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ABSTRACT BOOK

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CHANGING THE CHEMICAL COMPOSITION OF LONG TUBULAR BONES OF RATS WITH DIABETES MELLITUS TYPE I

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Introduction: Diabetes mellitus (diabetes mellitus) is a group of metabolic diseases is determined by hyperglycemia, which is the result wrong insulin action, or two factors together. One of the chronic complications of diabetes is diabetic osteopathy, which pronounced from moderate osteoporosis to spontaneous fractures of the tubular bones. Metabolic changes that occurs in diabetes mellitus lead to a violation of the processes of remodeling of bone tissue. In condition shortage of insulin the activity of osteoblasts decreases, and the osteoclast activity, on the contrary, increases due to this bone reabsorption intensifies. With diabetes, the processes of demineralization of bones are clearly expressed. At the diabetes, the processes of demineralization of bones are clearly expressed.

Aim: The purpose:to explore the changes in the chemical composition of long tubular bones in aloxans-induced rats in native diabetic ranges.

Materials and methods: The determination of the chemical composition of the tubular bones was carried out using the atomic absorption method. Study of changes in the chemical composition of long tubular bones was performed on 40 white laboratory rats in mature age. The experiment lasts 30 days. For study, thighs and the shoulder bones of animals were modeled diabetes mellitus type I. During the course of the work, changes in the composition of calcium and phosphorus were detected on the 7th, 14th, 21st and 30th observation days throughout the experiment.

Results: Changes in the quantitative composition of calcium and phosphorus in the femur and shoulder bones of aloxane-induced rats are unidirectional.The amount of calcium in the control group rats was 2.46 mg., Phosphorus was 0.81. The amount of calcium decreased by 7 days by 0.8%, 14 days by 9.8%, 21 days by 2.5%, and by 30 days by 13.3% compared with the control, respectively. The content of phosphorus at 7, 14, 21, 30 days decreased by 2.5%, 6.5%, 6.5%, and 15.5%, respectively, compared to control.

Conclusions: This can be explained by the following. In a number of pathogenetic factors that induce the development of osteopenia in patients with diabetes mellitus is the formation and accumulation of end products of non-enzymatic glycosylation of proteins in the bones. The second probably cause of changes in bone mineral content in type I diabetes may be a shortage of vitamin D, which leads to loss of calcium and phosphorus in the urine. The above circumstances may explain the decrease in the content of calcium and phosphorus in bones in experimental type 1 diabetes mellitus.

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