

The diagnosis of acute acetic acid poisoning encompasses clinical evaluations, laboratory analyses, and instrumental assessments at various stages of poisoning.

Clinical Symptoms of Acid Poisoning Include:

- Onset of pain, primarily localized in the upper neck region, described as burning and stabbing in nature. Pain exacerbates during swallowing. Sharp pain is also experienced in the esophagus and epigastric region. Vomit may have a vinegar-like odor; severe cases may lead to bleeding.
- Bleeding.
- Excessive drooling.
- Sensation of throat burning.
- Difficulty in swallowing.
- Alteration in taste perception.
- Coughing and breathing difficulties due to burns in the pharynx and respiratory tract.

*A) Laboratory Studies:*

Assessment of free hemoglobin levels in both blood and urine, along with monitoring hemoglobin levels upon admission and over time.

Determination of total protein, ALT, AST, bilirubin, urea, and creatinine levels in the blood to monitor liver and kidney function.

Evaluation of the acid-base balance (ABG), a crucial study for gauging the severity of metabolic acidosis in the early stages of poisoning.

Coagulogram, encompassing prothrombin index, fibrinogen levels, hematocrit, and plasma recalcification time.

Complete blood count with a detailed leukoformula and leukocyte intoxication index. These laboratory tests assist in evaluating the extent of poisoning and predicting potential complications.

*B) Instrumental Investigations:*

Esophagogastroduodenoscopy (EGDFS) is a pivotal method for diagnosing chemical burns of the gastrointestinal tract.

Ultrasound of the abdominal cavity and kidneys can reveal signs of toxic hepatitis and nephritis, such as diffuse thickening of the liver, increased echogenicity of the kidneys, and pallor of the kidney parenchyma.

Electrocardiography (ECG) is essential for analyzing cardiac function.

These instrumental diagnostic methods provide a more precise assessment of the patient's condition and facilitate the selection of optimal treatment strategies.

**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 studying 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.

In the course of analyzing 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 makes it possible to neutralize the toxic effects of acetic acid and restore biochemical processes in the body.

An important aspect of the treatment of acetic acid poisoning is also the question 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 tactics of intensive care for acetic acid poisoning include early diagnosis, adequate infusion therapy, balanced nutrition and comprehensive monitoring of the patient. Attentive medical care and timely intervention contribute to a significant improvement in prognosis and reduction in mortality among patients with this pathology.

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