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Morphological Changes In The Walls Of Foot Veins Under The Influence Of Experimental Diabetes

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Abstract: A total of 75 white rats aged 4-6 months were used as the object of study. The morphological changes in the walls of the pancreatic and foot blood vessels of rats under the influence of experimental diabetes were investigated. Comparison of the body weight between the control and experimental groups revealed that during the experiment, the weight gain in the experimental group was 1.6 times lower than that in the control group. Moderate lymphocytic infiltration was observed in the Langerhans islets of the pancreas at all stages. Morphological studies at various stages of postnatal ontogenesis showed that, compared to the control group, the experimental group exhibited delayed development and formation of vascular wall components, as well as destructive changes. Vascular wall damage in the arteries was detected from the early days of the experiment.

Keywords: Diabetes mellitus, alloxan, pancreas, blood vessels.

Introduction: Diabetes mellitus (DM) is recognized as a non-infectious epidemic and represents one of the most serious medical and social problems. This is primarily explained by the severity of the disease and the high number of its complications [1, 2]. According to the World Health Organization (WHO), more than 220 million people worldwide currently suffer from diabetes mellitus, and 10–20% of them have type 1 diabetes [4].

In 2005, diabetes mellitus caused the death of 1.1 million people globally [3], and WHO experts predict that this figure may continue to increase between 2005 and 2030.

Diabetic angiopathies are among the main manifestations of diabetes mellitus. They are characterized by generalized damage to arterioles, capillaries, and venules, which determines the clinical course and prognosis of the disease, as well as being one of the leading causes of mortality.

Globally, 2.7–4.5 million high-level amputations of limbs are performed annually due to diabetes-related complications. Microvascular complications typical of diabetes develop as a result of endothelial dysfunction. Understanding the mechanisms of pathological changes occurring in the body during diabetes mellitus remains a pressing issue of modern medicine.

To develop corrective methods that can alleviate or reduce the consequences of diabetic complications, it is essential to identify the mechanisms involved in their pathogenesis.

Purpose of the Experiment. The main objective of this study was to investigate the morphofunctional alterations in the pancreas and foot vessels of rats under experimental diabetes mellitus conditions.

METHODS

The study was conducted on 60 white Wistar rats aged 4–6 months, weighing 180 ± 2.00 g. The model of experimental diabetes mellitus was induced by a single intraperitoneal injection of alloxan at a dose of 35 mg/kg. Blood glucose levels were determined in samples taken from the tail vein using the glucose oxidase method. As a result of the direct toxic effect of alloxan, 4 rats died, and 2 were resistant to its action. Further investigations were carried out only on rats with blood glucose levels above 10 mmol/L.

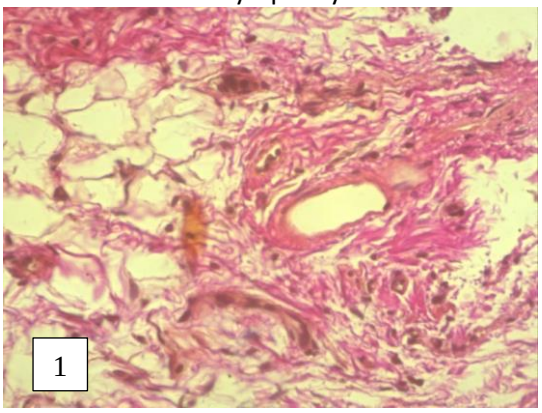
The animals were sacrificed after 14, 30, 45, and 60 days. The pancreas and foot blood vessels were examined histologically using hematoxylin-eosin, Van Gieson, and Weigert stains, and vascular radiography was performed. The obtained data were processed statistically using Microsoft Excel 2010 software.

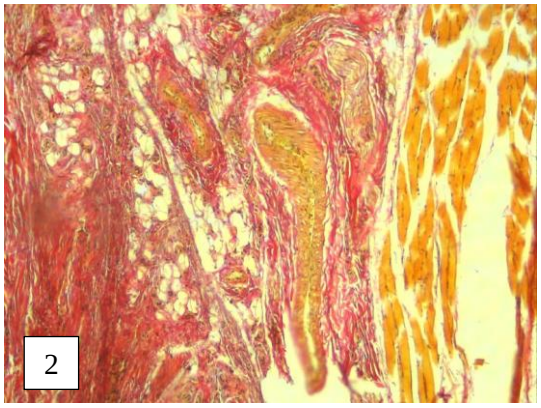
RESULTS AND DISCUSSION

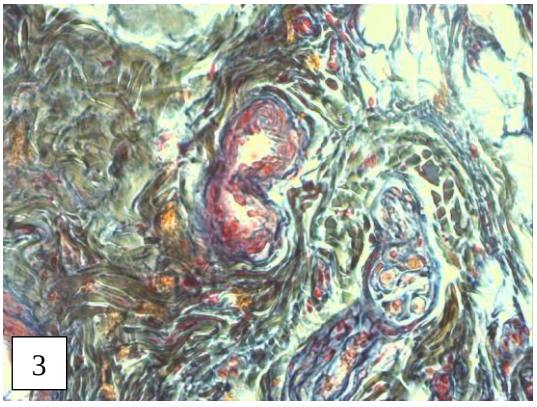
There was no significant difference in the initial body weight of the rats in the control and experimental groups. However, during the experiment, the body weight gain in the diabetic rats was 1.6 times lower compared to the control group. The growth rate in the control group was 50%, while in the experimental group, it was only 21%. The development of experimental diabetes mellitus in rats was accompanied by persistent hyperglycemia. The blood glucose level in the control group was 5.3 ± 1.1 mmol/L, while in diabetic rats it increased up to 18.8 ± 4.2 mmol/L. Five days after alloxan administration, the glucose level in the experimental group was 3.1 times higher than that of the control group, and it remained consistently elevated (around 15.8 mmol/L) during the following days.

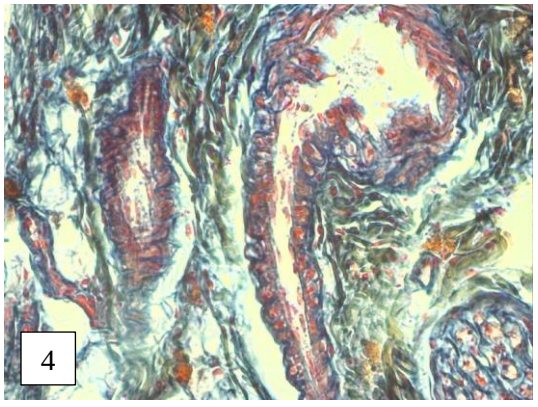
Microscopic analysis revealed degenerative and destructive changes in the pancreatic tissue, particularly in the Langerhans islets. As early as day 5, edema of the interlobular connective tissue was observed. By days 45–60, necrosis of β-cells had intensified. Moderate lymphocytic infiltration was noted in the islets at all time points. Capillaries were fully blood-filled; endocrine cells in the central zone showed necrosis, while those in the peripheral zone were hypertrophied. The islet area was reduced by 24% compared to the control group.

Radiovazographic analysis demonstrated that five days after the onset of the experiment, arterial vessels in the muscles and skin of the rats' feet were dilated. Between days 5 and 15, inflammatory and atrophic processes developed in the vascular wall. Morphological studies at different stages of postnatal ontogenesis showed delayed development and formation of vascular wall components in the experimental group. These changes were characterized by thinning of the arterial wall, dilation of the vascular lumen, reduced number of endothelial nuclei, and desquamation of some endothelial cells into the vessel lumen. The muscular layer appeared stretched and consisted of only 1–2 rows of cells. Moreover, the internal elastic membrane was fragmented and partially disintegrated.









Morphological appearance of the venous vessels of the rat foot during different stages of the experiment. Observation day 45. 1. H&E stain, 2. Van Gieson stain, 3–4. Micrographs. Desquamation of the endothelial layer of venous vessels.

By day 30 of the experiment, sclerotic and destructive changes predominated in the microvessels: the arterial wall became thickened, the basal membrane expanded, and there was an intensive growth of fibers between the internal and external elastic membranes in the muscular layer. Some regions of the elastic membranes were supplemented with newly formed elastic elements, and the membranes themselves

appeared slightly thickened.

The venous vessel walls also showed thickening and deformation, associated with hyperchromatosis of endothelial cells and alterations in the basal membrane.

By day 45 of the experiment, the morphological changes became chronic in nature. The endothelial lining consisted of flattened cells, and in some areas, these cells protruded into the vessel lumen. The basal membrane was unevenly thickened and strongly eosinophilic. In the later stages, hair loss and epidermal desquamation were observed on the skin of the rats' feet. By day 60, trophic ulcers appeared on the heels and toes.

Histological and histochemical analyses revealed that destructive alterations in the vascular wall occurred from the early days of the experiment. The vessels were blood-filled, with thin walls and dilated lumens. Endothelial cells were swollen, and some had detached into the vascular lumen. The internal elastic membrane appeared fragmented in some areas. The muscular layer was stretched, consisting of only one to two rows of cells, and the internal elastic membrane was thinned and fragmented. In the early days of the experiment, these changes were less pronounced.

According to histochemical studies, the PAS reaction showed a strongly positive result in the 30- and 45-day experimental rats. After 30–60 days, the above changes became even more pronounced: the number of spastically contracted vessels increased, and in some areas, vessels disappeared or formed incomplete capillary loops. In the venous system, blood stagnation (venous congestion) became more intense. Such hemodynamic disturbances led to distinct morphological alterations in tissue structures.

CONCLUSION

The obtained results demonstrate that type 1 diabetes mellitus causes significant morphological alterations in the pancreas and the foot blood vessels. Functional changes were observed during the early stages of the experiment, whereas at later stages, structural disturbances associated with impaired tissue metabolism developed.

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