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CHANGES IN ANATOMICAL PARAMETERS OF INTERNAL ORGANS WHEN EXPOSED TO ETHYL ALCOHOL (REVIEW OF LITERATURE)

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Annotation. *This article presents modern medical views on the analysis of research on the morphological aspects of alcohol effects on the internal organs of the human body.*

The analysis of the literature confirms that the changes in the internal organs observed during alcohol intoxication are practically irreversible, as the compensatory capabilities of the body are disrupted.

Key words: *ethanol, acetaldehyde, patamorphological changes, acute and chronic alcohol intoxication.*

At the present stage, the study of the mechanisms of alcohol's influence on the human body, as well as the likelihood of irreversible changes in the functioning of the human body with regular consumption of large doses of alcoholic beverages, is a popular topic of scientific research. In this regard, the study of the problem of alcohol's effect on the human body is very relevant today.

Alcohol abuse is one of the most important factors influencing the occurrence and course of chronic diseases, as well as one of the direct or indirect causes of early disability of a relatively young and able-bodied population. In direct dependence on the level of alcohol consumption by the population, there is a dangerously high prevalence of alcoholic pathology of internal organs (alcoholic cardiomyopathy, alcoholic liver disease, alcoholic encephalopathy, etc.) [14].

The progress of civilization should be connected with the spiritual self-improvement of people, with the rejection of the absolutization of material values and the revival of harmony between man and nature in the spirit of the best achievements of the people. The imbalance in human relations with nature is the root cause of all

diseases of civilization. Modern civilization is characterized by a significant increase in the number of diseases, which are based on the perverted inclinations of the individua. These include tobacco smoking, alcoholism, drug addiction, substance abuse, AIDS, hepatitis, and others [32,33].

Recently, the use of surrogate drinks has increased significantly, which reduces the quality and life expectancy of users, which subsequently leads to disability and premature death. According to the World Health Organization (WHO), about 2.5 million people die each year as a result of consuming surrogate drinks [10,24,26].

The research of I.V. Beynikov (2016) established that alcohol surrogate poisoning causes a significant synchronous change in the blood plasma and erythrocytes of patients and a violation of one of the links in the pathogenesis of severe tissue and organ disorders, which should be taken into account when developing approaches to correcting metabolic disorders [4].

With sudden death from alcoholic cardiomyopathy, morphological changes in the kidneys are observed, manifested in the form of tubular epithelial dystrophy. As a result of damage to the histohematic barrier of the renal capillaries, dyscirculatory damage occurs, which exacerbates the violation of vascular permeability of the vascular wall [18, 28, 31].

The results of epidemiological and experimental studies have shown that excessive ethanol consumption causes more than 60 types of diseases and, depending on the dose, is a cause contributing to the development of a large number of other diseases [27,29].

According to literature sources, in some cases, people who consume alcoholic beverages in moderation may show signs of alcoholic damage to internal organs [6].

The condition of KHAI occurs when alcohol is consumed in quantities exceeding the individual capabilities of the body, as well as the consumer's dehydrogenase systems to oxidize the incoming latter and its metabolites, resulting in the development of alcoholic illness, which can acquire mental or somatic pathology with a predominant lesion of the "target organs" [13].

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Morphological changes in the studied organs, indicating the toxic effects of ethanol, develop in parallel and interrelated ways [1].

The morphometric characteristics of the effect of prenatal alcoholism on the vessels of the microcirculatory bed and neurons of the human brain have been studied [19].

Under conditions of prenatal alcoholism, a decrease in the diameter and perimeter of capillaries was found, leading to a deterioration in vascularization of brain tissue, as well as a decrease in the size of neuroblasts [20].

The organs of the digestive system are the first to experience the effects of alcohol upon contact. In the organs of this system, metabolic processes inside cells are disrupted [9].

It should be emphasized that ethanol has hyperosmic and lipotropic effects, which in the area of ionic membrane channels contribute to a change in the state of aggregation of lipids [11] and an increase in cholesterol content occurs in them, the structure of the phospholipid layer changes, and the function of membrane enzymes is disrupted [13].

Ethanol, having lipophilicity, leads to an increase in vascular permeability of the microcirculatory bed of the blood-brain barrier [22].

In mitochondria, acetaldehyde is oxidized by aldehyde dehydrogenase. Most of all, aldehyde dehydrogenase is found in the mitochondria, and in the cytosol in a smaller amount. An intermediate metabolite, acetaldehyde, has a hepatotoxic effect, which is released in large quantities during alcohol intoxication. When an intermediate metabolite, acetaldehyde, enters the bloodstream, functional and structural disorders develop in other tissues and organs. There are three main forms of morphological changes in the liver in alcoholic lesions: fatty liver (steatosis), alcoholic hepatitis and cirrhosis of the liver [13, p. 480].

When activated by cytokines or acetaldehyde, the following structural changes occur in liver cells, such as a decrease in vitamin A reserves and the production of fibrous tissue. Fibrous tissue grows around the vessels of hepatocytes, as a result of which oxygen delivery to them is disrupted [8,16].

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Today, a combination of chronic viral hepatitis B and especially With chronic alcoholic hepatitis is most often observed. The frequency of detection of hepatitis B and C virus markers has increased significantly [2]. When the hepatitis C virus and alcoholic beverages are combined, the process of fibrogenesis in the liver increases [12].

Studies have shown that in acute alcohol poisoning, ethyl alcohol disrupts gluconeogenesis in the liver, inhibits the oxidation of fatty acids and causes a violation of lipid metabolism. As a result, this leads to a change in the lipid spectrum and an increase in the concentration of phospholipids in the blood plasma and liver of animals [3].

The problem of alcohol's influence on the development of gastroduodenal pathology is becoming particularly relevant because the organs of the gastrointestinal tract function as the first barrier for alcohol to enter the body and are the first to experience its negative effects [30].

The analysis of pathomorphological changes in the epithelium of the small intestine under the influence of alcohol revealed an increase in intestinal villi. There was a decrease in the size of the bordered cells and an increase in the size of the goblet-shaped cells that make up the bulk of the villi epithelium. The goblet-shaped cells were overflowing with mucosal secretions [21].

Chronic alcohol intoxication increases the pathology of the endocrine system, where duct sclerosis and parenchymal atrophy occur in the pancreas. With pancreatitis, there is a violation of glycogenesis associated with glucose deficiency. The changes in the hypothalamic-pituitary-germinal system are reduced to testicular and ovarian atrophy. There is sclerosis and atrophy of the cortical substance in the adrenal glands [25].

As a result of alcohol entering the duodenum, it disrupts its motility and increases pressure in the pancreatic duct, which impedes the outflow of pancreatic juice and calcinates form in it, but not all authors confirm this phenomenon.Следует отметить, что в первые месяцы жизни человека деление клеток сердца–миоцитов – практически прекращается, и сердечная мышца после воздействия

спиртных напитков почти не регенерирует, а на месте погибших мышечных тканей образуются рубцы [17].

With chronic alcoholism, stromal vascular disorders and organ edema are observed in the testes, leading to dystrophic changes in the spermatogenic epithelium. This leads to malnutrition and the inability of full-fledged spermatogenesis [2].

Under the influence of ethanol, biochemical processes in macrophages of lung tissue slow down, resulting in a sharp decrease in their phagocytic activity. Ethanol and its metabolites increase the permeability of the vascular wall, which leads to dystrophic and destructive changes in lung tissue and the subsequent development of pneumosclerosis [5].

According to the results of a study by V.S. Paukova and Yu.A. Erokhina (2004), patients with alcoholic cirrhosis of the liver develop hepatorenal syndrome, and tubular epithelium dystrophy occurs in renal structures [15].

Alcoholic nephropathy is caused by the toxic effects of ethanol associated with alcohol excess, which is combined with acute alcoholic hepatitis. In this case, we can talk about alcoholic glomerulonephritis, which note a decrease in the glomerular area by 3 times compared with the norm [7,13,15].

The effects of acute and chronic alcohol intoxication on parenchymal organs are described in detail in scientific literature. The pathogenetic mechanisms of kidney damage in acute and chronic alcohol intoxication have not yet been fully studied, and the current points of view are quite controversial. It should be noted that there are few scientific studies on the toxic effects of alcoholic beverages on the kidney structure, and the conclusions given in them are contradictory [23, 34].

Thus, our analysis of data from domestic and foreign literature shows that many authors are currently considering the issues of morphogenetic changes in internal organs in alcoholic injuries. In addition, the etiological and pathogenetic mechanisms, as well as morphological features in alcoholic hepatopathy and alcoholic nephropathy, were formulated. The details of the morphogenesis of alcoholic lesions of internal organs are widely discussed in the scientific literature. At the same time,

the microanatomic and immunohistochemical cellular structures of the kidneys remain insufficiently studied when exposed to ethanol in the experiment.

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