SCIENTIFIC JUSTIFICATION OF MORPHOLOGICAL CHANGES IN THE LIVER DUE TO DIABETES

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Abstract: Diabetes is a malfunction of metabolic processes, which occurs due to a decrease in the level of insulin and an increase in the amount of sugar in the blood. The disease is chronic and there is often a risk of worsening. Conditions caused by diabetes can lead to death (that is hyperglycemic and hypoglycemic coma). According to statistics, diabetes is the second most common disease caused by metabolic disorders (obesity is in first place). Globally, diabetes is diagnosed in one tenth of the population. Considering that the disease can pass without symptoms, scientists assume that the percentage of patients with diabetes is actually much higher.

Keywords: liver, insulin, ketoacidosis, glucose

The purpose of the study: To study the dynamics of morphological changes in the liver of inbred white rats with diabetes in experimental studies

Relevance of the topic: It is known that diabetes mellitus can be divided into 2 types:

1. Insulin-dependent type I
2. Non-insulin-dependent type II

Type I diabetes begins when a person is young, during adolescence, when the function of the cells of the islet of Langerhans of the pancreas is impaired. As a result, the pancreatic ß-cells do not produce insulin, which leads to an increase of sugar in blood. This, in turn, affects the main organs of the human body, especially the liver.

In type II diabetes, pancreatic islet cells of Langerhans remain functional, but blood sugar levels increase. Due to this, there is a decrease in the activity of insulin-sensitive receptors in liver cells. In this case, acetone in the blood and excretion in the urine is almost not observed. The age of a person plays an important role in this.

Currently, as a result of scientific research, it is determined that insulin dependence (I-type) causes great changes in a person. If insulin is normal in the body, the work of organs responsible for carbohydrate metabolism is normal. In particular, as a result of lack of carbohydrates in the liver, the process of gluconeogenesis accelerates. The liver compensates for the lack of glucose in the body through gluconeogenesis. This process, in turn, increases the catabolism of proteins (amino acids with a glucose radical) and fats. This leads to a decrease in reserve substances in the body. Knowing that the body does not accumulate proteins as a reserve, the consumption of cell proteins leads to the occurrence of protein deficiency diseases.

In fat catabolism, the synthesis of ketone bodies in the cell increases as a result of the consumption of fats in the cells. This leads the body to "Ketoacidosis". Although ketoacidosis is common in diabetic patients, it is not the main cause of death today. In this high-density in the liver cells leads to a decrease of lipoproteins, which leads to an increase in the amount of

cholesterol in the liver. A disease called *cachexia* (extreme thinness) develops as a result of excessive breakdown of fats. In order to mobilize fats, it is necessary to have a normal glucose metabolism in the body. As a result of glucose non-absorption in patients with diabetes, ketone bodies formed from fats are not mobilized and accumulate in the body, and the patient's mouth constantly smells of acetone.

The more low-density lipoproteins accumulate in the hepatocytes of the liver, the greater the number of glycogen inputs in the cells. Complications lead to the onset of microangiopathy and atrophy in the transverse muscles.

Microangiopathy begins as a result of glucose reacting with the basement membrane in the blood vessels, swelling of the basement membrane cells. This leads to the development of microangiopathy in the small blood vessels between the muscles.

Research materials and methods: The study was conducted on 28 white male laboratory rats aged 1 to 6 months, with body weight 150-170 g. Rats were divided into 4 experimental groups (5 in each): 1-4 groups - rats in the period of diabetes were separated. Group 1 was monitored for 60 days, group 2 - 90 days, group 3 - 120 days, group 4 - 180 days. Each experimental group was divided into 2 to 4 control groups with no changes in morphological parameters.

The work was carried out on experimental material. 20 white male rats weighing 150-170 g were prepared for an experimental model of diabetes (D2 type) by intraperitoneal injection of a solution of alloxan tetrahydrate at doses of 150 and 250 micron per 1 kg of body weight. Alloxan was administered after a daily fast. Blood glucose level was determined beforehand. Blood was collected from the tail vein and glucose levels were determined on days 1, 60, 90, 120, and 160. Healthy rats were selected for the experiment.

The results obtained: In diabetes mellitus, gylcogen inclusions accumulate in hepatocytes of liver tissue in the form of neutral mucopolysaccharide, it has been detected that cell nuclei shift to the periphery, cells increase in volume, cytoplasm becomes cloudy. In intercellular connections, local destructions with foci in intercellular barriers, many sparse fibrous structures gathered around triads are detected. At the same time, hepatocytes with focal hydropic and hyaline drops are detected. In centrolobular hepatocytes: necrobiosis, paranecrosis (fatty dystrophy of various degrees) hepatocencides are detected. Sinusoidal spaces are of different widths, and it is determined that Kupffer cells migrate around deeply dystrophied hepatocytes. Hepatocytes with monocellular necrosis are detected in the focus.

It was found that the majority of perilobular hepatocytes suffered from hyaline droplet dystrophy, as a result of the sharp expansion of perilobular vein blood vessels, hepatocytes in this area underwent compression atrophy.

It was found that a lot of Kupffer cells were gathered around blood vessels in triads, and the process of necrosis and necrobiosis was increased in hepatocides of this area.

Full-blooded and focal diapedesis bleeding was found in most large-caliber venous blood vessels.

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