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MORPHOLOGICAL CHANGES IN THE LIVER IN NORMAL AND CHRONIC ALCOHOL POISONING

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Purpose of the study: to assess the morphological changes in the liver in normal and chronic alcohol poisoning and the features of its changes.

Materials and methods: to solve the set tasks and achieve the goals, it is planned to use experimental, histological, morphometric, and statistical research methods**.** It was obtained from 90 adult white rats of both sexes, weighing 180-280 g, which were quarantined for 14 days in the experimental vivarium. The feeding of the animals will consist of a standard food ration. All animals are divided into 3 groups.

Conclusion: If we consider 100% of micropericular pathologies observed in chronic alcohol intoxication, then 85-90% of them are parenchymal fatty degenerations with small, medium, large drops (alcoholic steatosis), 10-13% - alcoholic hepatitis (this is confirmed by the appearance in the cytoplasm of some hepatocytes of Mallory eosinophilic bodies, hydropic and balloon oxidative dystrophies in hepatocytes, accumulation of macrophages around these hepatocytes) alcoholic fibrosis was 3-5% (confirmed by the formation of fibrous tissue and sinusoidal tissue in the portal and periportal zone of hepatocytes killed by the toxic effect of alcohol, activation mast cells under endothelial cells under the influence of alcohol and the transformation of fibroblast cells into fibroblasts similar to the basement membrane - capillarization of sinusoids)

Key words: liver, alcoholic fibrosis, alcohol intoxication.

INTRODUCTION

Currently, many clinical and experimental studies are devoted to the problem of damage to the internal organs and systems of the human body in chronic alcohol poisoning. The role of ethanol in causing alcoholic damage to liver structures was determined [1,2,3,21,22].

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However, despite many studies on the effects of alcohol, insufficient attention has been paid to the study of morphological and functional changes in the tissues of internal organs against the background of chronic alcohol poisoning[4,5,6,23,11].

Chronic intoxication with alcohol is accompanied by fatty degeneration of the liver, disruption of metabolic processes in the organ[7,8,9,15,16,19].

Chronic consumption of ethanol leads to a decrease in the ability of mitochondria to oxidize acetaldehyde, and the imbalance between its formation and degradation increases. Acetaldehyde is 30 times more toxic than ethanol itself. The toxic effect of acetaldehyde on the liver is as follows: stimulation of lipid peroxidation and the formation of free radicals that damage the hepatocyte and its structures; binding of acetaldehyde to cysteine and glutathione leads to disruption of the formation of reduced glutathione, which in turn contributes to the accumulation of free radicals; reduced glutathione in mitochondria plays an important role in maintaining the integrity of the organelle; functional disorders of enzymes associated with hepatocyte membranes and direct damage to the membrane structure; increased intrahepatic cholestasis due to inhibition of hepatic secretion and binding of acetaldehyde to hepatic tubulin; activation of immune mechanisms (acetaldehyde is included in immune complexes involved in the formation of alcoholic liver disease) [9,10,11,26,27,28].

MATERIALS AND METHODS.

It was obtained from 239 adult white rats of both sexes, weighing 180-280 g, which were quarantined for 14 days in the experimental vivarium. The feeding of the animals will consist of a standard food ration. All animals are divided into 3 groups. All experimental studies were reviewed and approved by the bioethics committee of the Ministry of Health of the Republic of Uzbekistan.

Safety performed in 2 stages of the experimental level: the first stage - the study of morphological and morphometric parameters of the liver of newborns at 3, 6 and 9 months. The second stage - morphological and poisoning of the liver of 3,6,9-month-old rats, humane slaughter of animals (under ether anesthesia) and histological studies, record the research in journals, give a statistical view. report and report research report.

1) study of anatomical parameters (capsule load, cortical layer), parts of hepatocyte (vessel diameter, liver triad) in early late postnatal ontogeny.

2) Dynamic differences in the anatomical parameters of the liver of rats suffering from safe alcohol poisoning depending on age.

3) Study of morphometric indicators of liver microvessels under normal conditions and in mild alcohol poisoning.

Animal care and handling were in accordance with international standards and regulations for the handling of vertebrate laboratory animals.

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Laboratory animals fit on the shelves in special cages. The cage of the experimental animals shows the total number of breeding rats, the date of the experiment and the name of the researcher responsible for its installation.

RESULTS:

Morphological and morphometric indicators of the liver in chronic alcohol intoxication were studied. The livers of the control group, 3-month-old, 6-month-old, and 9-month-old white rats, and 3-month-old, 6-month-old, and 9-month-old white rats that received chronic alcohol intoxication were studied.

We used hematoxylin-eosin method and Van Gien's method to study the liver of non-white rats in the following groups, and micropreparations were prepared.

Photographing of micropreparations was carried out in x=4x10, 10x10, 40x10, 100x10 dimensions of the microscope.

The classical structure of the liver of the 3-month-old purebred rats in the control group is a liver lobe, i.e., a hexagonal prism. The periphery of the lobe contains vessels that bring the hepatic blood vessels to the liver lobe, including the interlobular vein (VII type), interlobular artery (VIItype), and interlobular bile duct. . Venous veins and arterioles pump blood into the sinusoidal capillaries. In the inner part of the lobe, hepatocytes form liver lobes in two rows, hepatocytes are located radially towards the central vein. One surface of the hepatocytes merges into the sinusoidal space and the other surface faces each other to form the bile duct, the sinusoidal capillary is formed by endotheliocyte cells that are in one layer, the difference is that the endotheliocyte cells are connected to each other but do not have a basal layer. Between the endotheliocyte and the hepatocyte, the gap of Disse is formed. Star-shaped Kupffer cells and Ito cells located in the sinusoidal cavity, and a central vein is visible in the center of the lobe. The blood is poured into the sinusoidal cavity or sinusoidal capillary from the arteries and veins located on the periphery of the liver lobe, and then flows into the central venous vessels and collects.

When examining the liver sections of the 3-month-old control group, the diameter of the aro vein is 36.6+-1.8 μm, the aro artery is 40.1+-2.3 μm, the aro bile duct is 15.1+-1.2 μm, and the aro triad is formed, the sinusoidal capillary space is 11+ -1.7 μm, hepatocytes are large, with round basophilic nuclei, the number of hyperchrome-stained nuclei is mostly mononuclear and a small number of two- and multinucleated hepatocytes, the surface of hepatocytes is 487.2 +-11.6, of which the surface of the nucleus is 58.91 +-1.88, the surface of the cytoplasm is 428.29+- 1.6, the ratio of nucleus to cytoplasm was 13.8%.+-0.07 and the ratio of stroma to parenchyma was 18%.

When examining the liver slices in the 6-month control group, the diameter of the aro vein is 37.2 +-1.9 μm, the aro artery is 43.3 + -2.6 μm, the aro bile duct is 17.1 + -2.3 μm, and the slices form the aro triad, the sinusoidal capillary space is 12.6 + -2.3

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μm, hepatocytes are large, with round basophilic nuclei, the number of hyperchrome-stained nuclei is mostly mononuclear and a small number of two- and multinucleated hepatocytes, the surface of hepatocytes is 495.9+-9.7, of which the surface of the nucleus is 59.7+-2.1, the surface of the cytoplasm is 436.2+-1.6 , the ratio of nucleus to cytoplasm is 13.7+-1.1% and the ratio of stroma to parenchyma is 19%.

Changes and indicators of liver morphometric parameters in experimental chronic alcohol intoxication.

Alcohol-induced changes in liver tissue in the liver tissue of 3-month-old outbred rats.

Various changes in liver vessels and hepatocytes were observed when 118 sections were prepared from 3-month, 6-month and 9-month-old rats' livers subjected to chronic alcohol intoxication.

Morphometric parameters of purebred rats in the control group.

No		3 months old	6 months old	9 months old
1	The diameter of the bubbles is μm	36.3 +-1.8	37.2+-1.9	38.21+-
2	The diameter of the intercalary artery is μm	40.1+-2.3	43.3+-2.6	43.7+-1.9
3	Interlobular bile duct diameter is μm	15.1+-1.2	17.1+-2.3	17.9+-1.7
4	Sinusoidal capillary gap μm	11+-1.7	12.6+-2.3	12.9+-1.3
5	Surface of hepatocytes	487.2+-11.6	495.9+-9.7	496.3+-10.5
	Nuclear surface	58.91+-1.88	59.7+-2.1	59.9+-3.4
7	Cytoplasmic surface	428.29+-1.6	436.2+-1.3	436.4+-1.5
8	Nucleus/cytoplasm ratio %	13.8+-0.07	13.7+-1.1	13.7+-0.08
9	Stroma/parenchyma ratio %	18	19	19.5

Liver morphometric parameters experimental in chronic alcohol intoxication changes and indicators.

Alcohol-induced changes in liver tissue in 3-month-old outbred rats.

The changes were mainly observed in the area of the portal tract of the liver and in sinusoids located close to the portal area. The size of the hepatocytes in the peripartum area increased, the eosinophil staining in the cytoplasm was hypochromic, when stained with hematoxylin-eosin, vacuoles of different sizes were detected in the cytoplasm, the nuclei were round. basophils are stained and shifted to the cell periphery, while the inclusions within the hepatocyte decrease in size toward the central vein. Vacuoles inside the hepatocytes in some parts completely cover the cell, their nuclei are not anicized, due to the increase in the size of the hepatocyte, the sinusoidal cavity is unevenly narrowed. In some preparations, hydropic vacuoles are found in hepatocytes in the peripartum branch, homogeneous eosinophilic hyaline in the cytoplasm of hepatocytes in the peripartum branch, and necrosis is detected in some hepatocytes. Around the necrotic hepatocytes and in the portal tract, there is an infiltration of macrophages, which decreases toward the central vein. When washing

micropreparations, septa of different sizes entering the sinusoidal capillary space from the portal tract are detected, and capillarization of endothelial cells similar to the basement membrane is observed in the sinusoidal endothelial cells. We can see that the walls of the central vein are thickened and the diameters are slightly narrowed.

CONCLUSION

If we consider 100% of micropericular pathologies observed in chronic alcohol intoxication, then 85-90% of them are parenchymal fatty degenerations with small, medium, large drops (alcoholic steatosis), 10-13% - alcoholic hepatitis (this is confirmed by the appearance in the cytoplasm of some hepatocytes of Mallory eosinophilic bodies, hydropic and balloon oxidative dystrophies in hepatocytes, accumulation of macrophages around these hepatocytes) alcoholic fibrosis was 3-5% (confirmed by the formation of fibrous tissue and sinusoidal tissue in the portal and periportal zone of hepatocytes killed by the toxic effect of alcohol, activation mast cells under endothelial cells under the influence of alcohol and the transformation of fibroblast cells into fibroblasts similar to the basement membrane - capillarization of sinusoids)

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