shape of the necrotic area after an ischemic stroke. The greatest changes were observed in the time groups 48 h and 7 days.

Treatment with use minocycline affects the decrease in the volume of the necrotic area.

HSP70 – DEPENDENT MECHANISMS OF ENDOGENOUS NEUROPROTECTION. NEW TARGET LINKS OF PHARMACOLOGICAL CORRECTION AFTER CHRONIC PRENATAL HYPOXIA

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Prenatal hypoxia (PH) causes pathological changes in the brain and can lead to irreversible long-term disorders of its development and the emergence of neuropsychiatric pathologies in children. The aim of this research was to study the effect of drugs (Angiolin, Thiotriazoline, Tamoxifen, Glutoredoxin, Cerebrocurin, Mexidol, L-arginine and Piracetam) on the expression of HSP70 protein as a factor of endogenous neuroprotection to further substantiate their use in the treatment of prenatal CNS damage in a model of chronic hemic PH. Expression levels of mRNA HSP70 and the content of HSP70 in the cytoplasmic and mitochondrial fractions of the brain of rat on the 60th day of life after PH were determined by real-time PCR and enzyme immunoassay. It has been established that chronic PH inhibits transcriptional processes in neurons and suppresses the synthesis of HSP70. The studied drugs are able to modulate HSP70-mediated mechanisms of endogenous neuroprotection. The most active among HSP70 modulators in conditions of chronic PH are cerebrocurin and angiolin, which outperform other studied drugs in terms of increased expression of HSP70 mRNA and HSP70 protein concentration in the brain of experimental animals and can be considered as promising neuroprotective agents in complex therapy after PH.

CHARACTERISTICS OF INFILTRATING CELLS WITHIN BRAIN HEMATOMA IN A RAT MODEL OF INTRACEREBRAL HEMORRHAGE

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The some studies of the role of endogenous mesenchymal stem cells in the central nervous system recovery after stroke are encouraging. The characteristics of cells infiltrating into a brain hematoma were investigated in this work. The intracerebral hemorrhage was modeled in the right internal capsule, by two-stage injection of autologous blood of 0.02 ml. Hemorrhage area and cellular responses at 1, 3, 10, 30, and 60 days were studied and compared with changes along the needle track in the brain of rats, without blood injection. The degree of hemorrhage and cell infiltration were ranked as points: 1 – small hematoma / single infiltrated cells; 2 – average hematoma / group of cells around hematoma; 3 – large hematoma with neuroinflammation/multiple infiltrates. The CD34, CD44, CD68, CD90 and CD146 positive cells occur in the hematoma with the highest intensity on the 3rd and the 10th day, whereas they are mainly absent on the 60th day. There is no relationship between the intensity of infiltration and the degree of hemorrhage, but a tendency for the appearance of CD44 positive cells in neuroinflammation was noted. Given the fact that mesenchymal cells express the investigated CD markers, we suggest their participation in the recovery processes after a hemorrhagic stroke.

THE INFLUENCE OF THE KETOGENIC DIET ON METABOLIC CHANGES AFTER TRAUMATIC BRAIN INJURY

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The ketogenic diet (KD) is a low-carbohydrate, high-fat diet in which the main energy source are ketone bodies. This diet, used primarily in the treatment of drug-resistant epilepsy, is nowadays considered a therapeutic solution in various neurological conditions, including traumatic brain injury (TBI). The aim of our experiment was to evaluate the effect of KD application after TBI. 27-days-old rats were divided into 2 groups receiving ketogenic or standard diet (SD). Penetrating brain injury was performed at postnatal day 30 in half of the animals from each group. The blood glucose, ketone levels, and body mass were monitored for the following 30 days. The analysis revealed a reduced weight gain in injured KD animals compared to the control group on KD. No such effect was observed for the SD rats. In rats on KD, weight gain was lower than in animals on SD, despite higher caloric intake in the groups on KD. Glucose level was lower in KD rats, however, a keto-adaptation occurred in the control group 19 days after KD introduction, which in TBI rats was observed 14 days later. The