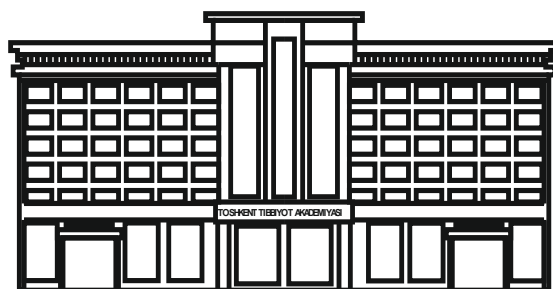


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**MANIFESTATIONS OF MORPHOLOGICAL CHANGES IN EXPERIMENTAL DIABETES MELLITUS**

Niyazov N.K., Akhmedova S.M., Nisanbaeva A.U.

**ПРОЯВЛЕНИЯ МОРФОЛОГИЧЕСКИХ ИЗМЕНЕНИЙ ПРИ ЭКСПЕРИМЕНТАЛЬНОМ САХАРНОМ ДИАБЕТЕ**

Ниёзов Н.К., Ахмедова С.М., Нисанбаева А.У.

**EKSPERIMENTAL QANDLI DIABETDA MORFOLOGIK O'ZGARISHLARNING NAMOYON BO'LISHI**

Niyozov N.K., Axmedova S.M., Nisanbaeva A.U.

*Tashkent Medical Academy*

**Цель:** изучение морфофункциональных нарушений поджелудочной железы крыс при экспериментальном сахарном диабете. **Материал и методы:** эксперименты проведены на 4-6-месячных белых крысах породы Wistar. Модель экспериментального сахарного диабета воспроизводили однократным интраперитонеальным введением стрептозотоцина в дозе 60 мг/кг. Изучены морфологические изменения поджелудочной железы. **Результаты:** в течение эксперимента масса крыс экспериментальной группы была меньше контроля в 1,7 раза. Во всех сроках эксперимента в панкреатических островках отмечалась умеренная лимфоцитарная инфильтрация. Изменения в виде задержки развития и формирования отдельных компонентов стенки сосудов по сравнению с контролем наблюдались в различные сроки постнатального онтогенеза. Деструктивные изменения в стенке артерий у всех подопытных животных регистрировались с первых дней эксперимента. **Выводы:** сахарный диабет 1-го типа приводит к структурным изменениям поджелудочной железы.

**Ключевые слова:** сахарный диабет, поджелудочная железа, сосуды.

**Maqsad:** tadqiqot ishi qandli diabetda kalamushlarning pastki oyoq-qo'llarining oshqozon osti bezining morfo-funksional buzilishlarini o'rganish. **Material va usullar:** tajribalar 90 ta 4-6 oylik Wistar oq kalamushlarda o'tkazildi. Eksperimental qandli diabet modeli 60 mg/kg dozada streptozotosinning bir marta qorin bo'shlig'iga kiritilishi bilan chaqirildi. Oshqozon osti bezi devorlaridagi morfologik o'zgarishlar o'rganildi. **Natijalar:** tajriba davomida eksperimental guruh kalamushlarining vazni nazoratdan 1,7 baravar kam bo'lgan. Tajribaning barcha davrlarida oshqozon osti bezi orollarida o'rtacha limfotsitar infiltratsiya qayd etilgan. Nazorat bilan solishtirganda qon tomir devorining individual tarkibiy qismlarining rivojlanishi va shakllanishining kechikishi shaklidagi o'zgarishlar postnatal ontogenezning turli vaqtlarida kuzatilgan. Barcha eksperimental hayvonlarda arteriyalar devoridagi halokatli o'zgarishlar tajribaning birinchi kunlaridan qayd etilgan. **Xulosa:** 1-toifa diabet me'da osti bezining qon tomirlarida o'zgarishlarga olib keladi.

**Kalit so'zlar:** qandli diabet, oshqozon osti bezi, qon tomirlari.

The high prevalence of diabetes mellitus (DM) is recognized as a non-communicable epidemic and is a serious medical and social problem. This is due to the severity of its course, a large number of complications [1,5,6]. According to the WHO, there are currently more than 220 million patients with diabetes in the world [2], 10-20% of them are patients with type 1 diabetes [2,4]. In 2005, DM caused 1.1 million deaths worldwide [2], and between 2005 and 2030, WHO experts expect. Diabetes mellitus is a systemic metabolic disease that has assumed a pandemic nature of distribution, which prompted the United Nations in December 2006 to adopt a resolution calling for "creating national programs for the prevention, treatment and prevention of diabetes and its complications and including them in the state programs for healthcare." Diabetic angiopathy is 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 are performed annually in the world for diabetic lesions of the lower extremities. Microvascular complications characteristic of diabetes mellitus are realized through the development of endothelial dysfunction.

Understanding the mechanisms of adverse changes that occur in the body in diabetes mellitus is an urgent problem of modern medicine. To develop methods of correction that could mitigate the consequences of complications of DM, it is necessary to know what mechanisms are violated in this case.

This data shows the study of structural changes in pancreatic signs in diabetes mellitus.

**Objective:** To study morphofunctional disorders of the pancreas and vessels of the lower limb of rats in experimental diabetes mellitus.

**Materials and research methods**

The object of the study were 90 white rats (males) of the Wistar line with an initial weight of  $180 \pm 2.64$  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. Determination of blood glucose from the tail vein was performed by the glucose oxidase method. From the direct action of streptozotocin, 3 rats died. 2 animals were not sensitive. Only rats with elevated glucose levels ( $>11$  mmol/l) were used for further study. The rats were slaughtered 5,15,30,60,90 days after the start of the experiment. To study the pancreas and vessels of the hind limbs, both in

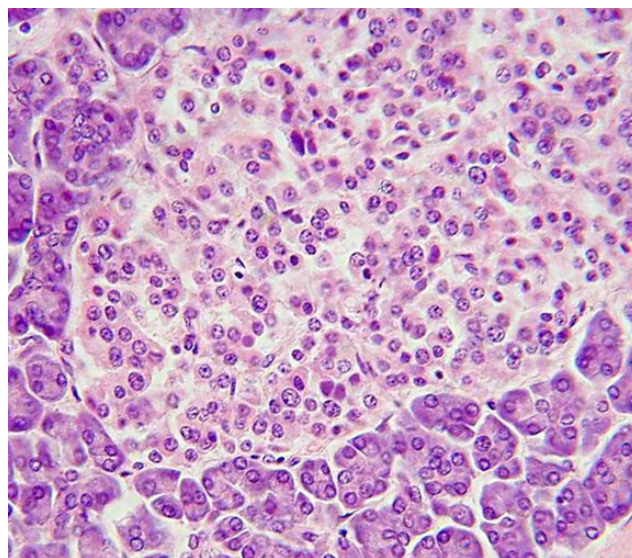
intact animals and in ESD rats, histological preparations were stained with hematoxylin and eosin, according to Van Gieson and Weigert. As well as x-rays of blood vessels. For mathematical data processing, Microsoft Excel 2010 applications were used in the section of descriptive statistics, determination of standard deviations and comparison of samples with the determination of the arithmetic mean  $M$ , the average error of relative values  $m$  and the coefficient of reliability of the difference

### Results and their discussion

Comparison of the body weight of the experimental and control groups showed that at the beginning of the experiment there was no significant difference in both groups. During the experiment, the mass of rats of the experimental group remained in development compared to the control group by 1.7 times. At the same time, the growth rate of the control group was 50%, and in the rat pups of the experimental group it was 21%. The development of experimental diabetes mellitus in experimental animals was accompanied by persistent hyperglycemia. The level of glucose in the blood of rats with DM increased to  $19.4 \pm 4.3$  mmol/l compared with  $5.2 \pm 1.1$  mmol/l in the control group. The blood glucose level in rats with ESD on the 5th day after administration of streptozotocin was significantly higher by 3.2 times when compared with animals in the control group. During the following days of observation, the amount of sugar in the blood of rats of the experimental group of animals also remained stably high - 15.9 mmol/l, practically unchanged ( $\pm 0.38$  mmol/l) compared with the 5th day of the study. At the same time, the level of fasting glycemia reached its maximum value by the 30th day of the study and amounted to 19.4 mmol/l.

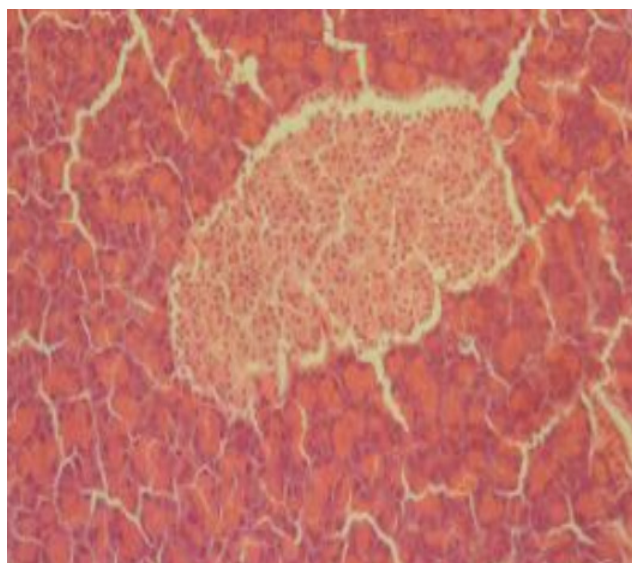
Microscopic examination of the pancreas experimental rats established degenerative and destructive changes in its tissue, especially the endocrine part - the islets of Langerhans. Already on the 5th day of ESD, edema of the interlobular connective tissue was observed. On the 60th day of the experiment, necrotic changes in  $\beta$ -cells were noted, which increased by the 90th day of the experiment. In all periods of the experiment, moderate lymphocytic infiltration was noted in the pancreatic islets. The capillaries of the islets were sharply plethoric, the endocrinocytes located in the central zones were necrotic, and those located in the peripheral parts of the islet were hypertrophied. The volume fraction of islets decreased by 25% in comparison with rats in the control group. Insulin-positive cells were located singly or in the form of small clusters in the central sections of the islets around full-blooded capillaries. There was a significant decrease in the area occupied by endocrinocytes in all areas of the pancreas compared with the control group of animals. X-ray vasography data established that in all animals of the experimental group within 5 days after the experimental modeling of diabetes mellitus, there was a noticeable expansion of intramuscular arterial vessels in the muscles of the leg and skin. So, in the early stages (5-15 days) of observation, inflammatory-destructive changes progress in combination with unexpressed atrophic processes. Morphological studies of vessels at different times of postnatal ontogene-

sis showed that, compared with the control group, there were changes in the form of developmental delay and the formation of individual components of the vessel wall (Fig. 1). We noted destructive changes in the arterial wall in all experimental animals from the first days after the experiment.



**Fig. 1. Photomicrograph of the islets of Langerhans Islets of Langerhans stained with hematoxylin-eosin.**

They were characterized by thinning of the wall and expansion of the lumen of the vessel, a rare arrangement of endothelial nuclei, and desquamation of individual endothelial cells into the lumen of the vessel. The muscle layer is stretched, stands for 1 rows of cells. Fragmentation of the internal elastic membrane is also noted. On the 30th day of the experiment, sclerotic and destructive changes predominate in the walls of microvessels (Fig. 2).



**Fig. 2. Photomicrograph of the islets of Langerhans Staining with hematoxylin and eosin.  $\times 100$ . Hypertrophied OL, consisting of accumulations of a large number of endocrinocytes.**

The wall of the arterioles is thickened as a result of an increase in the basement membrane. Microscopic examination of the pancreas experimental rats established

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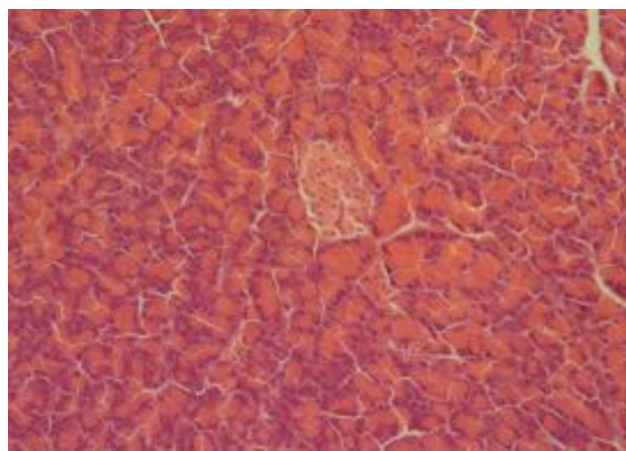
X-ray vasography data established that in all animals of the experimental group within 5 days after the experimental modeling of diabetes mellitus, there was a noticeable expansion of intramuscular arterial vessels in the muscles of the leg and skin. So, in the early stages (5-15 days) of observation,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

Morphological studies of vessels at different times of postnatal ontogenesis showed that, compared with the control group, there were changes in the form of developmental delay and the formation of individual components of the vessel wall. We noted destructive changes in the arterial wall 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, a rare arrangement of endothelial nuclei, and desquamation of individual endothelial cells into the lumen of the vessel. The muscle layer is stretched, stands for 1 rows of cells. Fragmentation of the internal 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 the arterioles is thickened as a result of an increase in the basement membrane and wide surrounding connective tissue. In the middle shell of intramuscular vessels, a network of thin fibers connecting the inner and outer elastic membranes is revealed. Fragments of elastic membranes are replenished with new elastic elements. The membranes themselves thicken somewhat. The wall of venules is also thickened and deformed due to hyperchromasia of endothelial cells and basement membrane.

On the 60th day of postnatal ontogenesis, morphological changes in the walls of blood 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 layer and a significant protrusion towards the lumen of the vessel. The basement membrane is tortuous, unevenly thickened and intensely stained with eosin, in some places due to thin and merges with the fibrous structures of the interstitial connective tissue. In the later

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 start 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 have destructive changes in the walls of intramuscular vessels from the first days after ESD. Blood-filling vessels, their walls are thinned, the lumen is expanded. Many endothelial cells are swollen, cell nuclei are rarely located, some of them desquamate into the lumen of the vessel. The muscular layer of the vessels is stretched, consists of 1-2 rows of cells. The internal elastic membrane is thinned and fragmented in places (Fig. 3). Moreover, in animals in the initial days of the experiment, destructive changes in the walls of intramuscular vessels are less pronounced. CHIC - reaction in animals of the experimental group is positive. Especially in 30 and 90 day old animals of the experimental groups, it is sharply positive. In subsequent periods (up to 30-60 days), the above vascular and tissue changes progress. There is an increase in the number of spasmodically constricted vessels. Often there are few and avascular zones, blind capillaries, especially in areas subject to atrophic changes. However, it should be noted that congestion in the venous bed is markedly pronounced. Such a picture of hemodynamic disorders leads to pronounced morphological changes in tissue structures.



**Fig. 3. Micrograph of the exocrine pancreas of rats. Stained with hematoxylin and eosin.  $\times 100$ . Acini are round or oval, inside lobules are densely packed. The cytoplasm of acinar cells is eosinophilic, the nuclei are shifted to the basal departments.**

Thus, the results obtained indicate that type 1 diabetes mellitus leads to changes in the vessels and muscles of the extremities. In the early period of the experiment that we studied, the development of diabetes mellitus leads to functional changes, and in the subsequent periods of the experiment, structural changes associated with impaired tissue metabolism.

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## MANIFESTATIONS OF MORPHOLOGICAL CHANGES IN EXPERIMENTAL DIABETES MELLITUS

Niyazov N.K., Akhmedova S.M.

**Objective:** To study morphofunctional disorders of the pancreas and vessels of the lower limb of rats in experimental diabetes mellitus. **Material and methods:** Experiments were carried out on 90 4-6 month old Wistar white rats. The model of experimental diabetes mellitus was reproduced by a single intraperitoneal administration of streptozotocin at a dose of 60 mg/kg. Morphological changes in the pancreas and lower vessel walls were studied. **Results:** During the experiment, the weight of the rats of the experimental group was 1.7 times less than the control. In all periods of the experiment, moderate lymphocytic infiltration was noted in the pancreatic islets. Changes in the form of delayed development and formation of individual components of the vascular wall compared with the control were observed at different times of postnatal ontogenesis. Destructive changes in the wall of arteries in all experimental animals were recorded from the first days of the experiment. **Conclusions:** Type 1 diabetes mellitus leads to changes in the pancreas and blood vessels of the extremities.

**Key words:** diabetes mellitus, pancreas, blood vessels.