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CHANGES IN THE STATE OF PROTEINOXIDATION MODIFICATION INDICES AND NITROGEN OXIDE METABOLISM IN THE BLOOD OF RATS WITH DIABETES MELLITUS IN THE DYNAMICS OF CEREBRAL ISCHEMIA-REPERFUSION

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Objective of the work was to examine changes in the state of protein oxidation modification indices and nitrogen oxide metabolism in rats with diabetes mellitus complicated by ischemic-reperfusion damage of the brain.

Diabetes mellitus was simulated by means of Streptozotocin (Sigma, USA) injection into the intra-abdominal cavity of 2-month male rats in the dose of 60 mg per 1 kg of the body weight. Clips were applied on both common carotid arteries of 6-month rats with diabetes and without it for 20 minutes. Early consequences of ischemia-reperfusion were examined one hour later when blood circulation was renewed, and the late consequences – on the 12^{th} day. Numerical data were processed by means of the package of the applied software programs "Statistica" ("Statsoft", USA).

The results of investigation of the content of the proteinoxidation modification products and nitrogen oxide metabolites in the blood of animals from different experimental groups are indicative of the fact that an hour later after beginning of reperfusion inanimalswithoutdiabetesthe products of protein oxidation modification of a neutral and major character 23 and 81 % increased respectively, and the content of nitrogen oxide metabolites did not undergo reliable changes. On the 12^{th} day the content of protein oxidation modification products of a neutral and major character became normal, and the content of nitrogen oxide metabolites 35 % increased concerning the control indices, and 27% concerning the index of an early post-ischemic period.

Rats with diabetes demonstrated higher indices of nitrogen oxide metabolites and the products of protein oxidation modification both of a neutral and major character – 29, 129% and 3 times as much respectively. In the early post-ischemic period the content of nitrogen oxide metabolites in rats with diabetes 22% decreased, and the content of protein oxidation modification products of a neutral character 6% increased. On the 12^{th} day the content of nitrogen oxide metabolites remained 32% lower in the blood of animals with diabetes, and the content of products of protein oxidation modification of the major character 22% decreased; the latter was 16% lower in comparison with the early term of observation. Moreover, the content of products of protein oxidation modification of a neutral character remained increased concerning the index of rats with diabetes.

20-minute carotid ischemia with one-hour reperfusion in rats without diabetes mellitus is found to increase the content of products of proteinoxidation modification. These indices return to the control values during late post-ischemic period. In animals with diabetes an increased content of products of protein oxidation modificationof a neutral character is observed in both periods of observation. In spite of the fact that rats with diabetes did not respond to cerebral ischemia-reperfusion at the early term by changes in the products of protein oxidation modification of a major character, on the 12^{th} day of the post-ischemic period their level decreases. Irrespective of the character of changes in the content of products of protein oxidation modificationwith cerebral ischemia-reperfusion, their content is reliably higher than corresponding indices in animals without diabetes during both periods of observation, which is indicative of higher intensity of their oxidation. The content of nitrogen oxide metabolites on the 12^{th} day of the post-ischemic period in rats without diabetes is indicative of nitrogen-induced stress. In case of diabetes during both terms of the post-ischemic period the content of nitrogen oxide metabolism is reduced.