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Morphofunctional Aspects 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. For all the apparent simplicity of identifying people suffering from alcohol addiction 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 stereotyping of lesions of internal organs in persons with chronic alcohol intoxication allows us to identify a number of pathomorphological signs reflecting chronic alcoholism, which dictates the need to distinguish between the pathology of internal organs, which forms the main and immediate causes of death, and pathology, which is a reflection of the toxic effects of eubiotic, coupled with the underlying disease, not related to alcohol.

Keywords: chronic alcoholism, encephalopathy, alcoholic hepatitis, cardiomyopathy, Sertoli cells.

Brain damage

With chronic alcohol intoxication in the Cerebral nervous system, degenerative changes occur in all structures of the brain. 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 swelling of the brain tissue and, as a result, smoothness of the gyrus. Dystrophic changes in the frontal lobe cause a decrease in the mass of brain matter, which leads to intellectual disorders. Motor dysfunctions and spatial orientation disorders indicate degenerative lesions of 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 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. Thickening of the walls of small arteries is a sign of atherosclerosis. The soft medulla oblongata 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 hepatotoxic doses of alcohol are consumed. There are three main forms of alcoholic liver disease - cirrhosis of the liver, fatty degeneration and alcoholic hepatitis. The most common liver lesion in alcoholic disease is fatty degeneration - a pathological process characterized by a violation of lipid metabolism in liver cells, which ultimately 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 dystrophy. Microscopic examination of pathological material reveals hepatocytes, in the cytoplasm of which fatty inclusions of different diameters are determined. In accordance with this, fatty 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. By increasing the degree of fatty infiltration of hepatocytes, fatty liver dystrophy is divided into minor, moderate and pronounced forms. With small-drop steatosis, a large number of small fat droplets are found in the hepatocyte, the nucleus remains in the center. It is assumed that the accumulation of fatty inclusions is associated with damage to the mitochondrial apparatus of the cell and activation of lipid synthesis. Steatosis is characterized by anisocariosis, expansion of the sinuses of the nose, in the lumen of which single neutrophils are detected, but inflammatory infiltration of the portal pathways is not observed. In pathoanatomic examination, the liver is enlarged in size, the anterior edge is smooth, the surface is smooth, yellowish-brown in 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 restoration of functional activity. With ongoing alcoholism, the next stage of progression of alcoholic liver damage is alcoholic hepatitis. This is a serious disease that develops against the background of prolonged alcohol excess, while very often the onset of the disease is preceded by fatty degeneration. The main mechanism of damage leading 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 further leads to hypoxia and necrosis. Necrotized elements will provoke the development of the inflammatory process that underlies hepatitis. This pathology is characterized by pronounced microscopic and macroscopic patterns that allow for a correct diagnosis. Microscopic examination of liver tissues reveals small-nodular 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. Alcohol 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, spotted [5, 7].

Lesions of the heart muscle

A common pathology in people who abuse alcohol is a lesion of the cardiovascular system, represented by acute microcirculation disorders, signs of cardiac fibrillation and dystrophic damage to cardiomyocytes, which are considered as a manifestation of alcoholic cardiomyopathy. The main mechanism of sudden cardiac death is 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, disruption of the connection between contraction and excitation, damage by free radicals, lipid metabolism disorders, imbalance of catecholamines and ions. These mechanisms eventually 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. Under microscopic examination, alcoholic cardiopathy is characterized by vacuolization and small-drop fatty degeneration of the cytoplasm of cardiomyocytes. There is also a deposition of lipids in the myocardial stroma, around intramural vessels. The content of lipofuscin is increased, pigment grains are found throughout the cytoplasm. The 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, the expansion 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 exacerbation of the disease, the expansion will progress. The

myocardium becomes yellowish due to 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 body systems directly involved in the disposal and excretion of alcohol decomposition products is the bronchopulmonary system. In almost half of the cases, the causes of death of patients who have abused alcohol are pathologies associated with the respiratory system. Alcohol intake aggravates the course of the pathological process, making it protracted, provoking abscesses 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 thickening of the walls of blood vessels with perivascular cellular infiltration, also characterized by overflow of blood capillaries and small veins, but along with this, emptying of blood vessels is also observed. Vascular disorders are invariably combined with sclerotic processes in 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 gland cells are extremely sensitive to the toxic effects of ethanol and its metabolic products. The effect of ethanol on pancreatic tissues leads to primary hypersecretion of the pancreas, 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 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 enzymatic 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, swelling of the gland tissue is determined, the appearance of scattered small foci of fatty necrosis. Further development involves an increase in the area of the lesion, followed by the replacement of 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 testicular tissues up to the development of hormonal organ failure. Morphofunctional changes in the testicles of people who abuse alcoholic beverages simultaneously indicate suppression of hormone-producing and reproductive functions, which increases with increasing duration of chronic alcohol intoxication. Irreversible sclerotic processes in the stromal component, developing under the influence of chronic toxic effects of alcohol on 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 poisoning with ethyl alcohol, the testes of growing animals lag far behind in their development.

With continued drunkenness, productive capacity decreases, as sexual aspirations decrease, azoospermia and testicular atrophy occur" [25].

Alcohol is a mutagen, and mutated cells in the body destroy their own immune system. If he 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, since this is the period of sperm life.

The main mechanisms of influence on the reproductive system:

* Direct traumatic effect on the tissues of the genital glands, spermatozoa due to blood clot, capillary thrombosis, oxygen starvation of testicular cells.

* Degenerative changes in the genitals: narrowing of the seminal tubules, a decrease in the size of the testicles, a decrease in 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 violations of the reflex activity of the central nervous system, which is responsible for the realization of sexual function. Hypothalamic impotence develops.

Impact on the reproductive system of women

Systematic excess of the permissible alcohol dose (several times a week, more than 20 ml of ethanol at a time — for women, more than 30 ml of ethanol - for men). Conception in a state of heavy alcohol intoxication or alcohol consumption during pregnancy increases the risk of health problems in the child.

Conclusion

Thus, the above 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 staged 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, its harmful effects on the body of an individual and on the population as a whole can be avoided.

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