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SECTION 19.
PSYCHOLOGY AND PSYCHIATRY

SECTION 20.
MEDICAL SCIENCES AND PUBLIC HEALTH

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BRAIN GLUTAMINE SYNTHETASE EXPRESSION DURING HUMAN LIVER CIRRHOSIS

Hepatic encephalopathy (HE) being a frequent complication of liver cirrhosis is linked to high levels of systemic and brain ammonia [1]. Astrocytes are key brain cells to ammonia utilization as they contain glutamine synthetase (GS) [1]. Blood-borne and liquor-borne ammonia crossing the BBB or brain-cerebrospinal barrier, once in the brain, is metabolized solely by glutamine synthetase (GS) into glutamine at the astrocytic perivascular end-feet [2]. Studies have evidenced that GS is upregulated in different brain regions in acutely developed hyperammonemia [2], but not altered, downregulated or elevated in chronic forms of HE or liver disease without HE [3]. Given these mixed findings regarding GS changes in acute/chronic hyperammonemia, this question still needs to be addressed. The present study aimed to evaluate GS level in 6 brain regions of cirrhotic patients during liver cirrhosis. We examined postmortem material of 90 cirrhotic patients of classes A, B and C according to Child-Pugh classification. Immunohistochemically, using rabbit polyclonal anti-GS (Thermo Scientific, USA) antibodies, we studied cortex, white matter, hippocampus, thalamus, striatum and cerebellum. GS expression in control was found to be the highest in the cortex and the lowest in the white matter. In cirrhotic groups, GS expression gradually increased along with aggravation of liver cirrhosis. Class A of cirrhosis was characterized by elevation of GS in all studied regions with the highest values in the cortex – 2.36-fold and the least in the white matter – 1.39-fold. In class B, GS elevation gained maximal values to 3.62-fold in the cortex, 3.45-fold in thalamus and the least increase in striatum – 2.08-fold. The most prominent augmentation of GS was identified in class C. Cortical and thalamic regions presented the highest indicators of GS, respectively: 4.34-fold and 4.26-fold. The least elevation of GS scores was found in striatum and cerebellum, respectively: 2.95-fold and 2.99-fold. GS expression differed significantly in all pairs of subsequent cirrhotic classes only in white matter. In the cortex, hippocampus and thalamus, GS level differed significantly between A vs. B and A vs. C, but not between B vs. C. Wherein, in striatum and cerebellum GS expression differed significantly between A vs. C and B vs. C, but not between A vs. B classes. Summarizing, astrocytic GS expression in liver cirrhosis elevates from class A to class C in all 6 studied brain regions with a maximum increase in the cortex and thalamus. A significant gain in GS indirectly points to the brain hyperammonemia and neurotransmitter imbalance. Territorial heterogeneity in GS alterations is conditioned by both regional predominance/absence of glutamate neurotransmission and the diverse sensitivity of local astroglial populations to hepatotoxic factors.

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