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ASSESSMENT OF FEATURES OF MORPHOMETRIC STRUCTURAL CHANGES IN THE TESTES AT CONDITIONS OF POSTRESECTION PORTAL HYPERTENSION

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Abstracts

Introduction. Postresection portal hypertension leads to complex general biological processes that occur and develop in the organs and systems of the body during its adaptation to a new level of life [10]. It should be noted that detailed and objective knowledge of compensatory-adaptive processes in the testes during resection of various volumes of the liver, their role in the development of organ failure to date have been insufficiently studied and need to be addressed.

Objective of the research: to study morphometric structural changes of testes in the conditions of postresection portal hypertension and multiorgan failure.

Materials of the research and their discussion. The testes of 40 white male rats were divided into 3 groups studied morphologically. Group 1 consisted of 12 intact animals, 2 - 18 rats with postresection portal hypertension, 3 - 10 animals with postresection portal hypertension and poliorganic failure. Euthanasia of rats was performed by bloodletting under

thiopental anesthesia a month after the start of the experiment. Micropreparations from the testes stained hematoxylin-eosin, toluidine blue, using the Weigert method, van Gieson, Mallory, Morphometrically, on the testicular micropreparations, the diameters of the testicles tubules, the thickness of their walls, the number of cells of the epithelio-spermatogenic layer in the tubule, the number of Sertoli cells per tubule, the tubulo-interstitial index, the stromal-parenchymal ratio, the Leydigas index, the sperm index, were determined. Quantitative morphological parameters were processed statistically.

Results of the research and their discussion. It was found that the investigated morphometric indices of the testes in the 2 and 3 groups of observations varied markedly in comparison with the control values. Thus, the diameter of the seminile tubules in the conditions of postresection portal hypertension was statistically significantly ($p<0.001$) decreased by 11.8 %, with the development of multiple organ failure - by 34.0 % ($p<0.001$), and the number of cells of the epithelio-spermatogenic layer in tubules respectively by 11.3 % and 32.9 % ($p<0.001$), which indicated the presence of atrophy of the studied structures.

The wall thickness of the tubules in the 2 group increased by 8.8 %, in the 3 group - by 26.9 % ($p<0.001$), and the Leydig index - by 22.9 % and 57.8 %, respectively ($p<0.001$). The number of Sertoli cells in one testicle tubule in the 2nd group decreased by 1.6 % and in the 3rd by 8.06 % ($p <0.001$).

Stromal-parenchymal ratios in the testes with simulated pathology statistically significantly increased ($p<0.001$) by 11.3 % ($p<0.01$) and 31.8 % ($p<0.001$), while the tubulo-interstitial index decreased respectively by 8.9 % ($p<0.001$) and 31.8 % ($p<0.001$). The revealed changes of the investigated morphometric parameters showed a pronounced increase in the number of stroma in the testes. The spermatogenesis intensity index also decreased by 2.9 % ($p<0.05$) and 38.8 % ($p<0.001$), respectively.

Obtained morphometric parameters indicate that the simulated pathology leads to a decrease in the diameter of the tubules and the thickening of their walls. In the latter, optically sclerosis was observed. In the lumen of the seminiferous tubules, histologic examination revealed spermatocytes of the first order and spermatogonia, rarely observed spermatocytes of the second order. Protein detritus and rarely sperm were detected in the tubules.

Conclusions. So postresection portal hypertension and multiorgan failure lead to pronounced structural restructuring of the vascular, interstitial and endocrine components of the testes, characterized by dilatation, plethora, varicose extensions, stasis, hemorrhage, perivasal swelling of the vessels gemomicrocirculatory bad and, hyperplasia, moderate

hypertrophy Leydig cell dystrophy. The detected morphological changes dominate in multiple organ failure.

Key words: testes; portal hypertension; multiple organ failure; morphometry.

Introduction. Liver resection is often performed today in modern surgical clinics for benign and malignant tumors, metastases, liver injuries, intrahepatic cholangiolithiasis, alveolar echinococcosis, liver transplantation [1, 2, 3]. Resection of large volumes of liver can lead to various postresection complications: bleeding from varicose veins of the esophagus, stomach, rectum, ascites, splenomegaly, secondary hypersplenism, parenchymal jaundice, portosystemic encephalopathy, liver failure, hypertension, portal hypertension. Postresection portal hypertension leads to structural rearrangement of the organs of the portal hepatic and inferior vena cava, as well as remodeling of their structures. The testes belong to the organs from which venous drainage is carried out through the inferior vena cava and hemodynamic disorders in them are complicated by various morphological changes in the vessels and structures of these organs. It should be noted that the features of remodeling of testicular structures in postresection portal hypertension have not been studied enough [3, 4, 5].

Objective of the research: to study morphometric structural changes of testes in the conditions of postresection portal hypertension and multiorgan failure.

Materials of the research and their discussion. The testes of 40 laboratory adult white male rats, which were divided into 3 groups, were examined by a set of morphological methods. The 1st group consisted of 12 intact animals, 2 - 18 rats with postresection portal hypertension, 3 - 10 animals with postresection portal hypertension and multiorgan failure.

Postresection portal hypertension was modeled by removal of the left and right lateral lobes of the liver [6]. In 10 animals after this surgery developed multiple organ failure. Euthanasia of rats was performed by bloodletting under conditions of thiopental anesthesia one month after the start of the experiment. Pieces were cut from the testes, which were fixed in a 10 % solution of formalin, passed through ethyl alcohols of increasing concentration and placed in paraffin. Microtome sections after dewaxing were stained with hematoxylin-eosin, according to the method of Weigert, van Gizon, Mallory, toluidine blue [7].

Morphometric methods on testicular micropreparations determined the diameters of seminal tubules (DST), their wall thickness (WT), the number of cells of the epithelial-spermatogenic layer (ESL) in the tubule, the number of Sertoli cells (SC) per tubule, tubulo-interstitial index - TI (the ratio of the area of the seminal tubules to the interstitial tissue), stromal-parenchymal relations (SPR), Leydig index (LI) - the relative number of Leydig cells

surrounding the seminiferous tubule, the index of spermatogenesis intensity (ISI) - the ratio of epithelial-spermatogenic cells to Leydig cells [8]. Quantitative morphological parameters were processed statistically. Processing of the latter was performed in the department of system statistical researches of Ternopil National Medical University named after I. Ya. Gorbachevsky Ministry of Health. The difference between comparative morphometric parameters was determined by the test of Student and Mann-Whitney [9, 10]. Experiments and euthanasia of experimental animals were performed in accordance with the "General Ethical Principles of Animal Experiments" approved by the First National Congress on Bioethics (Kyiv, 2001), in accordance with the European Convention for the Protection of Vertebrate Animals for Research and Other Scientific Purposes. also the Law of Ukraine "On protection of animals from cruel treatment" (from 21.02.2006) [11].

Results of the research and their discussion. It was found that resection of the left and right lateral lobes of the liver led to postresection portal hypertension, which was characterized by dilation of the portal hepatic vein, plethora and dilation of the mesenteric veins, visible venous bed of the small and large intestines, splenomegaly, ascites. The obtained morphometric parameters of testicular structures are shown in the table. A comprehensive analysis of the data in this table showed that the studied quantitative morphological parameters in the 2nd and 3rd groups of observations changed markedly compared with the control values.

Table – Morphometric characteristics of testes structures of experimental animals (M±m)

Indicator	Group of animals		
	1st	2nd	3rd
DST, μm	315,6±2,7	280,3±2,4***	208,3±1,8***
WT, μm	9,30±0,06	10,12±0,05***	11,80±0,09***
ESL	97,2±0,9	86,6±0,7***	65,2±0,7***
SC	6,20±0,05	6,10±0,05	5,70±0,04***
LI	8,30±0,05	10,20±0,06***	13,10±0,12***
SC	11,60±0,09	11,26±0,12*	7,10±0,06***
SPR	0,440±0,005	0,490±0,006**	0,580±0,007***
TI	0,560±0,004	0,510±0,004***	0,415±0,005***

Note. * - p<0,05; ** - p<0,01; *** - p< 0,001, compared with the 1st group

Thus, the diameter of the seminal tubules in postresection portal hypertension was statistically significantly (p<0,001) decreased from (315.6 ± 2.7) μm to (280.3 ± 2.4) μm, ie by 11.8 %, with the development of multiorgan failure - by 34.0 % (p<0.001), and the number

of cells of the epithelial-spermatogenic layer in the tubule, respectively - by 11.3 % ($p<0.001$) and 32.9 % ($p<0.001$), which showed on the presence of atrophy of the studied structures [8].

The wall thickness of the seminal tubules in the 2nd group of observations was increased by 8.8 %, in the 3rd group - by 26.9 % ($p<0.001$), and the Leydig index - by 22.9 % and 57, respectively, 8 % ($p<0.001$). The number of Sertoli cells in one seminal tubule of intact testes was equal to (6.2 ± 0.05), with postresection portal hypertension - (6.10 ± 0.05). The latter figure was reduced by 1.6%. With the development of multiorgan failure, the studied morphometric parameter with a pronounced statistically significant difference ($p<0,001$) decreased by 8.06 %.

Stromal-parenchymal ratios in the testes in the simulated pathology were statistically significantly increased ($p<0.001$) by 11.3 % ($p<0.01$) and 31.8 % ($p<0.001$), and the tubulo-interstitial index decreased respectively by 8.9 % ($p<0.001$) and by 31.8 % ($p<0.001$). The revealed changes of the studied morphometric parameters testified to a pronounced increase in the number of stroma in the testes [8]. The intensity index of spermatogenesis also decreased by 2.9 % ($p<0.05$) and 38.8 % ($p<0.001$), respectively.

The obtained morphometric parameters indicate that the simulated pathology leads to a decrease in the diameter of the seminal tubules and thickening of their walls. In the latter, optical sclerosis was observed optically. In the lumens of the seminiferous tubules on histological examination revealed spermatocytes of the first order and spermatogonia, rarely observed spermatocytes of the second order. Protein detritus and, rarely, spermatids were found in the tubules. There was plethora of mostly venous vessels. Plethora, dilatation, erythrostasis, varicose veins, sacculation, perivascular edema, sclerosis, foci of diapedetic hemorrhage are often found in the venous vessels of the hemomicrocirculatory tract of the testes. The walls of the arteries are thickened with the proliferation of endothelial cells, muscle cells and signs of sclerosis and narrowing of their lumen. In the interstitium, the phenomena of edema, sclerosing foci and foci of lymphoid cell infiltration. Leydig cells with signs of hyperplasia, moderate hypertrophy and dystrophy.

Studies and results show that in the simulated pathology, structural changes occur in the vascular, interstitial and endocrine components of the testes, which dominate in multiorgan failure. Detected morphological changes can lead to impaired spermatogenesis [8].

Conclusions. Postresection portal hypertension and multiorgan failure lead to a pronounced structural rearrangement of vascular, interstitial and endocrine components of the testes, which is characterized by dilatation, plethora, varicose veins, stasis, hemorrhage, translational edema, mainly venous vessels, hemomicrocirculator, hyperplasia, moderate

hypertrophy, Leydig cell dystrophy. The detected morphological changes dominate in multiorgan failure.

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