International Journal of
Health Systems and Medical Sciences

ISSN: 2833-7433
Volume 2 | No 4 | April -2023

Clinical Aspects and Epidemiology of Acute Alcohol Poisoning

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Abstract: The problems associated with alcohol and, in particular, with its excessive consumption, are on a par with major public health problems in the world and cause serious damage to human health, well-being and life. The problem of alcoholism is a complex, open dynamic system, dependent on and closely related to the biological and socio-economic systems. Undoubtedly, the increased negative impact of alcohol on the health of the population is due not so much to an increase in the consumption of alcoholic beverages, but to an increase in their toxicity. Studying the problem of alcohol poisoning, are few and consider it mainly from the standpoint of the clinic, pathogenesis and resuscitation. There is practically no study and analysis of the medical, socio-economic aspects of alcohol poisoning, their structure and prevalence.

Keywords: toxicology, concentration, intoxication, alcohol

Relevance : The generally accepted statistical indicators of the admission of patients with acute alcohol intoxication and the incidence of alcoholism do not reflect the real situation, since the number of identified and registered patients largely depends on the activity of toxicological and narcological services [1, 3]. To assess the epidemiological situation, a number of indirect indicators are used: the number of patients admitted to hospitals, the dynamics of alcohol consumption in a particular region, mortality from liver cirrhosis, the incidence of alcoholic psychosis, traffic accidents, injuries, etc. [fourteen]. Each of these indicators separately, of course, is ambiguous and can be disputed, but in combination they are quite informative.

Purpose of the study: To study the epidemiology of acute alcohol poisoning.

Results and discussions: Based on the analysis of some statistical data, it can be concluded that the prevalence of alcoholism in Uzbekistan is increasing, and the severity of its painful manifestations is steadily increasing [6]. According to WHO, in 2016 there were more than 180 million alcoholics (with “alcohol dependence syndrome”) in the world, and the prevalence rate was 2% [8]. And at the same time, the “alcohol” situation in developed countries is different [7].

M.A. Schuckit [7] characterizes the situation in the USA by 2015 as follows: 90% of Americans drink alcohol during their lifetime, 60-70% systematically use alcohol, have alcohol-related more than 40% of problems, 20% of men and 10% of women abuse alcohol, have alcohol dependence, i.e. 10% of men and 3.5% of women suffer from alcoholism [7]. These indicators have significant differences in different groups of the population and depend on the social status and cultural characteristics. The highest rates are distinguished, on the one hand, by strata of society with a high social status in terms of education and economic security, and, on the other hand, by some low-income groups of the population [7]. The prevalence of alcoholism among the population is determined by him as 19% among men and 3-5% among women, and alcohol abuse - 20% among

men and 10% among women. Begin to drink alcohol more often at the age of 15-20; the peak of the prevalence of alcohol abuse falls on 20-40 years [6]. "Alcohol" mortality was about 5% of total mortality. Among the more common causes of death were heart disease, malignant tumors and cerebrovascular disease. Moreover, while the percentage of alcohol-related road traffic accidents (RTCs) has fallen, fatal crashes still accounted for 50% of all crashes; with a steady trend towards gradual reducing the consumption of alcohol by high school students, 92% of them tried alcohol, 23% systematically drank, more than 28% from time to time got drunk to the point of severe intoxication [7]. In Uzbekistan, according to WHO, in 2016 alcohol consumption per-capita was 3.6 liters of pure ethyl alcohol per year, which is not a very high figure, but the number of acute alcohol poisoning and the number of people suffering from alcoholism is steadily increasing [8]. General characteristics of alcohol intoxication. Ethyl alcohol (wine alcohol, ethyl alcohol, ethanol) refers to narcotic drugs. Once in the body, alcoholic beverages act directly on the cellular structure of the body, as well as on organs through the central nervous system. When alcohol comes into contact with tissues, irritation develops in them, the degree of which depends on the place and duration of exposure and the concentration of alcohol. Alcohol dehydrates protoplasm, dissolves fats, coagulates proteins and causes tissue compaction. Alcohol acts much more strongly on young cells and cells rich in water. When taken orally, 20% of alcohol is absorbed into the blood through the stomach, and 80% through the intestines [1, 3, 4].

The absorption of alcohol into the blood occurs quite quickly and after 5 minutes it is determined in the blood, spreads through the bloodstream and easily penetrates the tissue barrier. Alcohol reaches its maximum concentration in the blood 1-2 hours after ingestion. Hagger (1937) found that after the introduction of alcohol through a tube into the stomach at the rate of 3 ml per 1 kg of body weight, 57.7% is absorbed after 30 minutes, 88.5% after 60 minutes, 93.4% after 1.5 hours, 100% of the total amount of alcohol administered - after 2 hours. R.O. received similar data. Feitelberg (1938) [1, 3, 4, 6]. Thus, alcohol administered orally is completely absorbed into the blood through stomach and intestines for 2 hours. Concentrated alcohol is absorbed faster, and on an empty stomach - very quickly. Foods, especially proteins and fats, delay the absorption of alcohol. After absorption, alcohol is distributed in the body almost evenly. The rate of distribution depends on the blood filling of the organs. Within an hour and a half after drinking alcohol, a balance is established throughout the body. The degree and speed of the concentration of alcohol in the blood and in tissues depend on the amount and degree of dilution of alcohol and the method of administration - immediately or in small doses, as well as the degree of filling of the stomach, the state of the internal organs and the central nervous system, as well as the individual characteristics of the person. However, the central nervous system (CNS) is the most sensitive to alcohol [1, 3, 4]. At high doses, the inhibitory effect of alcohol on the central nervous system is manifested by a violation of consciousness (blackout), a decrease in the severity of reflexes, relaxation striated muscles, i.e., the state of alcoholic anesthesia. The next, even deeper stage of alcohol intoxication is characterized by coma, which can result in death due to the toxic effect of alcohol on the hypothalamic region and vital centers of the brain stem, often with respiratory paralysis [1, 3, 4]. Ethyl alcohol is excreted with exhaled air (7%), with urine, feces, sweat and through the skin (3% in total). Small number in women excreted in milk. At an alcohol level of 0.5 g/l, approximately 0.1 g of alcohol is excreted in 200 g of milk, that is, an amount that does not harm a nursing baby [1, 3, 4, 6]. Mechanisms of development of acute alcohol intoxication. Acute alcohol intoxication is the most common pathological condition that occurs when drinking alcohol. The severity of disorders in acute alcohol intoxication is determined mainly by the amount of alcohol taken and the duration of the toxic effect. The lethal dose of ethanol at a single dose is from 4 to 12 g / kg of body weight (average 300 ml of 96% ethanol in the absence of tolerance to it). Tolerance to ethanol changes with age [1, 3, 4]. So, for example, a superficial (uncomplicated) coma in children develops at ethanol concentrations in the blood of 0.8-3.0 g / l, and in adults - 2.0-6.0 g / l. The state of deep coma is fixed when the content of ethanol in the blood in children is 2.0-5.4 g/l, and in adults - 3.0-8.5 g/l. At in the elderly, resistance to the narcotic and toxic effects of alcohol is sharply reduced. Sensitivity to the toxic effect of alcohol increases with overwork, malnutrition, sudden changes in the diet, in women during pregnancy, with certain diseases (liver, pancreas, diabetes mellitus, etc.), under the influence of a number of drugs and other

factors [1, 3, 4]. Gastrointestinal disorders are an indispensable attribute of acute alcohol intoxication and post intoxication state. They are manifested by acute pain in the stomach and diarrhea. They are most severe in patients with alcoholism [1, 3, 4, 6]. Pain in the stomach area is caused by erosive-hemorrhagic damage to the mucous membrane of the stomach and small intestine, especially in the duodenum and jejunum. Internal damage in the distal intestines is less pronounced. Diarrhea is a consequence of a rapidly emerging lactase deficiency and the associated decrease in lactose tolerance, as well as impaired absorption of water and electrolytes from the small intestine. In acute and subacute alcohol intoxication, there is a violation of absorption processes in the intestine [1, 3, 4, 6]. It concerns, first of all, folates, water, salts and long-chain fatty acids. Malabsorption of neutral lipids occurs due to the direct effect of ethanol on the membranes of the epithelium of the small intestine and is accompanied by an increase in the release of lipids from the epithelial cells into the intestinal lumen. Folate deficiency causes the development of secondary damage to the mucosa of the gastrointestinal tract, increasing the disruption of intestinal absorption processes. Alcohol in acute and chronic intoxication causes a sharp selective increase in the permeability of the mucosa of the jejunum and ileum for macro molecules (albumin, beta-lactoglobulin, etc.). Acute and subacute intoxication that occurs when drinking beer causes a decrease in the activity of maltase and sucrase in the intestine, despite the high content of maltose in beer [1, 3, 4, 6].

Toxic encephalopathy (complex of mental, cerebellar, extrapyramidal and vegetovascular disorders). The clinical picture, as a rule, is dominated by various types of impaired consciousness and mental functions - from symptoms of CNS excitation (psychomotor agitation with euphoria, delirium, hallucinations, convulsive syndrome) to depression (lethargy, stupor, stupor). The main manifestations and severity of toxic alcoholic encephalopathy are determined, first of all, by the nonspecific effect of ethanol on the membranes of the cells of the central nervous system [1, 2, 4]. The first phase of the narcotic action is manifested by psychomotor agitation and vegetative disorders. As intoxication develops, this phase is replaced by a phase of oppression, up to the development of a coma. At the same time, there is a consistent involvement in the process of inhibition first of the higher parts of the central nervous system (loss of control over the situation, impaired attention, perception, speech, coordination of movements) and then - subcortical structures (decrease in pain and temperature sensitivity, violation of thermoregulation, tonic function of smooth muscles, involuntary urination, vomiting with aspiration of vomit, respiratory disorders, dysregulation of vascular tone and cardiac activity) [1, 3, 4]. The situation during this period is significantly complicated due to the increase in metabolic and hypoxic disorders associated with the oxidation of ethanol and the action of its metabolites (acetaldehyde, acetate, ketone bodies). There are serious disorders of homeostasis (violations of the acid-base state and water-electrolyte balance, microcirculation and hemocoagulation) [1, 2, 3]. The initial period of development of acute alcohol intoxication is characterized by a violation of the water and electrolyte balance as a result of repeated vomiting of central origin (less often - as a result of damage to the stomach and pancreas), which leads to loss of fluid, electrolytes and the development of hypotonic dehydration [1, 3, 4, 6] . Another mechanism of disturbance of water-electrolyte metabolism is associated with a decrease in the secretion of antidiuretic hormone as a result of the direct action of ethanol and with an increase in renin secretion. This leads to an increase in diuresis, which increases by 30-40% with alcohol intoxication of moderate severity [6]. Severe and longer intoxication leads to more pronounced tonic stimulation of the renin system, which, with the participation of angiotensin-11, aldosterone and catecholamines, causes a significant retention of sodium and chlorine, as well as the development of hypokalemia. It should be noted that the activation of the renin system has a direct attitude to the development of alcoholic, including postintoxication hypertension. Along with the imbalance of monovalent cations, in acute alcohol intoxication, calcium and magnesium metabolism disorders develop. Ethanol increases the excretion of calcium and magnesium in the urine and makes it difficult for them to be absorbed in the intestine, which at the initial stages of intoxication is accompanied by a decrease in the intracellular depot of these ions in the bone and other tissues of the body, and then leads to a decrease in their plasma levels [6]. The violations listed above are accompanied by the development of metabolic acidosis, which persists even in the post-intoxication period. The severity of acidosis correlates with the severity of intoxication and post intoxication

disorders. Acidosis is the result of increasing metabolic disorders (hyperketonemia, hyperlactatemia), as well as a deficiency of bicarbonate ions that leave the body with vomit and urine [1, 3, 4, 6]. Later to metabolic acidosis is often accompanied by respiratory acidosis, which is the result of depression of the respiratory center, a decrease in alveolar ventilation, an increase in the "dead space" of the lungs and aspiration of vomit. Metabolic alkalosis is considered the most unfavorable form of acid-base balance disorder. The causes of the latter are the loss of hydrochloric acid with vomiting and the development of pronounced hypokalemia. Metabolic extracellular alkalosis in hypokalemia due to the compensatory movement of hydrogen and sodium ions into cells and the development of intracellular acidosis. This condition, in combination with hypocalcemia and hypomagnesemia, is the cause of the development of disturbances in myocardial excitability and contractility and causes the development of generalized hyperexcitability of the central nervous system (convulsive syndrome). The favorable prognosis of metabolic alkalosis is determined by the body's capabilities in terms of the formation of compensatory respiratory acidosis [1, 3, 4, 6].

Respiratory disorders are the dominant cause of death in cases of alcohol poisoning and its surrogates. The most common acute respiratory failure caused by violations of the function of external respiration of an aspiration-obstructive nature (retraction of the root of the tongue, aspiration of vomit, nasopharyngeal mucus, laryngobronchospasm, etc.) [1, 6]. Deep depression of consciousness in alcoholic coma is accompanied by respiratory failure of the central type, which is a consequence of gross metabolic disorders and the development of cerebral edema. Less common is the so-called parenchymal respiratory failure, caused by the development of shock against the background of adult respiratory distress syndrome, pulmonary edema that occurs against the background of acute renal failure and hemodynamic disturbances against the background of severe myocardial dystrophy or confluent pneumonia. Pulmonary edema occurs due to general overhydration of the body and usually accompanied by the appearance of peripheral edema and cerebral edema [1, 6]. Circulatory disorders in acute alcohol intoxication include exotoxic shock, acute cardiac or cardiovascular failure (primary toxigenic and secondary somatogenic collapse, hemodynamic pulmonary edema), and various conduction and heart rhythm disorders [1, 3].

Toxic alcoholic hepatopathy, which is based on dystrophic and necrotic changes in the liver parenchyma, is accompanied by a violation of all its vital functions. Liver damage is manifested by a decrease in synthetic, detoxifying and regulatory (influence on interstitial and other types of metabolism) functions. Most often, acute alcoholic hepatopathy occurs in the form of a cytolytic syndrome, which is understood as a violation permeability of cellular and intracellular membranes of hepatocytes [1, 3]. Clinical manifestations of this syndrome are a sudden increase and soreness of the liver, icterus of the sclera and skin, general intoxication, fever, and, in advanced cases, hepatic encephalopathy. Destruction products of the liver parenchyma are directly involved in the formation of endogenous intoxication, which, in turn, leads to an increase in degenerative-dystrophic diseases. changes in the liver and other parenchymal organs, contributing to the development of complications, including infectious ones [1, 3, 4, 6].

Alcoholic hypoglycemia usually occurs in malnourished individuals, most often in alcoholics, as well as in healthy people who have previously fasted for 36-72 hours. It can occur both in the intoxication phase and in the post-intoxication period and quickly lead to the development of hypoglycemic coma. Ethanol-induced hypoglycemia is associated with inhibition of gluconeogenesis and impaired regulatory functions of the hypothalamic-pituitary system. Severe hypoglycemia can develop in insulin- or sulphonuretic-treated diabetics after taking relatively small amounts of alcohol. Ethanol has the ability to potentiate glucose-stimulated insulin secretion, provoking the development of reactive hypoglycemia. It should also be taken into account that that against the background of taking ethanol, the release of insulin increases in response to the administration of arginine and tolbutamide [7, 8]. Acute alcohol intoxication is characterized by encephalopathy of mixed (toxic and hypoxic) genesis, manifested by coma and neurological disorders, impaired respiratory function by aspiration-obturation (airway obstruction by the secret of the tracheobronchial tree, saliva, vomit, retraction of the tongue), and in advanced cases and the central type, pathology from the cardiovascular system (myocardial dystrophy and relative hypovolemia with the development of

circulatory collapse), acute metabolic disorders (metabolic acidosis). Diagnosis of alcohol poisoning is based on anamnesis data, the clinical picture of intoxication, the results of an additional examination, including a chemical-toxicological analysis of the remains of the fluid taken, gastric lavage and other biological material (blood and urine) [1, 3, 8].

It is necessary to pay attention to the dynamics of acid-base balance - with severe alcohol intoxication, the appearance of metabolic acidosis is noted. There is also an increase in blood lactate over 2.0 mmol / l, in proportion to the severity of intoxication. Often there is an increase in liver enzymes - ALT, AST, alkaline phosphatase and blood diastase - which indicate the development of acute or chronic damage. liver and pancreas. Alcohol poisoning, especially against the background of chronic use, is accompanied by hypercoagulability, which is manifested by an increase in hematocrit, a shortening of VSC, an increase in PTI and fibrinogen levels [1, 8]. On the ECG, the identification of hypotrophy of the left ventricle and signs of diffuse damage to the heart, not associated with changes in homeostasis in acute poisoning, are important: low voltage of the teeth, serration of the OKB - which in young people is an indirect confirmation of alcoholic myocardiopathy [1, 8]. Ultrasound of the liver in chronic intoxication reveals the appearance of toxic hepatitis, later with the transition to alcoholic cirrhosis of the liver. On ultrasound of the kidneys, there is a decrease in echogenicity, pallor of the parenchyma. In patients against the background of the third stage of chronic alcoholism, the appearance of alcoholic nephropathy, wrinkled kidneys is detected [1].

CONCLUSIONS: Acute alcohol poisoning is the leading and most complex pathology in the structure of chemical injuries. For prevention, it is necessary to develop a comprehensive rational neurometabolic therapy for patients with acute alcohol intoxication against the background of chronic alcohol abuse. It is necessary to create an emergency round-the-clock drug treatment service for patients with various complications of chronic alcoholism, equipped with modern laboratory equipment and having a full-fledged intensive care unit.

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