MORPHOLOGICAL CHANGES OF INTERNAL ORGANS IN CHRONIC ALCOHOLISM

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Abstract: *It has been established that in the structure of mortality in recent years, death from chronic alcoholism and acute alcohol intoxication, as well as associated complications, occupies a leading position, second only to mortality from cardiovascular pathologies and malignant neoplasms [1]. Affecting not only the physical, but also the mental sphere of life, alcohol has been, is and is becoming more and more a serious problem, threatening dangerous consequences not only for the individual, but also for the entire population as a whole.*

Key words: chronic alcoholism, Encephalopathy, alcoholic hepatitis, cardiomyopathy, Sertoli cells.

However, despite the apparent simplicity of identifying people suffering from alcohol dependence and the corresponding chronic alcohol intoxication, it is the design and formulation of the official part of the diagnosis that is associated with a number of features that slow down statistical work and sometimes distort its work . However, the monotony and stereotypical nature of internal organ lesions in people with chronic alcohol intoxication allows us to identify a number of pathomorphological signs that reflect chronic alcoholism, which dictates the need to distinguish between the pathology of internal organs that form the main and immediate causes of death, and the pathology that reflects the toxic .

Organ changes in chronic alcoholism

Brain damage

With chronic alcohol intoxication, degenerative changes in all brain structures occur in the Cerebral Nervous System. Encephalopathy develops under the influence of both direct intoxication with ethanol and its derivatives, and alimentary insufficiency (deficiency of B vitamins, enzymopathy). Macroscopic examination shows edema of the brain tissue, and as a result, smoothness of the convolutions. Dystrophic changes in the frontal lobe cause a decrease in the mass of the brain matter, which leads to intellectual disorders. Motor dysfunctions and disorders of spatial orientation-indicate degenerative lesions of the cerebellar tissues. Spot hemorrhages in the lumen of the third ventricle are often observed. Under the microscope, small hemorrhages are detected, which lead to atrophy of the brain parenchyma 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. Thickening of the walls of small arteries is a sign of arteriosclerosis. The soft medulla is thickened, hyperemic, and sometimes sclerotic. [9, 10]

Liver damage

The main manifestation of liver damage in the systematic use of alcohol is alcoholic liver disease. Alcoholic liver disease is a set of morphogenetic manifestations of changes in the structure and functions of the liver that occur when using hepatotoxic doses of alcohol. There are three main forms of alcoholic liver disease - cirrhosis of the liver, fatty dystrophy and alcoholic hepatitis. The most common liver damage in alcoholic disease is fatty dystrophy - a pathological process characterized by a violation

of the metabolism of lipids in liver cells, which eventually leads to the accumulation of fat in the cytoplasm of hepatocytes, mainly triacylglycerides. Fatty liver dystrophy is found in the literature as liver steatosis, hepatosteatosis, fatty hepatosis, fatty liver . Microscopic examination of the pathological material reveals hepatocytes, in the cytoplasm of which fat inclusions of different diameters are determined. In accordance with this, adipose dystrophy is divided into large-drop and small-drop.

In large-drop steatosis, the size of fat vacuoles exceeds or corresponds to the diameter of the cell nucleus, while the nucleus itself is located eccentrically. According to the increase in the degree of fat infiltration of hepatocytes, Fatty Liver Dystrophy is divided into minor, moderate and pronounced forms. In small-drop steatosis, a large number of small fat droplets are detected in the hepatocyte, the nucleus remains in the center. It is assumed that the accumulation of fat inclusions is associated with damage to the mitochondrial apparatus of the cell and activated lipid synthesis. Steatosis is characterized by anisocariosis, the expansion of the sinuses, in the lumen of which single neutrophils are detected, however, inflammatory infiltration of the portal tracts is not observed. In a pathoanatomical study, the liver is enlarged in size, the anterior edge is smooth, the surface is smooth, yellowish-brown color on the incision. However, fatty liver dystrophy is not an irreversible process. Stopping alcohol intake without exposure to other toxic factors leads to complete morphological normalization of liver cells with the restoration of functional activity. With continued alcoholism, the next stage in the progression of alcoholic liver damage is alcoholic hepatitis. This is a serious disease that develops against the background of a prolonged alcoholic excess, while very often the onset of the disease is preceded by fatty dystrophy. The main mechanism of damage that leads to the development of pathology is the toxic effect of acetaldehyde, in particular, its participation in the launch of lipid peroxidation of cell membranes, which later leads to hypoxia and necrosis. Necrotized elements will provoke the development of the inflammatory process, which is the basis of hepatitis. This pathology is characterized by pronounced microscopic and macroscopic pictures that allow you to correctly make a diagnosis. Microscopic examination of liver tissues reveals small-node monolobular cirrhosis of the liver, characterized by variability in the size of hepatocytes, individual necrosis, inflammatory infiltration by neutrophils, mononuclears, pericellular fibrosis, as well as large-drop and small-drop steatosis. A distinctive feature of alcoholic hepatitis is the presence of a large amount of alcoholic hyaline, or Mallory bodies, in the cytoplasm of hepatocytes. Alcoholic hyaline has not only a cytotoxic effect, but also stimulates leukotaxis, has antigenic properties, which leads to the formation of circulating immune complexes. Macroscopically, the liver is enlarged in size, dense, the surface is fine-grained, mottled [5, 7].

Heart muscle lesions

A common pathology in people who abuse alcohol is a lesion of the cardiovascular system, represented by acute microcirculation disorders, signs of heart fibrillation and dystrophic damage to cardiomyocytes, which are considered as a manifestation of alcoholic cardiomyopathy. The main mechanism of sudden cardiac death is the electrical instability of the myocardium, leading to ventricular fibrillation. Alcoholic heart disease is a group of heart diseases, common to which is selective damage to the myocardium by products of ethanol metabolism. The main mechanisms of cardiopathogenic action are the effect on the energy supply of the cell, the direct toxic effect of ethanol and acetaldehyde, the disruption of the coupling between contraction and excitation, free radical damage, a violation of lipid metabolism, an imbalance of catecholamines and ions. These mechanisms ultimately lead to the

development of heart failure, which is characterized by a violation of the structure of the contractile apparatus of cardiomyocytes and their functional asymmetry. On microscopic examination, alcoholic cardiopathy is characterized by vacuolization and small-drop fatty dystrophy of the cytoplasm of cardiomyocytes. There is also a deposition of lipids in the myocardial stroma, around the intramural vessels. The content of lipofuscin is increased, the pigment grains are detected throughout the cytoplasm. A disordered arrangement of myofibrils in cardiomyocytes is characteristic. Also, additional signs of cardiopathy are the bizarre shapes of cell nuclei with a transparent perinuclear zone, dilatation of full-blooded vessels, an increase in the space between cardiocytes and capillaries. Interstitial and perivascular fibrosis of the entire myocardium with focal lymphohistiocytic infiltration is detected. Macroscopic examination shows the expansion of all the cavities of the heart. With further aggravation of the disease, dilatation will progress. The myocardium becomes yellowish due to the massive subepicardial accumulation of adipose tissue. An important sign is that the coronary arteries most often remain intact [6].

Defeat of the bronchopulmonary system

One of the main systems of the body that are directly involved in the utilization and elimination of the decomposition products of alcohol is the bronchopulmonary system. In almost half of cases, the causes of death in patients who have abused alcohol are pathologies associated with the respiratory system. Alcohol intake aggravates the course of the pathological process, making it prolonged, provoking abscending and the formation of bronchiectasis. Respiratory disorders caused by alcohol consumption are caused by a decrease in surfactant production, deterioration of mucociliary clearance, damage to normal microflora, and a decrease in humoral and cellular immunity. These mechanisms lead to a violation of gas exchange in the alveoli and inhibition of the protective properties of the body, which leads to an exacerbation of bronchopulmonary infection. Microscopic examination of the lung tissue reveals a thickening of the walls of blood vessels with perivascular cell infiltration, also characterized by an overflow of blood capillaries and small veins, but along with this, there is also a desolation of blood vessels. Vascular disorders are invariably combined with sclerotic processes in the interstitial lung tissue and atrophy of the pulmonary parenchyma. The macroscopic picture depends on the underlying disease of the respiratory system, the course of which was aggravated by alcohol intake.[4]

Damage to the pancreas

The main reason for the violation of the normal activity of the pancreas is alcohol. The cells of the gland are extremely sensitive to the toxic effects of ethanol and its metabolic products. The effect of ethanol on the pancreatic tissues leads to primary pancreatic hypersecretion, expressed by stimulation of the production of proteolytic enzymes and spasm of the sphincter of Oddi. This blocks the normal outflow of juice and increases the pressure in the duct, and the activated enzymes begin to digest the tissue of the gland itself, which leads to necrosis. Systematic exposure, on the contrary, leads to the progression of secretory insufficiency of the gland, but in any case there will be a gross violation of the functional activity of the pancreas. The most common lesion of the pancreas when drinking alcohol is acute pancreatitis. Since acute pancreatitis is based on primary destructive changes in acinuses caused by intra-organ (intracellular) activation of digestive enzymes produced by the pancreas, and the developing enzyme autolysis of acinous cells is accompanied by the formation of foci of necrosis and aseptic inflammation, detected by microscopic examination. Morphological changes depend on the

duration of the process. At the initial stage, the swelling of the gland tissue is determined, the appearance of scattered small foci of fat necrosis. Further development involves an increase in the area of the lesion, followed by the replacement of the dead connective tissue or the formation of pseudocysts [8].

Defeat of the reproductive system

With an increase in the duration of Chronic alcohol intoxication, pathomorphological changes in the structure of the testicle increase, characterized by sclerosis, destruction of the spermatogenic epithelium with a decrease in the spermatogenesis index by 2 times with a duration of Chronic alcohol intoxication of more than 10 years, a decrease in the endocrine activity of the testicular tissues until the development of hormonal organ failure. Morpho-functional changes in the testicles of people who abuse alcoholic beverages simultaneously indicate the suppression of hormone-producing and reproductive functions, which increases as the duration of Chronic alcohol intoxication increases. Irreversible sclerotic processes in the stromal component that develop under the influence of chronic toxic effects of alcohol on the testicular tissue cause an increase in the volume of these structures by 2 times with a duration of Chronic alcohol intoxication of more than 10 years. Sertoli cells in people who abuse alcoholic beverages are more resistant to the toxic effects of alcohol than Leydig cells, which is expressed by the manifestation of feminization in men. [24]

Under the influence of ethyl alcohol poisoning, the testicles of growing animals lag far behind in their development.

With continued drunkenness, the productive capacity falls, as sexual aspirations fall, azoospermia and testicular atrophy occur." [25]

Alcohol is a mutagen, and mutated cells in the body destroy their own immune system. If it does not cope — the person has cancer. Mutations in the germ cells do not bother a man and do not manifest themselves in any way, but they can manifest themselves in his children. That is why doctors recommend abstaining from alcohol for 2-3 months before conception, as this is the period of life of spermatozoa.

The main mechanisms of influence on the sexual system:

* Direct traumatic effect on the tissues of the genital glands, spermatozoa due to blood clot, capillary thrombosis, oxygen starvation of the testicular cells.
* Degenerative changes in the genitals: narrowing of the seminal tubules, reduction of the testes, decreased sperm production.
* Hormonal changes. Due to chronic alcoholism, the amount of testosterone decreases, which is replaced by the female hormone estradiol, which negatively affects libido.
* Decreased activity of the hypothalamic-pituitary system, which causes disorders of the reflex activity of the central nervous system, which is responsible for the implementation of sexual function. Hypothalamic impotence develops.

Impact on the reproductive system of women

Systematic excess of the permissible dose of alcohol (several times a week, more than 20 ml of ethanol at a time — for women, more than 30 ml of ethanol - for men). Conceiving while heavily intoxicated or drinking alcohol during pregnancy increases the risk of health problems in the child.

Conclusion

Thus, the given picture of the pathology of internal organs with the justification of the

mechanisms of damage allows us to confirm the concept of alcohol disease as a stage and stereotypical process characterized by an increase in the negative effects of ethanol in dynamics, ranging from minimal changes in the vessels of the microcirculatory bed to extensive multi-organ pathology with irreversible changes. This fact gives the right to assert that with the timely refusal of alcohol, it is possible to avoid its harmful effect on the individual's body and on the population as a whole.

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