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DAMAGING OF HEART IN ALCOHOLIC INTOXICATION OF RATS

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Summary
Alcohol abuse is an important medical and social problem of our time. According to World health organization statistics, heart disease has been the leading cause of death for over 20 years. Heart disease today accounts for 16% of all deaths in the world. Since 2000 the number of deaths from cardiovascular diseases increased by more than 2 million and in 2019 reached almost 9 million. The purpose of this article is to analyze, summarize and draw conclusions from the information provided by the authors.

Keywords: alcoholic intoxication, damage, heart, rats.

ПОРАЖЕНИЕ СЕРДЦА ПРИ АЛКОГОЛЬНОЙ ИНТОКСИКАЦИИ КРЫС

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Резюме
Злоупотребление алкоголем является важной медико-социальной проблемой современности. Согласно статистике Всемирной организации здравоохранения, сердечно-сосудистые заболевания являются основной причиной смерти на протяжении более 20 лет. Сегодня на болезни сердца приходится 16% всех смертей в мире. С 2000 г. число умерших от сердечно-сосудистых заболеваний увеличилось более чем на 2 млн и в 2019 г. достигло почти 9 млн. Цель данной статьи - проанализировать, обобщить и сделать выводы из предоставленной авторами информации.

Ключевые слова: алкогольная интоксикация, поражение, сердце, крысы.

Introduction

The Federal State Statistics Service (Rosstat-2011) in Muscovites identified the cause of death from alcohol associated with damage to the following organs of alcoholic liver disease - 36.8%, alcoholic cardiomyopathy - 33.2%, degeneration of the nervous system caused by alcohol - 12.4%.

For Russia, the abuse of alcoholic beverages is becoming a national disaster. At the same time, it is impossible to take into account the number of surrogates and falsified alcohol, which is quite widespread (Nemtsov A.V. 2007). In addition, statistics calculate alcohol consumption per capita, that is, including children, women and the elderly. And yet, alcohol consumption in Russia is 14.5 liters per capita.

At the same time, chronic alcohol intoxication (CAI), in addition to independent medical and social significance, contributes to the emergence, recurrence and progression of somatic diseases that suffer from people who abuse alcohol. Annually mortality in Russia from diseases associated with alcohol abuse reaches 700 thousand, and about 40 thousand die from poisoning with alcohol surrogates in the country (Nemtsov A.V. 2007).

According to Yu.P. Lisitsyn et al (1990) AR Lingford-Hughes at all. (2003), alcohol abuse attracts the attention of doctors only when patients develop alcoholism, characterized by physical mental dependence on alcohol and severe mental disorders. At the same time, it is not taken into account that behind the vivid manifestation of alcoholism there are many years of alcohol abuse, which is not accompanied by alcohol dependence and severe mental symptoms and is called "drunkenness".

Recovering from alcoholism, according to many psychiatrists, occurs only in 3-5% of cases, and despite the efforts of psychiatrists and narcologists from all over the world, Paukov V.S. et al. (2004) cannot get the problem out of the dead end. In addition, even those "cured of alcoholism" retain severe changes in almost all internal organs. This happens because before the development of alcoholism, a person goes through a long period of CAI, during which changes develop and progress in the organs and systems of the body. For many years they remain compensated and do not have pronounced symptoms. Only when the compensatory and adaptive capabilities of the organism are exhausted do psycho-pathological symptoms and the clinic of alcoholism appear. That is why the concept of alcoholism does not reflect the breadth of the pathogenesis of CAI and requires a different interpretation of the disease caused by long-term alcohol abuse. Understanding this led us to the need to develop new concepts of alcoholic disease (AD) as suffering with a much longer and more diverse patho and morphogenesis of alcoholism.

Tarasova OI, (2007),. Paukov V.S. et al. (2019). determined at the same time, analyzing the pathogenesis and morphogenesis of AD, it should be borne in mind that ethanol is a normal metabolite of the body, it participates in the metabolism of lipoproteins and phospholipids, increasing the level of high and very high density lipoproteins, regulates cholesterol metabolism, as well as the synthesis of cell and intracellular membranes ... When the possibility of basal liver metabolism is

exceeded, alcohol metabolites, primarily acetaldehyde, enter the bloodstream, which have a damaging effect on various structures of the body, primarily on the vessels of the microvasculature, as well as on cells of all organs and membranes of intracellular structures.

T.A. Golovanova (2012) studied the physiological properties of cardiomyocytes in the culture of myocardial cells of newborn rats. Found that the culture imitates the processes of cessation of division and the transition from hyperplasia to hypertrophy of myocardial cells. Under the conditions of primary culture of myocardial cells in adult rats, he revealed the proliferative activity of resident stem cells, but established their reduced capacity for cardiomyogenesis.

In clinical experiments on animals and on an isolated heart preparation Belovitsky O.V. (2008) found that ethanol reduces the size of the zone of myocardial ischemia, reduces the severity of electrographic changes and has a powerful antiarrhythmic effect in occlusion of the coronary artery

The study of P O Ettinger (1976) revealed, when modeling alcoholism in dogs, the HQ interval time increased by 9 m / s, and the QRS complex expanded by 18 m / s. These changes are rare when taken for a short period of time, do not appear at all when exposed to acute alcohol and are not associated with ventricular hypertrophy, necrosis, inflammation. Long-term alcohol consumption even with proper nutrition, visible disturbances in ventricular conduction and morphological changes are due to the stable toxic effect of ethanol.

The study of N.V. Shlyakhtin (2009) revealed the nature of the relationship about the increase in the severity of alcohol intoxication with the systolic and diastolic function of the left ventricular myocardium. Found a dependence of changes in heart rate turbulence indicators, namely an increase in Turbulence onset and a decrease in Turbulence slope, associated with the severity of acute alcohol intoxication.

Histochemical parameters (E.I. Bystrova 2009), activity of alcohol-oxidizing enzyme systems using computerized quantitative morphometry in cases of sudden death from ACMP (alcoholic cardiomyopathy) and IHD (ischemic heart disease) in comparison with rapid death from mechanical injury.

According to E.V. Kuznetsov (2013) macroscopic and organometric analysis of the heart in chronic combined intoxication with opiates and ethanol, which made it possible to reveal an increase in the mass and size of the heart, the height and width of both atria and ventricles, changes in the ventricular and cardiac indices. He found that with chronic combined intoxication with opiates and ethanol, pathomorphological changes develop, indicating the formation of secondary toxic cardiomyopathy in psychoactive substance users.

Development of ethanol tolerance in the experiment Luis M at all (2004) assessed body temperature and recovery of two reflexes after a high dose of ethanol in rats receiving chronic and acute ethanol consumption. The animals have previously been exposed to chronic or acute alcohol use. The results showed that 56-day-old animals developed greater tolerance with moderate to acute ethanol consumption. However, tolerance to the effects of motor impairment did not develop, as all groups took a long time to recover reflexes.

In chronic alcoholism, the amount of trace elements in the blood, especially potassium, decreases. Moses Elisaf at all. (2002).

Paukov V.S., et al. (2004) identified a lot of adipose tissue under the endocardium and around the intramural vessels in alcoholics. In the coronary arteries and in their branches there are flat atherosclerotic plaques, stenosing vascular lumens by 35–50%; sclerosis of their walls is expressed. In the vessels of microcirculation, hyalinosis, widespread, although irregular atrophy and desquamation of endothelial cells, stasis and sludge of erythrocytes. Plasmorrhage and fibrinoid necrosis of the vessel walls, perivascular edema, and diapedetic hemorrhages, especially in ethanol poisoning, were noted. The number of microcirculatory vessels is reduced. The myocardium is flabby, the heart cavities are stretched, the endocardium is thickened and sclerosed. Sometimes in cardiomyocytes, alcoholic hyaline was detected in the form of Mallory's little bodies, or "rod-shaped inclusions." Fatty degeneration and lipofuscinosis of muscle cells are expressed. Hypertrophied cardiomyocytes alternated with areas of muscle fiber atrophy. There are many foci of overcontraction and fragmentation of myofibrils, especially in those who died in a state of alcoholic delirium. In cases of sudden death from alcohol poisoning, there was a disappearance of the transverse striation of myofibrils as a result of melting of the Z discs, multiple foci of myocytolysis with lymphoid cell infiltration. All those who died in cardiomyocytes showed swelling of mitochondria, pronounced fragmentation of their cristae, blurred, indistinct membranes of intracellular structures. The amount of glycogen granules in the sarcoplasm is sharply reduced. The number of damaged cardiomyocytes was almost 1.5 times higher than their number in the myocardium of those suffering from drunkenness and increased by 6.2 times in comparison with the myocardium of the deceased from the control group. In all observations, diffuse focal cardiosclerosis was noted, the total area of which was 28 ± 0.5%, which is significantly 1.4 times more than in those suffering from drunkenness.

In acute heart attack against the background of alcohol intoxication A.G. Reznik (2013) found: obesity of the myocardial stroma, atrophy of cardiomyocytes along with their hypertrophy and the disappearance of striated striation. In muscle cells, areas of

significant lysis of myofibrils, the presence of large vacuoles, dilatation of the tubules of the sarcoplasmic reticulum, mitochondriosis, lipid infiltration of cardiomyocytes, and the accumulation of lysosomal formations are revealed. Some kernels can be small and pycnotic, while others are oversized and bizarre in shape.

In most cases, myocardial hypertrophy is detected in dilated cardiomyopathy, as a rule, it is accompanied not only by the flattening of the apex of the heart, as a result of which the heart acquires a spherical shape, but also by a uniform expansion of all its cavities. On the contrary, the same macroscopic sign in alcoholic cardiomyopathy is accompanied by expansion of only the left ventricle of the heart (Sokolova O.V. 2018). The most important and characteristic macroscopic sign of alcoholic heart disease is its obesity, manifested in the form of a pronounced proliferation of epicardial adipose tissue (Moiseev V.S. et al., 2012). This feature distinguishes alcoholic cardiomyopathy from other types of cardiomyopathy. However, an increase in the amount of epicardial fatty tissue may not be associated with alcohol consumption, but may be a manifestation of both general constitutional obesity and concomitant endocrine pathology.For alcoholic cardiomyopathy, typical in this case is the presence of a yellowish tint of the myocardium on the cut, associated with the development of parenchymal and mesenchymal fatty degeneration, caused by the constant long-term toxic effect of ethanol and its metabolites (Pigolkin Yu.I., Morozov Yu.E. et al., 2012). The study found that secondary vascular collapse of a toxic nature is due to depletion of the compensatory-adaptive functions of the circulatory system under the influence of hypoxia, a decrease in coronary artery perfusion, shock, endogenous intoxication syndrome and hepatic dysfunction. The developing circulatory disorders are often accompanied by disturbances in the activity of the heart, which are based on the onset of the low emission syndrome caused by the direct cardiodepressive effect of ethanol and associated with the development of acute toxic myocardial dystrophy. This syndrome is manifested by a drop in the contractile function of the myocardium and a reduction of the VCB (volume of circulating blood). Toxic myocardial dystrophy occurs as a result of a sharp disruption of energy processes in the myocardium, provoked by the cardiotoxic action of acetaldehyde, acetate and catecholamines.

According to scientists, morphological changes in the heart detected in alcoholic and dilated cardiomyopathies are not specific signs that allow differential diagnosis.

Thus, the authors provided a lot of literary information about the features of the morphofunctional structure of cardiomyocytes of the heart in alcoholism, but experimental studies of the morphogenesis of the cardiac conduction system of rats in postnatal ontogenesis are insufficiently sanctified and contradictory. The study of

morphofunctional changes in the conduction system of the heart in alcoholism, as well as methods for their diagnosis and correction, remains an urgent problem.

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