

PRO-APOPTOTIC FACTOR BAX IS INCREASED IN RATS WITH EXPERIMENTAL CEREBRAL DIABETES

Z. Kundurovic, S. Hasanagic, N. Bilalovic,
F. Gavrankapetanovic

*University of Sarajevo Faculty of Medicine, Department of Histology, Sarajevo, Bosnia and Herzegovina;
University of Sarajevo Clinical Center, Sarajevo, Bosnia and Herzegovina*

Objective: It was proposed that impairment of neuronal insulin signaling induced by intracerebroventricular (ICV) administration of streptozotocin (STZ) might cause condition designated as experimental cerebral diabetes. Moreover, cerebral diabetes has been linked to the development of Alzheimer s disease (AD). It is demonstrated that neuronal insulin receptor is dysfunctional inAD. On the other it has been shown that neuronal apoptosis is abundant in postmortem brains of chronically diabetic rats and humans with type 1 diabetes and this condition is often referred to as a diabetic encephalopathy. The objective of this study was to determine if ICV administration of STZ induced apoptotic cascade activation. **Material and methods:** Animals were treated either by STZ or vehicle. Immunohistochemistry was performed 3 months after the ICV administration of STZ (1 mg/kg). Temporal gyrus tissue samples were used. Anti-Bax polyclonal antibody (Santa Cruz) and anti-Bcl-2 polyclonal antibody (DAKO) were used. Staining was measured semiquantitatively as the percentage of immunopositive cells and intensity of staining. Cognitive functions were tested by Moris Water Maze Test. **Results:** Significantly impaired learning and memory performance was found in STZ-treated rats compared to the control group. In temporal gyrus of STZ-treated rats percentage of Bax-positive neurons as well as the intensity of its staining were significantly increased in comparison to the control. There was no difference in Bcl-2 expression between the two groups of animals. **Conclusion:** Results of this study indicate that ICV administration of STZ might induce activation of apoptotic cascade through overexpression of proapoptotic factor Bax.