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Morphological Changes in the Liver During Acetic Acid Burns

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Abstract: Acute accidental and suicidal poisoning with acetic acid are characterized by varying degrees of damage to the liver parenchyma and stroma. In suicidal poisonings, depending on the stage of the stress reaction preceding the poisoning, more significant changes in the structure of the organ were revealed. The volume fraction of cells with signs of dystrophy and necrosis increases, and reparative processes decrease.

Keywords: poisoning, suicide, dystrophy, necrosis, stress.

Acute poisoning with acetic acid ranks first among poisonings with cauterizing poisons in Russia. Acetic acid is widely used in household cooking, so it is often the cause of poisoning, which is characterized by high mortality (6-17% of the number of hospitalized patients in various regions of the Russian Federation), despite intensive pathogenetic and symptomatic treatment [4].

Many aspects of acetic acid poisoning have been studied quite well. The severity of acetic acid poisoning is determined by the degree of damage to internal organs, which is associated both with the specific effect of acetic acid (hemoglobinuric nephrosis against the background of intravascular hemolysis of erythrocytes), and with exotoxic shock. The most significant morphofunctional changes are observed in parenchymal organs (lungs, liver, spleen, kidneys), the damage to which largely determines the clinical picture and severity of the disease during the acute period of poisoning [2,4].

A number of authors indicate that poisoning of various etiologies is accompanied by stress [1,2,3,5,78]. As previously established by numerous researchers, stress, despite its protective and adaptive nature, can have a pronounced nonspecific damaging effect on internal organs, being one of the links in the pathogenesis of various diseases [3,5,6,7,8]. However, in the literature on toxicology, the role of a stress factor in the pathogenesis of poisoning is given a rather modest place, despite the fact that poisoning causes a special type of stress - toxic stress.

It is known that suicidal types of poisoning are preceded by a stressful, traumatic situation [6].

An analysis of the literature showed that the influence of a stressful state on the nature of the course of poisoning has been practically not studied [8]. Different reactivity of the body, associated with different stages of the stress reaction at which poisoning occurs, and the duration of stress before poisoning, should probably influence the clinical picture of poisoning.

All this indicates that further research is needed aimed at studying the effect of stress on parenchymal organs during acute poisoning with acetic acid.

Purpose of the study: to elucidate the dependence of morphofunctional changes in parenchymal organs during acute poisoning with acetic acid at various stages of the stress reaction.

Materials and methods

The experiment was carried out on 205 outbred white male rats, weighing 180-200 g in the autumn-winter period. In accordance with the assigned tasks, all animals were divided into 4 groups.

Episode 1 Determination of morphofunctional changes in the liver in experimental animals poisoned with acetic acid without pre-stressing (accidental poisoning model) - 60 animals.

Episode 2. Determination of morphofunctional changes in the liver in experimental animals during poisoning with acetic acid at the stage of anxiety-stress reaction (model of poisoning in a state of passion) - 60 animals.

Episode 3. Study of morphofunctional changes in the liver in experimental animals during poisoning with acetic acid at the stage of exhaustion of the stress response (model of poisoning in a state of long-term depression) - 60 animals.

Episode 4. Intact - 25 animals.

To obtain the anxiety stage of the stress reaction, non-anesthetized rats were immobilized for 6 hours in a horizontal position on their back. To obtain the stage of exhaustion of the stress response, daily 6-hour immobilization of non-anesthetized rats in a horizontal position on their back was carried out for 14 days according to the method of HS Kim et al. (2006). Stress exposure was carried out at the same time of day from 9 a.m. to 3 p.m. After completion of the stress exposure, acetic acid was injected into the stomach of the animals through a probe. Animals were removed from the experiment on days 1, 3, 5, 7, 10, and 14 after toxic exposure.

A 30% aqueous solution of acetic acid in an amount of 0.5 ml was used as a cauterizing poison, which in terms of the pure substance is less than LD25 for rats, causing mild poisoning [2,3].

Histological, histochemical and morphometric research methods.

The material for the study was taken on days 1, 3, 5, 7, 10 and 14 after the onset of stress or toxic substance intake. After decapitation of the animals, liver fragments were fixed in a 10% formaldehyde solution, and after soaking in alcohol, they were embedded in paraffin. Serial sections 8 μm thick were prepared and stained with hematoxylin-eosin.

To study the connective tissue stroma of the organ, micropreparations were stained with picrofuchsin using the Van Gieson method. Reticular fibers were identified by impregnation with silver nitrate using the Bielschowsky method modified by Yurina. To identify glycogen, the Shabadash PAS reaction was used (with amylase control).

Light microscopy and morphometric studies of sections were carried out at a magnification of 200 times, using an Avtandilov grid and an eyepiece micrometer. Measurements were carried out in at least 20 fields of view (G.G. Avtandilov, 1990).

Qualitative and quantitative changes in organ cells and tissues were studied. In the liver parenchyma, the volume fraction of cells in a state of balloon degeneration, necrosis, the volume fraction of vessels, the volume fraction of hepatocytes of various sizes, as well as binuclear hepatocytes, collagen and reticular fibers, and the intensity of leukocyte infiltration were determined. The amount of glycogen was assessed on a 4-point scale (0, 1, 2, 3 points), after which the histochemical indicator was calculated using the formula:

$$GHP = (0\times n1+1\times n2+2\times n3+3\times n4)/\Sigma(n1+n2+n3+n4),$$

where n1, n2, n3, n4 is the number of hepatocytes.

The obtained numerical data were processed by standard statistical methods using Student's t-test in Statistica 6.0.

Results and discussion

A study of the histostructure of the liver in case of accidental poisoning with DL25 acetic acid showed that from 1 to 5 days, from the moment of poisoning, destructive processes increase. The number of cells with signs of balloon dystrophy and foci of necrosis increases, the plethora of the organ increases, and hemorrhages into the stroma and parenchyma from destroyed vessels increase. Leukocyte infiltration increases around necrotic hepatocytes and foci of hemorrhage. Thrombi are detected in the lumen of the vessels. The amount of glycogen decreases as much as possible in the first day after poisoning. In subsequent periods of observation, its amount remains stably at a low level and by day 5 is only 47.71±0.24% of the norm. The structure of the hepatic beams changes significantly due to toxic and stress alterations. The number of collagen fibers decreases by 1.8 times by day 5. From the 7th day from the moment of poisoning, activation of reparative processes is noted. The volume fraction of cells with dystrophic changes and foci of necrosis of liver tissue decreases. Fibroblasts predominate around areas of necrosis, and connective tissue is formed. The population of hepatocytes stabilizes: the number of medium-sized and large-sized cells increases, and the number of binucleate hepatocytes increases. But, as before, the amount of glycogen remains low, which is not restored even by the 14th day of the experiment. The plethora of the organ is preserved. Along with sharply congested areas, from the 10th day, foci of parenchymal ischemia with pronounced cellular infiltration are noted. Restoration of the liver structure is not complete by 14 days; foci of balloon degeneration remain, both in the center of the lobules and on the periphery, and up to 1% of hepatocytes are in a state of necrosis. The liver lobules are deformed due to the replacement of necrotic foci with connective tissue; leukocyte infiltration remains in the stroma, exceeding the control values by 2.04 times.

It has been established that in the liver of experimental animals in the stage of anxiety-stress reaction, one day after intragastric administration of DL25 acetic acid, significant degenerative changes in the parenchyma are detected.

The number of cells in the stage of vacuolar and balloon dystrophy is 72.35±1.83%, with a predominance of balloon dystrophy, and the volume fraction of necrosis increases to 13.62±0.34% ($p<0.05$). Necrotic foci are located mainly centrilobular, but are also detected on the periphery of the lobules. Large foci of necrosis were also identified, occupying an area of 2 to 3 lobules, which is associated with rheological disturbances caused by poisoning, as well as thrombosis of blood vessels due to these disturbances.

The liver is full of blood, the volume fraction of blood vessels increases to 32.26±0.56% ($p<0.05$). Foci of hemorrhage from destroyed vessels, intravascular thrombi and numerous aggregates of red blood cells are noted.

The population of hepatocytes changes significantly: the number of small hepatocytes in the center of the lobules increases by 10.58 times and in the periphery by 42.67 times; accordingly, the number of medium cells decreases, both in the center and on the periphery of the lobules, and large cells increase. The number of binucleate hepatocytes in the center of the lobules was reduced to 9.48±0.46%, and in the periphery - to 10.56±0.17% ($p<0.05$).

Leukocyte infiltration is represented by neutrophils and macrophages and amounts to 23.45±0.29 cells per 10,000 μm2. The amount of glycogen in hepatocytes was reduced by 10.86 times compared to the control and by 4.86 times compared to group 1 ($p<0.05$). The volume fraction of collagen fibers was reduced to 4.07±0.12% ($p<0.05$).

By the 14th day of the study, hepatocytes with signs of dystrophy are still detected in the liver. Their number is slightly less than in series 1 of the experiment and amounts to 16.68±0.39%, and the number of necrotic cells is determined at the level of 1.59±0.03%.

The number of small hepatocytes in the center of the lobules is 7.33±0.41%, in the periphery - 8.49±0.47% ($p<0.05$). The number of medium-sized hepatocytes on the periphery of the lobules

is 74.46±1.87%. Large hepatocytes in the center of the lobules are determined in the amount of 11.25±0.34%, and in the periphery - 15.14±0.34%. The number of binucleate cells is 21.73±0.42% of the total number of cells in the center of the lobules.

Leukocyte infiltration decreases to 12.43±0.19 cells per 10,000 μm2. Mononuclear cells and fibroblasts predominate in the infiltrates. A significant number of new foci of connective tissue are revealed, replacing necrotic areas. Fibrosis liver parenchyma not only leads to disruption of the structure and architectonics of the hepatic lobules, but also disrupts the blood supply to hepatocytes, complicating the processes of bile formation and bile excretion. The amount of collagen fibers reaches 8.37±0.08% ($p<0.05$).

The volume fraction of vessels is 17.45±0.21%. Glycogen content remains low - 1.02±0.01 units. ($p<0.05$), which is 2.98 times less than the norm.

A study of the histostructure of the liver in case of poisoning with acetic acid at the stage of anxiety-stress reaction showed that when toxic and painful stress is superimposed on the primary stress alteration, there is a significant increase in the volume of destructive disorders in the organ. The alarm stage is prolonged, and in the resistance stage the regeneration processes slow down, as a result of which by 14 days after poisoning the number of cells in a state of degeneration and foci of necrotic hepatocytes remains 3.53 times higher than normal.

In animals with poisoning at the stage of exhaustion of the stress response, already on the 1st day of the study, the volume fraction of necrosis increases to 21.52±0.12% of tissue volume ($p<0.05$). The number of cells in a state of balloon and vacuolar degeneration is 74.65±1.14% ($p<0.01$), which indicates increased organ damage under the influence of chronic stress and secondary toxic and nociceptive factors.

The plethora of blood vessels significantly exceeds that in the previous series of the experiment. It is accompanied by hemorrhages into the surrounding tissues. The diameter of the vessels is significantly increased. In the lumen of the vessels, destroyed endothelial cells and aggregates of erythrocytes are determined. Leukocyte infiltration increases to 36.75±0.47 cells per 10,000 μm2 ($p<0.05$) and is represented predominantly by macrophages.

The liver lobules are deformed not only due to fibrosis, which developed in the pathogenesis of chronic stress, but also as a result of dystrophic changes in hepatocytes due to the action of a toxic substance on the animal's body.

The volume fraction of small hepatocytes in the center of the lobules is 28.15±1.83%, in the periphery - 21.34±1.12% ($p<0.05$). The number of medium-sized hepatocytes in the center is determined to be 35.21±1.46%, and in the periphery - 45.67±0.65% ($p<0.05$). The volume fraction of large hepatocytes in the center of the lobules is 38.75±1.36%, in the periphery - 33.13±0.74% ($p<0.05$). The volume fraction of binucleate hepatocytes in the center of the lobules decreases to 4.82±0.15%, and at the periphery to 7.36±0.32% of the total number of cells ($p<0.05$), which indicates a sharp decrease in regeneration processes.

The amount of glycogen in hepatocytes is reduced 19 times compared to the norm and amounts to 0.16±0.01 units. ($p<0.05$), which indicates a sharp energy deficiency and inhibition of the synthetic functions of the liver.

By day 14, the degree of damage to the liver parenchyma decreases, but the proportion of hepatocytes in a state of dystrophy still remains high - 32.03±0.25%, and foci of necrosis - 2.97±0.03% ($p<0.05$).

The volume fraction of blood vessels decreases to 30.54±0.84% ($p<0.05$). The structure of the endothelium is restored, but isolated diapedetic hemorrhages from small vessels are still observed. Macrophages are detected around the remaining foci of necrosis. Leukocyte infiltration is 26.56±0.15 cells per 10,000 μm2 ($p<0.05$).

The number of small hepatocytes in the center of the lobules continues to decrease and amounts to 4.14±0.13%, at the periphery their number increases to 19.18±0.76% ($p<0.05$).

The volume fraction of medium-sized cells in the center of the lobules is 77.41±1.28%, at the periphery 64.83±0.65% ($p<0.05$). The volume fraction of large cells is 18.55±0.98% in the center of the lobules, and 16.13±0.34% at the periphery ($p<0.05$). The number of binucleate hepatocytes increases in the center of the lobules to 17.64±0.26%, and at the periphery to 18.82±0.14% ($p<0.05$).

The amount of glycogen is 30.43±0.91% of normal values ($p<0.05$).

A study of the histostructure of the liver of animals poisoned with acetic acid at the stage of exhaustion of the stress reaction showed that the destructive processes are of a pronounced protracted nature. Reparative and synthetic processes are reduced until the end of the observation period. Replacement of large areas of necrosis with connective tissue leads to gross destructive and functional disorders.

Thus, the study of the histological structure of the liver in poisoning with acetic acid showed that in case of suicidal poisoning, more pronounced destructive processes of the liver parenchyma and stroma are observed, which requires additional measures in the complex treatment of this pathology.

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