MORPHOLOGICAL CHANGES IN THE LIVER AT ALLOXAN-INDUCED DIABETES IN RATS

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The spread of diabetes in the world gradually reached epidemic proportions. According to forecasts, over the next 25 years the number of patients with diabetes will increase to 300 million. Diabetes mellitus is characterized by the destruction of β -cells by autoimmune attack. Lacks of the modern methods of treatment of diabetes and insulin using with transplantation of pancreatic tissue stimulates the development and improvement of cell therapy, including finding a new alternative sources of β -cells.

Diabetes mellitus is caused by a combination of genetic predisposition and autoimmune damaging of β -cells, leading to progressive insulin deficiency, up to a full stop of its production. In the body of patients with diabetes type I produced autoantibodies to various components of pancreatic islets, as well as insulin. Induced immune system destruction of pancreatic islets in humans conceptually divided into six stages, beginning with genetic susceptibility. In the second stage run the mechanisms that lead to further autoimmune attack on the β -cells. Similarly unknown the environmental factors that cause the autoimmune processes aimed at β -cells, but they can be drugs, infectious agents. Stage three - a period of active autoimmune processes, however, still remains normal insulin secretion. The immune attack on the β -cells continues on the fourth stage and leads to progressive lowering of stimulating action of glucose for insulin producing. In the fifth stage there are clinical signs of diabetes with residual insulin secretion. The sixth stage is characterized by the complete destruction of β -cells. As a result, the patient becomes dependent on insulin, which is needed to maintain the normal blood glucose levels, prevent ketoacidosis and sustainability. Based on the modern concept of diabetes, one can assume that dogs and cats this disease goes through the same stages as humans. Unfortunately, in animals usually can not detect the diabetes before 5 th or 6 th stage where insulin is absolutely necessary. Diabetes is accompanied by all kinds of metabolism disorders, which leads to the pathological changes in organs and body systems.

The aim of this study is to investigate the morphological changes in the liver of rats with experimental diabetes.

Model of an alloxan-induced diabetes accompanied by structural changes in the liver, characteristic of chronic hepatitis.

Development of an alloxan-induced diabetes accompanied by the following pathological changes in the liver: gantry and intralobulyar necrosis of hepatocytes, portal fibrosis with lymphocytic infiltration, dilatation and stagnant of blood in central and portal vein, violation of the structure of the hepatic trabeculae, dilatation and plethora of sinusoids, proliferation of hepatic ducts, polymorphism of hepatocytes.

In the pathogenesis of morphological changes in animals with an alloxan-induced diabetes leading pathogenetic factor is carbohydrate starvation of vascular endothelial cells and hepatocytes, which are most sensitive to energy starvation.