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CURRENT SCIENTIFIC
GOALS, APPROACHES
AND CHALLENGES

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NEURONAL CASPASE-3 EXPRESSION IN THE BRAIN OF DECEASED PATIENTS WITH SEPSIS-ASSOCIATED ENCEPHALOPATHY

During sepsis, brain dysfunction with decreased consciousness less than 14 points of Glasgow Coma Scale indicates the development of sepsis-associated encephalopathy (SAE) [1]. This complex state is conditioned by neuroinflammatory and metabolic response of the brain neurogliovascular unit to dysregulated severe systemic infection with exclusion of the direct infection of the CNS, its vascular or traumatic damage, as well as other types of toxic/metabolic encephalopathies and neurodegenerative disorders [2, 3]. Morphological confirmation of glial reactivity in human septic brain have been partially described in our previous study [4], however, neuronal dysfunction and loss which determine clinical manifestations of human SAE are poorly described [5]. The current study was aimed to investigate neuronal caspase-3 expression in neuron-populated brain regions (cerebral cortex, dentate gyrus of hippocampus, thalamus, striatum, and cerebellum) of 35 deceased patients died from abdominal sepsis with SAE according to SOFA score. Control group included postmortem brains of 30 patients died from acute heart insufficiency. Caspase-3 expression was calculated as a percentage of immunopositive neurons from the total number of identified neurons in standardized fields of view of the microscope Scope A1 «Carl Zeiss» (Germany) using Videotest-Morphology 5.2.0.158 software.

The percentage of Caspase3+ neurons appeared much higher compared to control values: in cerebral cortex – 3.08-fold higher (by 208.17%), in the hippocampus – 4.16-fold (by 316.07%), in the thalamus – 3.73-fold (by 273.84%), in the striatum – 3.51-fold (by 251.25%), in the cerebellum - 2.95-fold (by 195.76%), respectively. Together with light-optically detected large number of ischemically-shrunken neurons, as well as widespread selective colliquative neuronal necrosis in the form of acute swelling and karyocytolysis, the immunohistochemically confirmed apoptotic mechanisms indicate a significant degree of irreversible neuronal damage, which causes brain dysfunction and failure in SAE conditions. A tendency towards greater increases in caspase-3 expression in the hippocampus compared to other brain regions may points to specific vulnerability and intense hippocampal tissue damage as well as subsequent more aggressive neuronal microenvironment, which is in line with the proposed earlier neuroanatomical specificity

concept of SAE [6, 7]. Considering close location of hippocampal dentate gyrus both to ventricular and subarachnoid spaces, as well as choroid plexus with more permeable BBB, complex toxic, metabolic and ionic impact on hippocampal tissue always appears relatively more intense, which can explain wide range of cognitive impairment in septic patients and in part of them long-term after sepsis [8, 9].

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