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PREDICTIVE VALUE OF THE CEREBELLAR AMMONIA LEVEL IN THE DECEASED CIRRHOTIC PATIENTS FOR PROBABILITY OF INTRAVITAL HEPATIC ENCEPHALOPATHY

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One of the most severe complications of decompensated liver cirrhosis (LC) is hepatic encephalopathy (HE) type C, which worsens the prognosis of patient's survival significantly [1]. Pathological diagnosis of HE is not developed and only can be proposed by the exclusion of other causes of brain failure [2]. Hyperammonemia and brain ammonia play the central role in the morphogenesis of HE and stimulates significant changes to the neurogliovascular unit (NVU) leading to HE symptoms [3]. As has been shown in our previous study [4], brain tissue ammonia level correlates with specific neuropathological changes, including Alzheimer type 2-astrocytosis [5]. For assessment of the predictive value of postmortem brain ammonia level for the probability of intravital manifestations of HE, logistic regression analysis with ROC curves and the determination of the cutoff threshold was used. The median levels of optical density of HC ammonia precipitates expressed in CUOD (according to Gutiérrez-de-Juan V. method of the morphological determination of ammonia in the paraffin slices [6]) in the postmortem cerebellar tissue of 90 deceased patients with LC of compensated, subcompensated and decompensated stage (according Child-Pugh score) were analyzed for the presence or absence of HE Grade 1-4 intravital symptoms or the clinical diagnosis of HE in their medical cards. According to the ROC analysis, the postmortem median HC ammonia level in the cerebellar tissue > 32.65 CUOD indicated a statistically significant probability (sensitivity = 85.71 %, specificity = 76.47 %, AUC=0.834, p<0.001) of the presence of clinical signs of HE Grade 1-4 during patient's lifetime. Based on the results obtained, the level of postmortem cerebellar tissue ammonia can be used for the postmortem diagnosis of the overt HE type C, although, reliable pathological diagnosis of HE requires evaluation of the complex additional morphological ammonia-associated changes of the brain and clinical-anatomical matching.

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