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COMPARATIVE CLASSIFICATION OF LIVER MORPHOMETRIC PARAMETERS IN THE LIVER AND IN EXPERIMENTAL CHRONIC ALCOHOLISM

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Abstract: Some diseases, on the basis of morphological changes of internal organs, methods of differential treatment of the direct cause of diseases have been developed. [2,7]. At the same time, the initial stage of diseases is not taken into account. [1,5].. In the current development of medical production, biological modeling has become the most important method of scientific knowledge, which creates a demand for such experimental models that most appropriately reflect the occurrence and mechanisms of human diseases in laboratory animals. In recent decades, scientists have devoted more time to the organization of extreme effects, changes in the actions of the internal organs of this alcohol poisoning. Some diseases, on the basis of quantified morphological changes in internal organs, each of which can lead to death, have eliminated the methods of differential diagnosis of the cause of death. It is noted that often extremely strong power load can start their potential danatogenic effect not in a single information, but in a certain period of time in each stress injury itself, which is unknown in advance.

Key words: Liver morphometric parameters, experimental chronic alcoholism.

INTRODUCTION

Some diseases, on the basis of morphological changes of internal organs, methods of differential treatment of the direct cause of diseases have been developed. [2,7]. At the same time, the initial stage of diseases is not taken into account. [1,5]. In the current development of medical production, biological modeling has become the most important method of scientific knowledge, which creates a demand for such experimental models that most appropriately reflect the occurrence and mechanisms of human diseases in laboratory animals. In recent decades, scientists have devoted more time to the organization of extreme effects, changes in the actions of the internal organs of this alcohol poisoning. Some diseases, on the basis of quantified morphological changes in internal organs, each of which can lead to death, have eliminated the methods of differential diagnosis of the cause of death. It is noted that often extremely strong power load can start their potential danatogenic effect not in a single information, but in a certain period of time in each stress injury itself, which is unknown in advance.

Analysis of the literature shows that the available information on the structural and functional state of the liver of laboratory rats is incomplete [5, 6,7]. Based on the above, we set out to study the age-related medical support of the liver of laboratory rats.

MATERIALS AND METHODS.

It is obtained from 239 mature white rats of both sexes, weighing 180-280 g, which passed 14 days of quarantine in the experimental vivarium. Animals under standard conditions. The feeding of the animals will consist of a standard food ration. All animals are divided into 3

groups. All experimental studies are reviewed, consulted and approved by the bioethics committee of the Ministry of Health of the Republic of Uzbekistan. fits. Morphological arrangement, logging in logs, its statistical processing and description of their report.

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, recording the research in journals, giving 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 manipulations will be in accordance with international norms and regulations for the handling of vertebrate laboratory animals.

Laboratory animals fit in special cages on shelves. In the cage of the experimental animals, the total number of pedigree rats, the date of the experiment and the name of the researcher responsible for its installation are marked in the cage.

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 aro artery and vein located on the periphery of the liver lobe, and then flows into the central venous vessels and collects.

When examining the liver slices of the 3-month-old control group, the diameter of the aro vein was $36.6+-1.8 \mu m$, the aro artery was $40.1+-2.3 \mu m$, the aro bile duct was $15.1+-1.2 \mu m$, and the aro triad was formed, and the sinusoidal capillary space was $11+-1.7 \mu m$, 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, nucleus cytoplasm ratio 13.8%.+-0.07 and stroma to parenchyma ratio made up 18%.

When examining liver slices in the 6-month control group, the diameter of the aro vein is 37.2 +-1.9 μ m, the aro artery is 43.3 + -2.6 μ m, the aro bile duct is 17.1 + - 2.3 μ m, and the slices form the aro triad, the sinusoidal capillary space is 12.6 + -2.3 μ m, 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, nuclear cytoplasm ratio 13.7+-1.1 % and stroma to parenchyma ratio It is 19%.

When examining the liver slices in the 9-month control group, the diameter of the aro vein was $38.21+-1.1 \mu$ m, the aro artery diameter was $43.7+-1.9 \mu$ m, the aro tube diameter was $17.9+-1.7 \mu$ m, and the aro triad was located and the aro triad was formed, the sinusoidal capillary space was $12.9+-1.3 \mu$ m, hepatocytes are large, with round basophilic nuclei, most of the number of hyperchrome-stained nuclei is single-nucleus, and a small number of hepatocytes are two- and multi-nucleus, the surface of hepatocytes is 496.3 + -10.3, of which the nuclear surface is 59.9+-3.4, the amount of cytoplasm is 436.4+-1.5, nuclear-cytoplasm

No		3 months old	6 months old	9 months old
1	The diameter of the bubbles is	36.3 +-1.8	37.2+-1.9	38.21+-
	μm			
2	The diameter of the intercalary	40.1+-2.3	43.3+-2.6	43.7+-1.9
	artery is µm			
3	Interlobular bile duct diameter is	15.1+-1.2	17.1+-2.3	17.9+-1.7
	μm			
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

ratio 13.7+-0.08 %. And the ratio of stroma to parenchyma It is 19.5%.

Morphometric parameters of purebred rats in the control group.

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.

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.

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. Macrophage infiltration is evident around necrotic hepatocytes and in the portal tract, 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.

Morphometric parameters of the liver of purebred rats subjected to chronic alcohol intoxication.

No		3 months old	6 months old	9 months old
1	The diameter of the bubbles is μm	36.6 +-1, 2	37.7+-1.3	38.3+-1.4
2	The diameter of the intercalary	40.4+-2.6	42.3+-2.2	43.8+-1.6
	artery is µm			
3	Interlobular bile duct diameter is	14.1+-1.9	15.1+-2.4	15.9+-1.6
	μm			
4	Sinusoidal capillary gap µm	9.1+-1,2	9.6+-2.1	10.9+-1.3
5	Surface of hepatocytes	499.2+-13.6	500.9+-10.7	506.3+-12.5
6	Nuclear surface	42.91+-1.88	41.7+-2.1	39.9+-3.4
7	Cytoplasmic surface	456.29+-1.3	459.2,2+-1,2	466.4+-1.4
8	Nucleus/cytoplasm ratio %	8.58+-0.07	8.32+-1.2	7.87+-0.08
9	Parenchyma/stroma ratio %	35	37	41

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

The results of the treatment of 3-month-old, 6-month-old, and 9-month-old white non-breed rats after chronic alcohol intoxication showed that positive results were observed in the above-mentioned microparameters, compared to rats with continued alcohol intoxication, the portal triad located at the periphery of the hepatic lobe showed a decrease in the diameter of the pulmonary artery, and the pulmonary artery. the decrease in diameter can be attributed to the decrease in signs of cholestasis in the bile ducts, the decrease in infiltration of macrophages in the periportal area, the relative expansion of the sinusoidal capillary cavity of the liver, the retention of fibrous septa from the portal zone to the periportal area, and the retention of fibrous tumors in the sinusoidal capillaries in some preparations. In hepatocytes, we can see fatty dystrophies with small droplets in the periportal branch, and we can see a decrease in fat droplets towards the central vein. A slight reduction in the size of hepatocytes can be seen in the nuclei, signs of proliferation, active mitosis, instead of necrotic hepatocytes, regeneration can be seen as a result of regenerative repair currents, macrophages gathered around hepatocytes with Mallory bodies in their cytoplasm can be seen, the sinusoidal capillary cavity is relatively widened, the blood flow is improved, and the diameter of the central veins is compensatory.

No		3 months old	6 months old	9 months old
1	The diameter of the bubbles is μm	37.3 +-1, 9	38.2+-1.9	38.91+-1.6
2	The diameter of the intercalary artery is µm	41.1+-2.6	42.3+-2.4	41.8+-1.5
3	Interlobular bile duct diameter is µm	15.7+-1.7	17.1+-2.3	18.9+-1.2
4	Sinusoidal capillary gap diameter µm	10.1+-1.3	11.6+-1.3	11.9+-1.3
5	Surface of hepatocytes	490.2+-13.6	495.9+-10.3	501.3+-10.3
6	Nuclear surface	52.91+-1.7	51.7+-2.1	50.9+-3.4
7	Cytoplasmic surface	437.29+-1.3	444.2,2+-1,1	450.9+-1.6
8	Nucleus/cytoplasm ratio	10.8+-0.06	10.42+-1.0	10.15+-0.07
9	Stroma/parenchyma ratio %	30	34	37

Changes and indicators of liver morphometric parameters in biocorrection after experimental chronic alcoholism.

CONCLUSION:

1. When comparing the morphometric indicators of the white rats in the control group, we can see that the morphometric indicators have increased relatively (vein diameter, arterial diameter, sinusoidal capillary, and central vein diameter). possible

2. In chronic alcohol intoxication, it was observed that the change in the liver started from the portal bridge and periportal branch and decreased towards the central vein.

3. If we consider 100% of the microperature pathologies seen in chronic alcohol intoxication, 85-90% of them are parenchymatous fatty dystrophies with small, medium, and large drops (alcoholic steatosis), 10-13% are alcoholic hepatitis (this is confirmed by the appearance in the cytoplasm of some hepatocytes eosinophilic Mallory corpuscles, hydropic and ballooning oxidative dystrophies in hepatocytes, and accumulation of macrophages around these hepatocytes can be seen) alcoholic fibrosis accounted for 3-5% (confirmed by the fibrous tissue and sinusoidal tissue formed in the portal and periportal zone of hepatocytes killed by the toxic effect of alcohol activation of mast cells under the endothelial cells under the influence of alcohol and fibroblast cells becoming fibroblasts similar to the basal membrane - capillarization of sinusoids)

4. Relatively increased pressure in the aorta vein (portal tract) (confirmed by narrowing of the sinusoidal cavity, capillarization of the sinusoidal vessels and fibrosis in the peripartum area) and relative cholestasis were observed.

5 In chronic alcohol intoxication, an increase in the number and accumulation of macrophages in the portal tract and periportal area was observed. (this happened as a result of increased dystrophy, necrosis, inflammation and pathological apoptosis in hepatocytes in this area)

6 Positive results were noted in changes in biocorrection of the liver after chronic alcohol intoxication. This can be confirmed by this, as we mentioned, 85-90% of chronic alcohol intoxication of the liver occurs with alcoholic steatosis, and this process is considered a reversible process, which means that the earlier alcohol consumption is stopped and the more positive results are obtained if it is properly treated. If chronic alcohol intoxication lasts longer, liver cirrhosis and liver failure will occur.

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