MORPHOLOGICAL MARKERS TO ASSESS THE EFFECTS OF CHRONIC HYPOPERFUSION IN THE RAT BRAIN

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Existing criteria for evaluating of key attributes and consequences of hypoxia are diverse and contradictory. Therefore, the searching of structural and/or functional manifestations of hypoxia in the body tissues and primarily in the brain is the actual direction for investigation. The morphological changes in the brain tissue of out bred male rats with diabetes mellitus, obesity and there combinations and SHR line male rats with obesity and arterial hypertension were estimated with light microscopy method. It is well known that all diseases are accompanied by blood stream lesions and following up of different forms of cerebrovascular pathology. According to the international epidemiological studies, acute forms of brain disorders in human are the second or the third place in the mortality structure. There is increasing evidence to support the idea that chronic hypoperfusion of the brain is responsible for the Alzheimer's disease pathogenesis and other cognitive disorders. [1] Key relationships between these states are not fully identified and require further research.

The obtain results. All studied pathological processes were accompanied by signs of varying severity of encephalopathy. The neurons degeneration, gliosis (hyperplasia astrocytes), edema (perivascular and/or pericellular) were found in all cases (see Fig. 1, 2). But the most pronounced pathological changes, which are the manifestation of chronic hypoperfusion and hypoxia of the brain, consequently, have been identified in the modeling comorbidity (hypertension and obesity, diabetes mellitus and obesity), as well as in the modeling of obesity. Morphologically, these changes were expressed as edema, irregular arrangement of cellular elements with local spongiosis, astrocyte gliosis, oligodenrocyte satellitosis, aggregation of microglial, neuron dystrophy and necrobiosis (cell bodies were wrinkled and had the triangular form, Nissl's granules had disappeared, eosinophilia of the cytoplasm, pyknotic nucleus of triangular form) (see Fig. 3-6) [2].

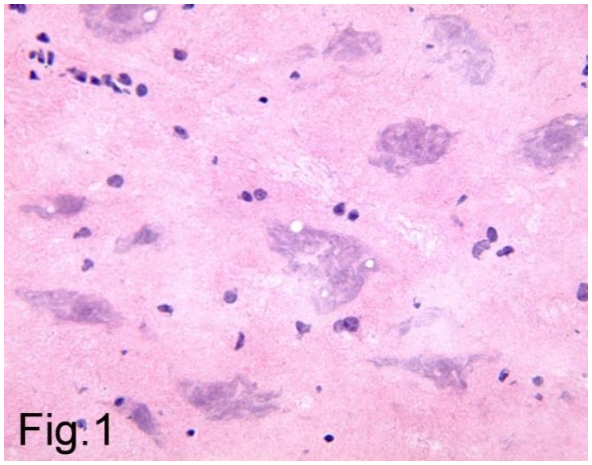

Fig. 1. Edema and degeneration of neurons in hypertension. Hematoxylin and eosin

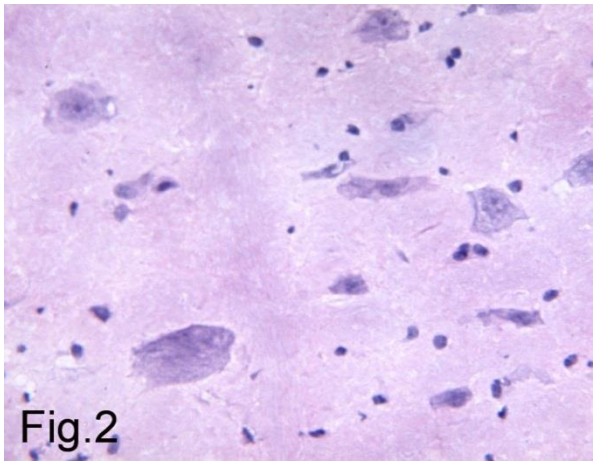

Fig. 2. Degeneration neurons in diabetes mellitus. Hematoxylin and eosin

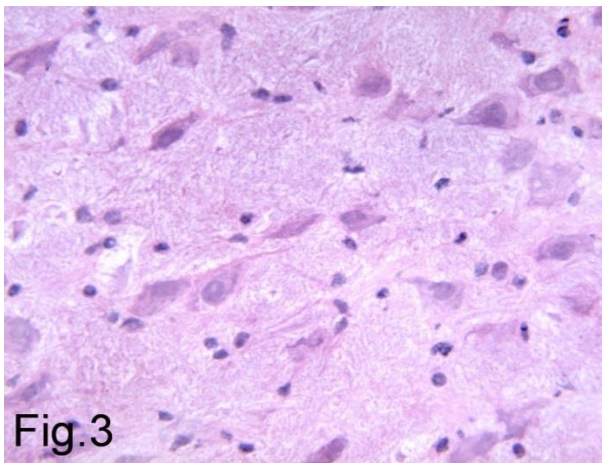

Fig. 3. Edema, degeneration of neurons, gliosis, satellitosis of oligodenrocytes in obesity. Hematoxylin and eosin

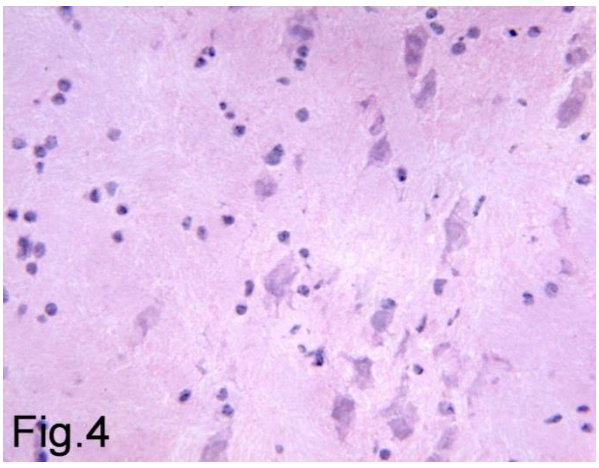

Fig. 4. Edema, degeneration of neurons, gliosis, satellitosis of oligodenrocytes in obesity. Hematoxylin and eosin

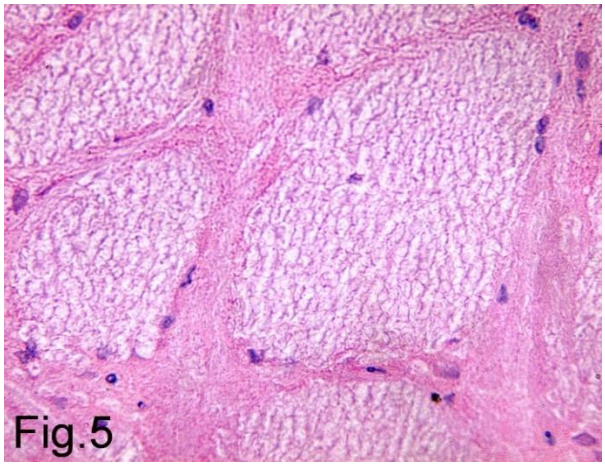

Fig. 5. Severe spongiosis stroma with hypertension combined with obesity. Hematoxylin and eosin

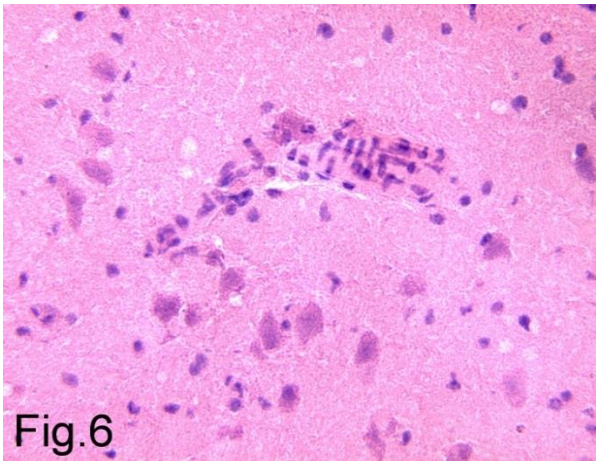

Fig. 6. Hypoxic degeneration of neurons, satellitosis of oligodenrocytes, gliosis in diabetes combined with obesity. Hematoxylin and eosin

Thus, the brain is one of the main targets for all kinds of pathology. With obesity and its combination with arterial hypertension and diabetes mellitus the brain changes are most significant. The ischemic encephalopathy, which is caused by severe vascular lesions, was formed. Vascular lesions are morphological markers for assessing the effects of chronic hypoperfusion in the brain.

References:

1. Toda N., Ayajiki K., Okamura T. Obesity-induced cerebral hypoperfusion derived from endothelial dysfunction: one of the risk factors for Alzheimer's disease. 2014. Vol. 11, No 8. P. 733-744.
2. Klatt E.C. Robbins and Cotran Atlas of Pathology. Elsevier Inc., 2006. P. 466-467.