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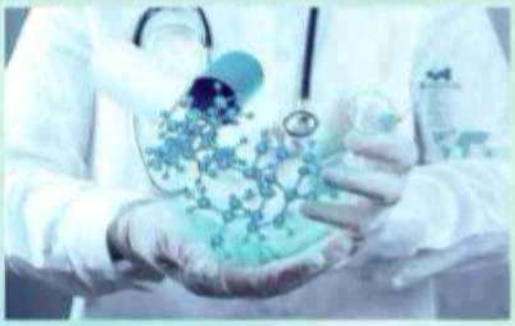

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ТИББИЁТДА ЯНГИ КУН

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www.bsmi.uz
https://newdaymedicine.com E:
ndmuz@mail.ru
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ACETIC ACID POISONING: CLINICAL CHARACTERISTICS, DIAGNOSIS, PATHOGENESIS, PROGNOSIS AND TREATMENT METHODS

Babanazarov Umid Turobkulovich https://orcid.org/0009-0000-3160-6273

Bukhara State Medical Institute named after Abu Ali ibn Sina, Uzbekistan, Bukhara, st. A. Navoi. 1 Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Resume***

Acetic acid poisoning is a dangerous condition that causes mucosal burns, hemolysis, intoxication, and multiple organ failure. Symptoms include severe pain, vomiting of blood, respiratory failure, and shock. Diagnosis is based on clinical presentation, laboratory tests, and endoscopy.

Treatment includes emergency detoxification, pain relief, infusion therapy and maintenance of vital functions. Possible complications are esophageal strictures, renal failure and death. Prevention comes down to controlling the storage of aggressive substances and raising public awareness. In this article, we will consider the9 main aspects of clinical characteristics and diagnostic criteria for acetic acid poisoning

Key words: burns, hypersalivation , hemolysis, acute renal failure, strictures

ОТРАВЛЕНИЕ УКСУСНОЙ КИСЛОТОЙ: КЛИНИЧЕСКАЯ ХАРАКТЕРИСТИКА, ДИАГНОСТИКА, ПАТОГЕНЕЗ, ПРОГНОЗ И МЕТОДЫ ЛЕЧЕНИЯ

Бабаназаров Умид Туробкулович

Бухарский государственный медицинский институт имени Абу Али ибн Сины, Узбекистан, г. Бухара, ул. А. Навои. 1 Тел: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Резюме***

Отравление уксусной кислотой – это опасное состояние, вызывающее ожоги слизистых, гемолиз, интоксикацию и полиорганную недостаточность. Симптомы включают сильную боль, рвоту с кровью, дыхательную недостаточность и шок. Диагностика основана на клинической картине, лабораторных анализах и эндоскопии.

Лечение включает экстренную детоксикацию, обезболивание, инфузионную терапию и поддержание жизненно важных функций. Возможные осложнения – стриктуры пищевода, почечная недостаточность и летальный исход. Профилактика сводится к контролю хранения агрессивных веществ и повышению осведомленности населения. В данной статье мы рассмотрим основные аспекты клиническую характеристику и диагностические критерии отравления уксусной кислотой

Ключевые слова: ожоги, гиперсаливация, гемолиз, острая почечная недостаточность, стриктуры

SIRKA KISLOTASI BILAN ZAHARLANISH: KLINIK XUSUSIYATLARI, PATOGENEZ, PROGNOZ VA DAVOLASH USULLARI

Babanazarov Umid Turobkulovich

Abu Ali ibn Sino nomidagi Buxoro davlat tibbiyot instituti, O‘zbekiston, Buxoro sh. A. Navoiy kochasi 1 Tel: +998 (65) 223-00-50 e-mail: info@bsmi.uz

✓ ***Rezyume***

Sirka kislotasi bilan zaharlanish shilliq qavatlarning kuyishi, gemoliz, intoksikatsiya va ko‘p a’zolar yetishmovchiligiga olib keladigan xavfli holatdir. Alomatlar orasida kuchli og'riq, qon qusish, nafas olish etishmovchiligi va shok mavjud. Tashxis klinik ko'rinishga, laboratoriya tekshiruvlariga va endoskopiyaga asoslangan.

Davolash favqulodda detoksifikatsiya, og'riqni yo'qotish, suyuqlik terapiyasi va hayotiy funktsiyalarni qo'llab-quvvatlashni o'z ichiga oladi. Mumkin bo'lgan asoratlar qizilo'ngachning strikturasi, buyrak etishmovchiligi va o'limni o'z ichiga oladi. Profilaktika agressiv moddalarning saqlanishini nazorat qilish va aholining xabardorligini oshirishdan iborat. Ushbu maqolada sirka kislotasi bilan zaharlanishning klinik xususiyatlari va diagnostika mezonlarining asosiy jihatlarini ko'rib chiqamiz

Kalit so'zlar: kuyishlar, gipersalivatsiya, gemoliz, o'tkir buyrak etishmovchiligi, strikturalar

Relevance

The degree of acetic acid poisoning depends largely on damage to internal organs [2]. This is due to the specific effects of acetic acid (manifestation of hemoglobinuric nephrosis against the background of intravascular hemolysis of erythrocytes and active resorption of the acetic acid molecule) and the development of exotoxic shock (because of pain syndrome, widespread chemical burns of the mucous membranes of the gastrointestinal tract and early bleeding from the stomach).

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].

Materials and methods

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].

Results and discussions

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.

A) Laboratory tests:

1. Study of the content of free hemoglobin in the blood and urine, as well as the hemoglobin level upon admission and over time.
2. Determination of the level of total protein, ALT, AST, bilirubin, urea and creatinine in the blood in order to monitor liver and kidney function.
3. Determination of the acid-base balance (ABB) is an important study for assessing the severity of metabolic acidosis in the early stages of poisoning.
4. Coagulogram , including prothrombin index, fibrinogen level, hematocrit and plasma recalcification time .

These laboratory tests help assess the severity of poisoning and predict possible complications.

B) Instrumental studies:

- Esophagogastroduodenoscopy (EGDFS) is an important method for diagnosing chemical burns of the gastrointestinal tract .
- Ultrasound of the abdominal organs and kidneys allows us to identify signs of toxic hepatitis and nephritis, such as diffuse liver compaction, increased echogenicity of the kidneys and pallor of the renal parenchyma.
- ECG is necessary for the analysis of cardiac function .

These instrumental diagnostic methods help to more accurately assess the patient's condition and choose the optimal treatment.

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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