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RACCOLTA DI ARTICOLI SCIENTIFICI CON GLI ATTI DELLA

V CONFERENZA SCIENTIFICA E PRATICA INTERNAZIONALE

«Ricerche scientifiche e metodi della loro realizzazione: esperienza mondiale e realtà domestiche»

Bologna
Repubblica Italiana

26 aprile
2024

In Viaggio Con il Levi: Scienza, Tecnologia e Impresa & ONG «Piattaforma scientifica europea»

DOI 10.36074/logos-26.04.2024

ISBN (online) 978-88-31277-22-8
ISBN (stampa) 978-617-8312-03-9

In Viaggio Con il Levi: Scienza, Tecnologia e Impresa | European Scientific Platform

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«Associazione Italiana di Storia Urbana»

Ucraina
«UKRLOGOS Group»

2024

ОСОБЛИВОСТІ ПРОФЕСІЙНОЇ ПІДГОТОВКИ МАЙБУТНІХ ФАХІВЦІВ ДО РОБОТИ З ДІТЬМИ З РОЗЛАДАМИ АУТИСТИЧНОГО СПЕКТРУ
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26 aprile, 2024;
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DOI 10.36074/logos-26.04.2024.083

ALTERATION OF AQP4 EXPRESSION IN THE BRAIN OF DECEASED SEPTIC PATIENTS WITH SEPSIS-ASSOCIATED ENCEPHALOPATHY

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Sepsis-associated encephalopathy (SAE) is one of the most common types of organ dysfunction during sepsis, which occurs secondary to systemic infection without overt CNS infection [1]. Its clinical manifestations include impaired consciousness, disorientation, cognitive impairment, or deep coma [2]. SAE pathophysiology includes non-disruptive BBB impairment, brain endothelia and neuroglia reactivation, diffuse neuroinflammation, impaired vascular autoregulation, ischemia, mitochondrial dysfunction, neuronal death, and neurotransmitter imbalance [3, 4]. BBB permeability closely depend on the activity of the main water channels of the brain – AQP4, which selectively belong to astroglial (astrocytes and ependymal cells) plasma membranes, especially those faced to vascular walls – vascular endfeet [5]. It has been previously reported that during experimental SAE as well as in deceased septic patients, expression of AQP4 increased substantially in different brain regions [6, 7]. Overexpression of

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aquaporins in septic brain can be a central factor for development of brain vasogenic edema in SAE [8, 9]. To reveal what brain regions are more characteristic for growth AQP4 expression in SAE patients, we studied postmortem brains of deceased patients with abdominal sepsis associated with SAE (n=35) – «SAE» group. In 57.14% of patients was diagnosed sepsis-associated liver injury (SALI). The relative area of AQP4-immunopositive material (IPM) (%) was studied immunohistochemically in cerebral cortex of four lobes, respective subcortical white matter, hippocampal dentate gyrus, thalamus, striatum, and cerebellum. Control group included the same brain regions of deceased patients (n=30) who died from acute heart failure without toxic-metabolic pathologies. It was revealed statistically significant ($p<0,05$) increase $AQP4^+$ IPM compared to control: in the cortex – 3.66-fold (by 266.76 %), white matter – 5.01-fold (by 400.8 %), hippocampus – 2.96-fold (by 196.94 %), thalamus – 4.79-fold (by 379.02 %), striatum – 3.67-fold (by 267.17 %), cerebellum – 3.66-fold (by 266.13 %) respectively. The trend in a more significant increase in AQP4 expression in the white matter is in line with the former studies evidenced the tendency to edematous changes of this brain region during sepsis which was largely supposed to be conditioned by systemic circulatory failure and decrease in local tissue perfusion. Besides impaired blood supply, explanation of thalamic 4.79-fold increase in AQP4 level can be also supplemented by predominant accumulation of tissue ammonia, which was shown in our recent study on septic brain of patients who suffered from SALI [7].

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