МАТЕРИАЛЫ КОНФЕРЕНЦИИ И ШКОЛЫ

GLIO-NEURONAL APOPTOSIS IN DRUG-RESISTANT TEMPORAL LOBE EPILEPSY

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Many conceptual aspects of the pathogenesis of pharmacoresistant epilepsy (PRE) have not yet been resolved. In recent years, much attention has been paid to the study of the role of neuronal apoptosis in the pathogenesis of PRE. The purpose of this work is a comprehensive study of apoptosis processes in epileptic foci in patients with pharmacoresistant posttraumatic temporal lobe epilepsy. The material for the study was sections of the cortex and the underlying white matter of the brain in the area of the epileptic focus from 20 patients of the rnhi named after prof. Polenova with temporal PRE, received intraoperatively, under the control of corticography. The material for the comparison group was obtained from autopsies of 6 patients without epilepsy. An immunohistochemical (IHC) study was performed with antibodies to caspase-3 (apoptosis effector protein), Western blotting (WB) with the detection of caspase-8 (proapoptotic protein) and Bcl-2 (antiapoptotic protein), electron microscopy to identify cells in apoptosis. If you WB in an epileptic lesion of the temporal lobe of the brain there was marked reduction of antiapoptotic protein Bcl-2, increased level of proapoptotic protein caspase-8. In all cases of IHC, positive nuclear expression of caspase-3 was detected not only in neurons, but also in glial cells of the cortex and white matter of the brain. Electron microscopy revealed a significant number of neurons in the cortex with signs of apoptosis at various stages. Among glial cells, apoptotic changes were observed mainly in oligodendrocytes in the cortex and in the white matter of the brain. Thus, the detected changes in the expression of pro- and antiapoptotic proteins in epileptic foci indicate the role of apoptosis in the pathogenesis of pharmacoresistant epilepsy. The death of glial cells in FRE indicates a violation of neuroglial interactions and, as a result, can cause demyelination and disease progression.

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