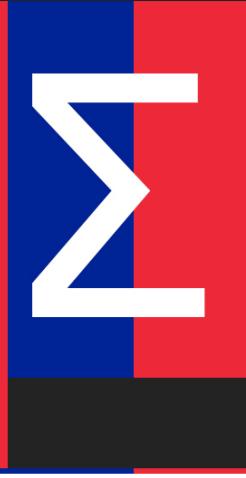


ΛΟΓΟΣ

COLLECTION DE PAPIERS SCIENTIFIQUES



ART DE LA PENSÉE SCIENTIFIQUE

SUR LES MATERIAUX DE LA IV CONFÉRENCE SCIENTIFIQUE ET PRATIQUE INTERNATIONALE

DÉBATS SCIENTIFIQUES ET ORIENTATIONS PROSPECTIVES DU DÉVELOPPEMENT SCIENTIFIQUE

11 NOVEMBRE 2022 • PARIS, RÉPUBLIQUE FRANÇAISE



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КОМПЕТЕНТНІСНИЙ ПІДХІД ЯК МЕТОДОЛОГІЯ ГАРМОНІЗАЦІЇ ОСВІТИ ТА РИНКУ ПРАЦІ
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ALTERATIONS OF THE BRAIN GLIAL FIBRILLARY ACIDIC PROTEIN DURING HUMAN LIVER CIRRHOSIS

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UKRAINE

Hepatic encephalopathy (HE) is one of the most severe complications of liver cirrhosis, manifesting as a complex neuropsychiatric disorder ranging from subtle attention deficits to hepatic coma [1]. It is associated with a poor survival prognosis and high mortality. HE pathophysiology is based on liver failure or portosystemic shunting, causing high systemic and brain ammonia levels. Elevated ammonia in the brain as well as systemic inflammation are considered key factors of acute HE [2]. Astrocytes are the most vulnerable brain cells to ammonia exposure being the only cells containing glutamine synthetase [3]. HE is characterized by generalized swelling of the astrocytes and acquiring of Alzheimer Type II phenotype [4]. Considering that glial fibrillary acidic protein (GFAP) plays a central role during structural and shape transformations of astroglia, discovering of the reactivity of local astroglia through the brain during liver cirrhosis seems actual. Thus, the present study aimed to postmortem study of GFAP level in 6 brain regions of cirrhotic patients in the dynamics of liver cirrhosis. For this purpose we discovered sectional material of 90 cirrhotic patients of classes A, B and C according to Child-Pugh classification. Using immunohistochemistry for GFAP we examined cortex, subcortical white matter, hippocampus, thalamus, striatum and cerebellum. Histopathology was analyzed with data from case histories. It was revealed that GFAP expression gradually decreased with raising the classes of cirrhosis. In all 3 classes, decreased expression was conditioned by shortening and thinning of GFAP+ processes, as well as by decrease in the number of GFAP+ astrocytes. Class A was characterized by decline of GFAP in all studied brain regions with the most prominent in thalamus – 2.19-fold, less pronounced in the cortex and hippocampus – 1.75-fold and the least in the striatum – 1.44-fold reduce. In the class B, decrease of GFAP level reached 5.13-fold in cortex, 3.78-fold – in thalamus and the least pronounced drop in cerebellum – 2.20-fold compared to control. The most expressed decline of GFAP was in class C: cortical region showed the lowest scores of GFAP expression – 6.74-fold decrease;

the second most altered was thalamus, where reduction was equal to 6.23-fold; the least valuable decrease was in cerebellum – 3.10-fold. In the cortex, hippocampus, striatum and cerebellum, alteration of GFAP expression differed significantly among groups depending on the dynamic aggravation of cirrhotic classes. Herewith, GFAP scores in the white matter and thalamus showed no difference between classes B vs. C. A significant dynamic decline in astroglial GFAP indicates morpho-functional remodeling of astrocytes, indirectly associated with changes in their shape and volume.

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