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THE EFFECT OF ALCOHOL ON THE BRAIN AND ITS STRUCTURES

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Abstract

Journals, materials of scientific conferences, as well as other information sources have been studied to collect reliable information about changes in the brain and its structures under the influence of alcohol.

It has been proven that alcoholism is an extremely serious disease that has negative consequences. With alcohol intoxication of the brain, damage to neurons in all parts of the brain is observed, as well as changes in the cerebral vascular system, the function of the hippocampus, cerebellum, hypothalamus, up to changes in the vascular plexuses of the lateral ventricles.

Keywords: alcohol, neurons, brain edema, hippocampus, cerebellum, lateral ventricles.

Relevance

The human brain is an incredibly complex organ that leads to the coordinated work of almost all systems in the body, in addition, responsible for cognitive skills and thought processes. Unfortunately, the fast pace and not the healthiest lifestyle slowly undermine the body, depleting its reserves and compensatory mechanisms. Today, there is an acute problem in the world associated not only with high morbidity, but also disability and even mortality from alcoholism [24,46].

The steady growth of alcoholism poses the task of further deepening knowledge in the field of its etiology, diagnosis, prevention and treatment. Recently, the problem of alcoholism has become increasingly relevant due to the epidemiological and social danger of this disease, and the problem of alcoholism remains relevant for theoretical and practical medicine. Alcohol can cause damage to most organs, it contributes to the development of more than 60 different diseases, makes a significant contribution to the morbidity and mortality of the population [7].

The medical and demographic consequences of excessive alcohol consumption are manifested in a decrease in the general state of health, an increase in morbidity, premature mortality of the population, which, in turn, contributes to their early initiation to alcohol. The social consequences of drunkenness and alcoholism are very wide. The problem of alcoholism is a whole complex of social aspects that affect all areas of normal functioning of society, which are studied not only by medical professionals, but also by specialists in other fields [6,13,14].

One of the first places among structures particularly sensitive to the toxic effects of ethanol is occupied by the Central nervous system [8]. Moreover, the spectrum of ethanol's influence on this system is quite wide: in small doses, alcohol exhibits a depressant effect; when consuming large doses of ethanol, a more widespread oppression of a significant number of different CNS structures develops, leading to disorganization and disruption of highly integrated processes [27,28].

In acute alcohol poisoning, edema of all parts of the brain comes to the fore. In the vascular plexuses of the brain, edema and swelling of the intercellular substance, basal membranes and stroma of the villi are also observed, which leads to compression and desolation of capillaries, necrosis and desquamation of the epithelium, etc. [10].

In the structures of the brain of rats with acute ethanol intoxication, signs of perivasal edema are determined, often areas of hemorrhages of diapedetic genesis, which can be both local and widespread. In some cases, hemorrhagic foci, in addition to white and gray matter, affect the subcortical and stem parts of the brain [36].

Currently, the study of the effect of alcohol intoxication on the rat body continues, the authors touch on such topics as: the intrauterine effect of alcohol on the reactivity of cerebral arterioles and its susceptibility to ischemic damage in adulthood [30,37]; changes in the level of circulating insulin and ghrelin in chronic alcohol intoxication [46].

Along with this, the intake of ethanol into the body is accompanied by significant violations of all types of metabolism, the functioning of neurotransmitter systems, the occurrence of endocrine disorders [10].

The toxic effect of ethanol in general negatively affects the functioning of almost all organs and systems of the body. At the same time, the absolute majority of severe health disorders as a result of alcohol consumption is caused by systematic excessive intake of alcohol, which is chronic alcohol intoxication (KHAI). [23,29]

Hyalinosis and sclerosis of the intramural arteries are found in the vessels of the base of the brain, which indicates the toxic effect of ethanol on the vessels. There is 28 damage to the 3rd and 5th layers of the brain tissue of the frontal lobes, as well as the molecular and ganglion layers of the cerebellar cortex in the form of an increase in the number of hyperchromic, reduced in volume neurons and a decrease in the number of normochromic cells [39].

Alcohol has a pronounced neurotoxic effect on the developing brain, causing cognitive impairment in young people [3,45,35].

The term "cognitive functions" is often used to refer to such higher integrative brain functions (VIFM) as memory, attention, thinking and executive functions [17,48,32].

There may be other mechanisms for reducing students' academic performance, including a decrease in cognitive functions due to an increase in the level of erroneous actions. A correlation was established between the activity of neurons in the anterior cingulate gyrus and the number of mistakes made in the experiment with the academic performance of students [45].

Other studies [40,43] revealed atrophy of the cortex (69%), atrophy of the corpus callosum (61,9%), cerebellar atrophy (30.9%), expansion of the furrows of the cortex, third and lateral ventricles (50%), vasogenic changes (26,1%). In most cases, atrophy of the corpus callosum was combined with atrophy of the cortex and less often with vasogenic disorders. More often, pathological changes were localized in the corpus callosum.

According to some data, cortical atrophy was most pronounced in the frontal lobes, and its degree correlated with the intensity of alcohol abuse [41].

With chronic alcohol intoxication (CHAI), degenerative changes in all brain structures occur in the central nervous system. Encephalopathy develops under the influence of both direct intoxication with ethanol and its derivatives, and alimentary insufficiency (deficiency of B vitamins, enzymopathy). During macroscopic examination, swelling of brain tissues is observed, and as a result, smoothness of the gyrus. Dystrophic changes in the frontal lobe are the cause of a decrease in the mass of brain matter, which leads to intellectual disorders. Motor dysfunctions and spatial orientation disorders are evidence of degenerative lesions of cerebellar tissues. Spot hemorrhages in the lumen of the III ventricle are often observed. Under the microscope, small hemorrhages are detected, which lead to atrophy of the parenchyma of the brain and to vacuole dystrophy of neurons. All this is expressed in the formation of small areas of necrosis and the presence of dark, wrinkled neurons and "shadow cells". Thickening of the walls of small arteries is a sign of arteriosclerosis. The soft meninges are thickened, hyperemic, and sclerosed in places [15].

The effect of ethanol on the brain provokes the development of acute (or transient), delayed and chronic reactions of nerve cells. The acute reaction that develops with ethanol poisoning is expressed by edema-swelling of neurons and is a consequence of a combination of alterating factors: toxic effects of ethanol and hyperproduction of catecholamines, excessive release of which causes hyperpolarization of neurons of the anterior limbic cortex. The acute reaction is more pronounced in the dopaminergic layer III of the anterior limbic cortex than in layer V [16]. The delayed reaction of neurons is caused by the weakening and cessation of acute effects of toxicants, as a result of which the signs of previous prolonged intoxication effects of ethanol, its toxic metabolites and increased concentrations of catecholamines that caused cell atrophy come to the fore.

In the brain of rats predisposed to alcohol dependence, morphofunctional differences in the anterior hypothalamus are determined, which are expressed in differences in structural organization at the tissue, cellular and molecular levels, including a significantly lower volume density of pericaryons of neurons of the supraoptic nucleus compared to animals not prone to alcoholization. An immunohistochemical study using primary antibodies against tyrosine hydroxylase revealed a lower degree of expression of immunoreactive material in the pericaryons of neurons and in the neuropile of supraoptic nuclei of the hypothalamus of rats prone to the formation of alcohol dependence, which indicates the presence of immunophenotypic differences and catecholamine metabolism in neurons of the nuclei of the anterior hypothalamus[5].

The direct toxic effect of ethanol and its metabolites on neurons is due to the induction of glutamate neurotoxicity as a result of a decrease in neurofilament protein synthesis or a violation of rapid axonal transport. Ethanol activates inhibitory GABA receptors and is an antagonist for glutamate receptors (NMDA). Chronic alcohol abuse leads to a violation of the function of these neurotransmitter systems with a compensatory decrease in GABA activity and an increase in glutamate. As a result of impaired ethanol metabolism, cytotoxic proteins are formed that reversibly affect the cells of the nervous system; there is also a dose-dependent effect of ethanol on the severity of damage to the nervous system. The resulting cytotoxic proteins also act on other tissues [9].

Qualitative and quantitative analysis of pathomorphological changes in the hippocampus of rats in the simulation of chronic alcoholism was carried out. Signs of reversible and irreversible damage were revealed, most pronounced in the pyramidal layer of CA3, as well as signs of the development of compensatory adaptive processes with a predominance of atrophy, which were most pronounced in the CA1 zone, the focal nature of the lesion of pyramidal neurons was revealed. Alcohol damage to neurons is detected in all parts of the brain. Particularly intense signs of neuronal damage are found in the hippocampus [36, 42].

The study showed that the morphological picture of brain damage in alcoholic and HCV-associated cirrhosis of the liver is based on pathological changes in the glio-angio-neuronal complex, which had some differences depending on the type of underlying liver disease. The most striking of them concerned the manifestations of the glial reaction, which is a characteristic morphological feature of PE [30,33,42]. It has been shown that in cirrhosis of alcoholic etiology, productive changes in astrocytes prevailed in the brain, including the appearance of multiple Alzheimer's type II astrocytes, as well as spongiform changes in gray and white matter. In contrast, in cirrhosis, the outcome of HCV infection was marked by proliferation of microglial cells by the type of microgliosis in the white matter of the brain[12].

Alcoholism in humans and chronic alcohol intoxication in animals has a distinct morphological expression in the brain. At the same time, alcohol primarily affects the vessels, especially the microcirculatory bed, which leads to an increase in the permeability of both the vessels themselves and the blood-brain barrier. As a result, ethanol and its metabolites easily penetrate the blood-brain barrier, causing severe damage to neurons up to their death. Hypoxia associated with hemodynamic disorders also

contributes to this [25, 26].

With alcohol intoxication in GM, there is swelling of its substance, fullness and swelling of the vascular plexuses of the ventricles. There is an increased formation of cerebrospinal fluid in the ventricles of the brain. Alcohol freely passes through the blood-brain barrier, while facilitating the passage of a number of amino acids. As the concentration of alcohol in the blood increases, the cells of the large hemispheres first react to its presence, then the medulla oblongata and spinal cord. The problem of the role of neurochemical reactions in the processes of tolerance and physical dependence on ethanol and in the mechanism of alcohol withdrawal is complex, contributes to numerous studies, but has not yet been solved [21, 22]. Alcohol has a potentiating effect on the development of cerebral edema, complicating the course of traumatic brain injury [4]

Other studies [39, 43] revealed atrophy of the cortex (69%), atrophy of the corpus callosum (61.9%), cerebellar atrophy (30.9%), expansion of the furrows of the cortex, third and lateral ventricles (50%), vasogenic changes (26.1%). In most cases, atrophy of the corpus callosum was combined with atrophy of the cortex and less often with vasogenic disorders. More often, pathological changes were localized in the corpus callosum. According to some data, cortical atrophy was most pronounced in the frontal lobes, and its degree correlated with the intensity of alcohol abuse [40]. R. Emsley et al. pronounced brain atrophy with predominant damage to subcortical structures was found in patients with alcoholism with Korsakov syndrome. The authors associate this fact with nutrition-dependent diencephalic pathology, but not with the neurotoxic effect of alcohol on the cerebral cortex [32].

A comparative assessment of the state of the key neurotransmitter systems of the brain stem and cerebellum of rats with chronic alcohol and morphine intoxication was carried out. Prolonged alcohol and morphine intoxication has a similar effect on the state of dopaminergic neurotransmission in the brain stem. This is manifested by a decrease in dopamine levels on the 14th and 21st days of administration of ethanol and morphine, as well as a significant increase in the concentration of homovanilic acid throughout the duration of the experiments. In addition, prolonged administration of alcohol and drugs has an identical inhibitory effect on the serotonin content in the brain stem on the 21st day of intoxication [2].

In the work of Semke V.Ya., Melnikova T.N., Bohan N.A., the existence of topographic features of the electrical processes of the brain of alcoholics is noted, which consist in a decrease in the cortical activity of the frontal zones and indicate frontal dysfunction. The authors explain this fact by atrophic changes in the frontal cortex, as the most sensitive to toxic effects [20]. The peculiarities of changes in the dynamics of intercentral relations reflect the influence of nonspecific brain structures on the cortex and may reflect a malfunction of homeostatic systems that support the background tone of the cortex [1]. Linking the pronounced inhibitory effect of alcohol on cortical structures with the various effects of stem and diencephalic structures on the right and left hemispheres, some scientists conclude that alcohol dependence mainly manifests the effect of mesodiencephalic structures [18,20].

Thus, our analysis of data from domestic and foreign literature shows that the spectrum of ethanol's effect on the central nervous system is quite wide, which dictates the need to use pathomorphological, morphometric, histochemical research methods for a detailed study of the changes occurring in all parts of the brain. It is necessary to develop ways to correct the negative effects of alcohol on the human body.

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