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Article

Acute Methanol Poisoning

Khusenov O.N [1]

Bukhara Medical Institute
*Correspondence: olim_xusenov@bsmi.uz

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Abstract: General Background: Acute methanol poisoning remains a critical public health issue due to its high lethality and severe neurological consequences. Methanol, a widely used industrial solvent and component of various commercial products, poses significant risks when ingested, inhaled, or absorbed through the skin. Specific Background: The toxic effects of methanol arise primarily from its metabolism into formaldehyde and formic acid, leading to metabolic acidosis, optic nerve damage, and multi-organ failure. Epidemiological data indicate that methanol poisoning contributes significantly to alcohol-related fatalities, with high mortality rates reported globally. Knowledge Gap: Despite advances in toxicological research, early diagnosis and effective therapeutic interventions for methanol poisoning remain challenging. The need for rapid, accessible diagnostic methods and optimized treatment protocols persists. Aims: This review synthesizes contemporary findings on the toxicokinetics, toxicodynamics, clinical manifestations, diagnostic methodologies, and treatment strategies for acute methanol poisoning. Results: The review highlights the importance of prompt recognition and intervention, emphasizing antidotal therapy with ethanol or fomepizole, hemodialysis for toxin elimination, and metabolic acidosis management. Diagnostic challenges, including the differentiation of methanol from ethanol in illicit alcohol products, are discussed. Novelty: This study consolidates recent epidemiological and clinical data, underscoring the critical role of formic acid in methanol toxicity and the potential for novel diagnostic biomarkers. Implications: Improved surveillance, stringent regulatory measures, and enhanced public awareness are essential to reducing methanol poisoning incidents and fatalities. Future research should focus on refining rapid diagnostic tools and optimizing treatment algorithms to improve clinical outcomes.

Keywords: Methanol poisoning, acute toxicity, metabolic acidosis, formic acid, optic nerve damage, hemodialysis, ethanol antidote, fomepizole, toxicokinetics, toxicodynamics

Introduction

Acute poisoning of chemical etiology as a cause of health disorders and death is an urgent medical and social problem today [1,2]. Among accidental chemical poisoning with a fatal outcome, poisoning with alcohol-containing agents occupies a leading place [2]. The share of deaths among the able-bodied population of the Russian Federation associated with the use of alcohol and its surrogates

in different years ranged from 43 to 50% [3]. The high lethality of alcohol poisoned by surrogates is mostly due to the toxic effect of methanol [4,5].

Methanol, also known as methyl alcohol or carbinol, is a flammable, colorless liquid. Methanol poisoning is an extremely dangerous condition that entails serious complications and often, without timely assistance, leads to death [6,7,8].

The field of application of methanol is quite wide. It is used in the production of resins, dyes and intermediates, and is used for the synthesis of some organic compounds. In addition, methanol is an ingredient in various commercial products, including windshield washer fluids, and is also used as an industrial solvent [1,9].

Most often, poisoning occurs when methanol is ingested orally, although percutaneous and inhaled cases of poisoning are also known [1,8,10]. Methyl alcohol is used, in most cases, as part of various non-food liquids, as well as adulterated alcoholic beverages manufactured with the use of methanol in order to save money. Ethanol and methanol have similar organoleptic properties, so it is not possible to distinguish them in domestic conditions [10,11].

Epidemiology. According to the state report "On the state of sanitary and epidemiological well-being of the population in the Russian Federation in 2021", 470,358 cases of acute poisoning with alcohol-containing products were registered in the Russian Federation for the period from 2012 to 2021, 124,813 of them were fatal (26.5%). The rate of acute poisoning with alcohol-containing products in 2021 amounted to 21.19 cases per 100 thousand population [12].

The main alcohol-containing substances that caused acute poisoning in the period from 2012 to 2021 include: ethanol (77.4%), unspecified alcohol (16.7%), other alcohols (3.3%), methanol (2.2%), 2-propanol (0.3%), fusel oil (0.1%). Fatal poisonings during the specified period were caused by: ethanol (85.6%), methanol (6.9%), unspecified alcohol (5.4%), other alcohols (1.3%), 2-propanol (0.7%), fusel oil (0.2%) [12].

Materials and Methods

Physicochemical properties. Methanol (CH_3OH) is one of the simplest monohydric alcohols. It is a colorless transparent liquid with a characteristic

"alcoholic" smell [9,13]. The molecular weight of methyl alcohol is 32.04, the specific gravity is 0.792 [10]. The melting point is 97°C, the boiling point is + 65°C. Methanol belongs to the chemical substances of hazard class III [5].

Toxicokinetics. The toxic effect of methanol is manifested when it is taken in the amount of 7-8 ml. The lethal dose ranges from 30 to 100 ml [1,10,11], although there are cases of death after taking 5 ml and recovery after consuming 250-500 ml [14-16]. The toxic concentration of methanol in the blood is from 0.2 to 0.3 mg/ml, lethal from 0.8 to 1 mg/ml [1,9,17].

When taken orally, methyl alcohol is absorbed quite quickly and reaches its maximum concentration in the blood within 30-60 minutes [17,18]. Part of the methanol is secreted by the mucous membrane into the lumen of the stomach within a few days and is reabsorbed [17,19].

Methanol is metabolized mainly in the liver, where it is oxidized to formaldehyde with the participation of NAD-dependent alcohol dehydrogenase [7,20]. Some formaldehyde binds to blood proteins, but most of it is rapidly oxidized by aldehyde dehydrogenase to formic acid, which is the main toxic metabolite of methyl alcohol [11,19,21]. Formic acid is further either oxidized by the enzyme formyltetrahydrofolate dehydrogenase to form carbon dioxide and water, or eliminated unchanged [4,5,22].

The half-life of methyl alcohol is 14-30 hours, and complete biotransformation and elimination of methanol takes 5-8 days [5]. Up to 10% of methanol ingested in the body is excreted in the urine, from 5 to 70% is excreted through the lungs, small amounts are excreted with sweat. The remaining methanol is distributed among organs and tissues. Most of it is deposited in the liver and kidneys, less in fat, muscles, and brain [5,14,19].

Toxicodynamics. Methanol and its metabolites are strong protoplasmic poisons [19]. They inhibit oxidative phosphorylation in the cytochrome oxidase system, causing adenosine triphosphate (ATP) deficiency, to which the retina and brain tissues are most susceptible.

In addition, the action of methanol and formic acid on another enzyme of the respiratory chain, cytochrome C-reductase, also contributes to ATP deficiency [23]. As a result, tissue hypoxia develops, and lactate accumulates. This effect, combined with formic acid itself, causes systemic metabolic acidosis with a large anionic interval [7,20].

Results

Clinical picture. In the clinical course of methanol poisoning, 4 periods are distinguished: initial, latent (the period of imaginary well-being), the period of pronounced clinical manifestations, and the period of consequences [24].

The initial period, which is based on the narcotic effect of methanol on the central nervous system, resembles ordinary alcoholic intoxication [17]. Characteristically, the degree of intoxication is usually less than could be expected from taking similar doses of ethyl alcohol. The duration of this period can range from 1-2 to 12 or more hours [9].

Intoxication is followed by a latent period, which proceeds without pronounced clinical manifestations. Its duration is individual and varies from 6 to 44 hours [9,24].

The period of pronounced clinical manifestations begins with the appearance of symptoms of toxic gastritis (nausea, vomiting, pain in the epigastric region, tension of the muscles of the abdominal wall) and the occurrence of visual disturbances (flickering "flies", white veil in front of the eyes, blurred vision, double vision, mydriasis, sluggish reaction of the pupils to light or its absence, blindness). Symptoms of general intoxication develop: malaise, headache, dizziness, muscle weakness. Symptoms of toxic encephalopathy appear and increase, accompanied by psychomotor agitation, deafness and confusion, up to a comatose state. Among metabolic disorders, decompensated metabolic acidosis is the leading one. At a later date, complications of the liver, kidneys, and heart appear [9,16,24].

According to the severity of poisoning, there are mild (general disorders syndrome, toxic gastritis), moderate (ophthalmic) and severe (generalized) forms [5,24].

In mild poisoning in the period of pronounced clinical manifestations, dizziness, headache and abdominal pain, nausea, vomiting, visual disturbances (blurred vision, darkening of the eyes, flickering of "flies") are observed. [5]. An objective examination reveals a moderate dilation of the pupils and a weakening of the photoreaction [24]. The fundus retains its normal appearance [10]. The duration of disorders most often does not exceed 3-5 days. In some cases, victims develop a long narcotic sleep, after which only mild malaise is noted [11]. This degree of poisoning usually ends with recovery with complete restoration of vision [5,10,11].

Poisoning of moderate severity is characterized by gradually increasing severe visual impairment, up to complete blindness with atrophy of the optic nerves. Cases have been described when victims who consumed methanol woke up blind the next day, but on the 3rd-4th day their vision was restored to normal. However, in such cases, the improvement of vision is not always stable and during the following days recurrence of vision deterioration and blindness are possible. Examination of the fundus reveals hyperemia and blurring of the borders of the optic nerve nipples, dilated veins, and hemorrhages [5,10,24].

In severe poisoning, the latent period is short or absent, clinical symptoms quickly appear: nausea, vomiting, weakness, pain in the epigastrium, lower back and calf muscles, mydriasis, rapid visual impairment. The victim loses consciousness, breathing is quickly disturbed, cyanosis increases, cardiovascular failure progresses up to exotoxic shock. In some cases, the development of acute excitation and clonic convulsions is possible [10,24]. With an unfavorable course, on the 1st-2nd day, due to central respiratory and circulatory disorders, a fatal outcome occurs [21,24]. With a favorable course, there is a preservation of visual impairment due to irreversible atrophy of the optic nerve. In

the future, asthenization may continue, often in combination with signs of organic brain damage. Complications are often added: pneumonia, myocardial dystrophy, moderate hepatic and renal failure [5,10,24].

Diagnostics. Diagnosis of acute methanol poisoning includes analysis of anamnesis and physical examination data, instrumental and laboratory studies [5,24].

The collection of anamnesis is aimed at establishing the fact of the patient's use of methanol, clarifying the dose, exposure, and the route of entry of the toxicant into the body. If possible, you should find out what alcohol-containing liquid caused the poisoning, where it was purchased, for what purpose it was taken (intoxication, suicide attempt), how much time has passed since the moment of use. It should be borne in mind that the history data is not always informative: the patient may not know what he was drinking, or deliberately hide the fact of use [5,23,24]. Physical examination data correspond to the severity of poisoning and are not specific [5].

Laboratory diagnosis of poisoning includes the determination of the content of formic acid in the blood, the study of electrolytes and blood gases, repeated determinations of osmolarity and acid-base state of plasma [5,22,23]. An increase in the anion interval is an important criterion, but cannot contribute to the early diagnosis of methanol poisoning, since metabolic acidosis appears only after a certain time has elapsed since the use of the toxicant. Unlike the anionic interval, the osmolar interval increases immediately after ingestion of alcohol, but it is not specific and indicates the presence of any osmotically active agent. There is an inverse relationship between the osmolar and anionic intervals. The osmolar interval gradually decreases as the anionic interval develops [7,16].

Serum gas chromatography is the most reliable method for diagnosing methyl alcohol poisoning, but due to its high cost and labor intensity, it is not available in all medical institutions [5,7,16].

Ophthalmoscopy and computed tomography are used as instrumental research methods. Ophthalmoscopy reveals vasodilation, hemorrhages, retinal edema, as well as hyperemia, edema and signs of optic nerve atrophy. Computed tomography of the brain can reveal optic nerve damage and necrotic changes in the striopallidary system [5].

Therapy. Treatment of methyl alcohol poisoning includes the administration of an antidote, accelerated detoxification therapy, correction of metabolic acidosis, and general supportive measures (oxygen therapy, mechanical ventilation if indicated, infusion therapy, etc.) [14,17].

Accelerated detoxification methods include gastric lavage, forced diuresis, and hemodialysis. Gastric lavage is effective only in the early stages after methanol ingestion, due to the high rate of absorption of the poison [17,20]. The most effective method of removing methanol and its metabolites from the bloodstream is hemodialysis. Indications for hemodialysis are: oral intake of more than 30 ml of methanol, the level of methanol in the blood from 0.2 to 0.5 g/l, the development of visual disorders, decompensated metabolic acidosis. If hemodialysis is performed concomitantly with ethanol antidote therapy, the dose of ethanol should be increased, as its excretion is also accelerated [17,22,25].

To correct metabolic acidosis, sodium bicarbonate is prescribed under control of arterial pH and serum bicarbonate levels [14,22,23].

Antidote therapy aims to reduce the conversion of methyl alcohol to its toxic metabolites. Ethanol and fomepizole are used as antidotes. Ethyl alcohol is a competitive substrate for alcohol dehydrogenase, and fomepizole is its reversible inhibitor. The affinity of ethanol for alcohol dehydrogenase is higher than that of methanol, so ethanol is mainly metabolized, disrupting the toxication of methyl alcohol [14,23]. During antidote therapy, it is necessary to maintain the serum ethanol concentration at 1 g/l [17,22]. Compared to ethanol, fomepizole has a minimum number of side effects, is easily dosed, and does not require constant monitoring of its concentration in the blood. However, fomepizole is used less often due to its high cost [4,7,16]. The administration of antidotes is continued until metabolic acidosis is compensated and the concentration of methanol in the blood drops below 0.1 g/l [17,20].

As an additional therapy, folic acid preparations are recommended to accelerate the elimination of formates [7,16,22].

Conclusion. Thus, the features of the biotransformation of methyl alcohol in the body, the mechanism of its toxic action, as well as modern methods of diagnosis and treatment of acute poisoning caused by methanol were considered.

Discussion

The findings of this study reaffirm the severe toxic effects of methanol poisoning, primarily attributed to its metabolic conversion into formic acid, which induces metabolic acidosis, optic nerve damage, and multi-organ failure. The study results align with previous research indicating that methanol ingestion leads to rapid systemic toxicity, with high mortality rates if untreated. The toxicokinetics of methanol metabolism suggest that the window for effective intervention is narrow, reinforcing the importance of early diagnosis and prompt administration of antidotal therapy. Compared to ethanol intoxication, methanol poisoning presents unique diagnostic challenges due to its delayed onset of symptoms and the inability to differentiate methanol from ethanol based on taste or odor.

The significance of these findings is evident in both clinical and public health contexts. From a theoretical standpoint, the study contributes to the understanding of methanol metabolism, particularly highlighting the role of formic acid as a key toxic mediator. This supports existing toxicological models that emphasize formic acid-induced inhibition of mitochondrial cytochrome oxidase, leading to cellular hypoxia and systemic acidosis. Practically, the study underscores the necessity of rapid screening methods, improved public awareness, and strict regulatory measures to prevent methanol poisoning cases. Previous research has indicated that regions with weak alcohol control policies report higher incidences of methanol poisoning outbreaks, reinforcing the need for tighter surveillance and control over methanol-containing products.

Policy implications of this study are substantial, advocating for mandatory methanol screening in alcohol products, stricter regulations on industrial methanol use, and increased healthcare preparedness for methanol poisoning cases. The high mortality and morbidity associated with methanol poisoning warrant the development of standardized emergency response protocols, particularly in regions with a high prevalence of counterfeit alcohol.

Despite its contributions, this study has several limitations. Firstly, the reliance on existing literature may introduce selection bias, as available data might not comprehensively represent all methanol poisoning cases, especially in low-resource settings. Secondly, variability in reported methanol toxicity thresholds and treatment responses across studies complicates direct comparisons and generalizability. Thirdly, while the study highlights treatment approaches such as ethanol and fomepizole administration, the cost and availability of these antidotes remain a challenge in many countries, limiting their practical application.

Future research should focus on improving early diagnostic capabilities through novel biomarkers and non-invasive detection methods to differentiate methanol poisoning from other alcohol-related toxicities. Additionally, studies investigating alternative, cost-effective antidotes and optimized treatment regimens tailored to resource-limited settings are needed. Longitudinal research on the long-term neurological and systemic effects of methanol poisoning could further enhance treatment strategies and post-recovery management. Furthermore, epidemiological studies assessing the impact of regulatory interventions on methanol poisoning incidence would provide valuable insights into policy effectiveness.

In conclusion, this study underscores the critical need for early diagnosis, effective treatment, and comprehensive policy measures to mitigate the burden of methanol poisoning. Strengthening public health interventions, improving clinical management, and advancing research in methanol toxicity will be essential in reducing morbidity and mortality associated with this life-threatening condition.

Conclusion

This review highlights the severe toxicological effects of acute methanol poisoning, emphasizing its high lethality due to metabolic acidosis, optic nerve damage, and multi-organ failure. The findings underscore the critical role of methanol metabolism, particularly the conversion to formic acid, in exacerbating toxicity. Diagnostic challenges persist, as early symptoms can mimic ethanol intoxication, delaying timely intervention. Current treatment strategies, including ethanol and fomepizole as antidotes, along with hemodialysis for toxin elimination, are effective but require early administration to prevent irreversible damage. The implications of this study stress the need for stringent regulatory measures to control methanol distribution, enhanced public awareness to prevent accidental or intentional ingestion, and the development of more accessible and rapid diagnostic tools. Future research should focus on novel biomarkers for early detection, optimized antidotal therapy, and advanced treatment protocols to improve survival outcomes and reduce long-term complications associated with methanol toxicity.

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