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MORPHOLOGICAL DISORDERS OF VASCULAR DISORDERS IN EXPERIMENTAL DIABETES MELLITUS

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Abstract: In this study, we examined the morphofunctional changes in the lower limb vessels of rats with experimental diabetes. The diabetes model was induced by streptozotocin administration, which led to the development of hyperglycemia in animals. Microscopic, histological, and X-ray vasographic studies were performed at different times during the experiment (5, 15, 30, 60, and 90 days). The results showed that diabetes causes destructive changes in the vascular walls, endothelial thinning, microcirculation disorders, and vascular wall sclerosis. These changes worsen over time, leading to deterioration of blood supply and the development of trophic ulcers on the limbs.

Keywords: diabetes mellitus, diabetic angiopathies, vascular changes, experimental diabetes mellitus, microcirculation, histological changes, streptozotocin, trophic ulcers, rats.

The high prevalence of diabetes mellitus (DM) is recognized as a non-infectious epidemic and is a serious medical and social problem. This is due to the severity of its course, a large number of complications (1, 2). According to WHO, there are currently more than 220 million patients with DM in the world (1), 10-20% of them are patients with type 1 DM (4). In 2005, DM caused the death of 1.1 million people worldwide (3), and in the period from 2005 to 2030, WHO experts expect. Diabetic angiopathies are the main manifestation of diabetes mellitus. They represent a generalized lesion of arterioles, capillaries and venules, thereby determining the clinical course and prognosis of the disease and are the most common cause of death. Over 2.7-4.5 million high amputations due to diabetic lesions of the lower extremities are performed annually worldwide. Microvascular complications characteristic of diabetes mellitus are realized by the development of endothelial dysfunction. Understanding the mechanisms of adverse changes occurring in the body in diabetes mellitus is an urgent problem of modern medicine. To develop correction methods that could neutralize the consequences of diabetes complications, it is necessary to know what mechanisms are violated.

The aim of the work was to study the morphofunctional disorders of the lower limb vessels of rats in experimental diabetes mellitus.

Material and methods of research. The object of the study were 90 white rats (males) of the Wistar line with an initial weight of 180 ± 2.64 g at the age of 4-6 months. The model of experimental diabetes mellitus was reproduced by a single intraperitoneal administration of streptozotocin in 0.1 M citrate buffer pH 4.5, to Wistar rats at a dose of 60 mg / kg. Blood glucose from the tail vein was determined by the glucose oxidase method. 21 rats died from the direct action of streptozotocin. Two animals were not sensitive. Only rats with elevated glucose levels (> 11 mmol/l) were used for further research. The rats were killed 5, 15, 30, 60, 90 days after the beginning of the experiment. To study the pancreas and vessels of the hind limbs in both intact animals and ESD rats, staining of histological preparations with hematoxylin and eosin, according to Van Gieson and Weigert was used. As well as X-ray

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vascularography. For mathematical data processing, Microsoft Excel 2010 applications were used in the section on descriptive statistics, determining standard deviations and comparing samples with determining the arithmetic mean M, the average error of relative values m and the reliability coefficient of the difference t.

Research results and their discussion. Comparison of body weights of the experimental and control groups showed that at the beginning of the experiment, both groups did not have reliable differences. During the experiment, the weight of the rats in the experimental group remained 1.7 times greater than that in the control group. The growth rate in the control group was 50%, while in the rats in the experimental group it was 21%. The development of experimental diabetes mellitus in the experimental animals was accompanied by persistent hyperglycemia. The blood glucose level in rats with diabetes increased to 19.4±4.3 mmol/l compared to 5.2±1.1 mmol/l in the control group. The blood glucose level in rats with EDM on the 5th day after streptozotocin administration was significantly 3.2 times greater than that in the control group. During the following days of observation, the blood sugar level in the rats in the experimental group also remained stably high – 15.9 mmol/l, remaining virtually unchanged (±0.38 mmol/l) compared to the 5th day of the study. Moreover, the fasting glycemia level reached its maximum value by the 30th day of the study and amounted to 19.4 mmol/l.

Microscopic examination of the pancreas of experimental rats revealed degenerative and destructive changes in its tissue, especially in the endocrine part – the islets of Langerhans. Already on the 5th day of ESD, edema of the interlobular connective tissue was observed. On the 60th day of the experiment, necrotic changes in β-cells were noted, which intensified by the 90th day of the experiment. At all stages of the experiment, moderate lymphocytic infiltration was noted in the pancreatic islets. The capillaries of the islets were sharply full-blooded, endocrine cells located in the central zones were necrotic, and those located in the peripheral parts of the islet were hypertrophied. The volume fraction of islets located in the intestinal, gastric and splenic zones decreased compared to the rats of the control group. Insulin-positive cells were located singly or in small clusters in the central parts of the islets around the full-blooded capillaries. There was a significant decrease in the area occupied by endocrinocytes in all zones of the pancreas compared to the control group of animals.

X-ray vasography data showed that all animals of the experimental group showed a noticeable expansion of intramuscular arterial vessels in the muscles of the lower leg and skin within 5 days after the experimental modeling of diabetes mellitus. Thus, in the early stages (5-15 days) of observation, inflammatory-destructive changes progress in combination with unexpressed atrophic processes.

Morphological studies of vessels at various stages of postnatal ontogenesis showed that, compared to the control group, changes in the form of delayed development and formation of individual components of the vascular wall. Destructive changes in the arterial wall were noted by us in all experimental animals from the first days after the experiment. They were characterized by thinning of the wall and expansion of the lumen of the vessel, rare location of endothelial nuclei, desquamation of individual endothelial cells in the lumen of the vessel. The muscular layer is stretched, consists of 1- rows of cells. Fragmentation of the internal

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elastic membrane is also noted. On the 30th day of the experiment, sclerotic and destructive changes predominate in the walls of microvessels. The wall of arterioles is thickened as a result of an increase in the basal membrane and wide surrounding connective tissue. In the middle shell of intramuscular vessels, a network of thin fibers is revealed, connecting the internal and external elastic membranes. Fragments of elastic membranes are replenished with new elastic elements. The membranes themselves are somewhat thickened. The wall of venules is also thickened and deformed due to hyperchromasia of endothelial cells and the basal membrane. On the 60th day of postnatal ontogenesis, morphological changes in the walls of vessels acquire a chronic course and are manifested by sclerotic and degenerative changes. The endothelial layer of the intima is represented by flattened cells, in others it forms a layering and a significant protrusion towards the lumen of the vessel. The basement membrane is tortuous, unevenly thickened and intensely stained with eosin, in places due to thin and merges with fibrous structures of the interstitial connective tissue. In the late stages of the experiment, hair loss and desquamation of the epidermis are observed on the foot of the limb. On the 90th day after the beginning of the experiment, trophic ulcers of various sizes appeared in the heel area or on the dorsum of the foot and toes. Histological and histochemical studies show that all experimental animals from the first days after ESD have destructive changes in the walls of intramuscular vessels. The vessels are filled with blood, their walls are thinned, the lumen is dilated. Many endothelial cells are swollen, the cell nuclei are rarely located, some of them desquamate into the lumen of the vessel. The muscular layer of the vessels is stretched and consists of 1-2 rows of cells. The internal elastic membrane is thinned and fragmented in places. Moreover, in the animals in the initial days of the experiment, destructive changes in the walls of the intramuscular vessels are less pronounced. The PAS reaction in the animals of the experimental group is positive. Especially in 30- and 90-day-old animals of the experimental groups it is sharply positive. In the subsequent periods (up to 30-60 days), the above-mentioned vascular and tissue changes progress. An increase in the number of spastically narrowed vessels is observed. Few- and avascular zones, blind-ending capillaries are often encountered, especially in areas subject to atrophic changes. However, it should be noted that congestion is noticeably expressed in the venous bed. Such a picture of hemodynamic disorders leads to pronounced morphological changes in tissue structures.

Thus, the obtained results indicate that diabetes mellitus type 1 leads to changes in the vessels and muscles of the extremities. At the stage of diabetes mellitus development studied by us, functional and at subsequent stages of the experiment, structural changes associated with tissue metabolism disorders are observed.

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