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REGIONAL ALTERATION OF ASTROGLIAL GFAP IN THE BRAIN OF SEPTIC PATIENTS WITH SEPSIS-ASSOCIATED ENCEPHALOPATHY: POSTMORTEM STUDY

Sepsis-associated encephalopathy (SAE) refers to acute diffuse brain dysfunction in response to dysregulated systemic inflammation in the absence of direct infection to the CNS [1]. Its clinical manifestations vary widely from degrees of delirium to coma [2]. The current pathogenesis includes BBB damage, amino acids and neurotransmitters imbalance, ischemia, mitochondrial dysfunction, and neuroinflammation [3, 4]. Glial fibrillary acidic protein (GFAP) is a type III intermediate filamentous protein representing the core component of the astrocyte cytoskeleton [5]. Astroglia being the primary parenchymal outpost of the brain facing all types of agents invading the BBB during sepsis quickly become reactive [1, 6]. GFAP is widely considered to be the hallmark of the reactive astrocytosis and astrogliosis in a spectrum of neuropathologies and this respond has been generally convinced to be heterogeneous in different brain regions [7, 5]. Moreover, the latter was evidenced in our previous studies of experimental CLP-septic rats and in deceased septic patients without specifying the presence of SAE [8, 9]. To clarify the mode of these differences in SAE brain, we studied postmortem brains of deceased patients with abdominal sepsis associated with SAE (n=35) – «SAE» group. 57.14% of patients also had clinical features of sepsis-associated liver injury (SALI). The relative area of GFAP-immunopositive material (IPM) (%) was investigated immunohistochemically in the cerebral cortex, white matter (WM), hippocampal dentate gyrus, thalamus, striatum, and cerebellum and showed statistically significant ($p < 0,05$) increase compared to control deceased patients (n=30) who died from acute heart failure without associated toxic-metabolic pathologies. GFAP⁺ IPM occupied bigger area in the cortex – 4.03-fold (by 303.31 %), white matter – 2.81-fold (by 181.64 %), hippocampus – 2.19-fold (by 119.29 %), thalamus – 1.83-fold (by 83.64 %), striatum – 2.03-fold (by 103.37 %), cerebellum – 2.02-fold (by 102.86 %) respectively. As it was shown, cortical region reflects the trend in more intense growth of GFAP expression compared other five brain regions. It can indicate a suspected more sensitive local cortical astroglial populations upon the neurotoxic impact of the systemic inflammatory factors as well as the brain region of the most active neuroinflammatory processes in SAE. The respectively lesser increase in GFAP expression in thalamic region might indicate the presence of partial astroglial structural remodeling under action of ammonia accumulation which predominantly affect thalamic region during liver injury in septic patients, which was shown previously [9].

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