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PREDICTIVE CAPACITY OF THE AMMONIA LEVEL IN THE POSTMORTEM BRAIN CORTICAL TISSUE FOR INTRAVITAL HEPATIC ENCEPHALOPATHY TYPE C

Decompensated liver cirrhosis (LC) is often complicated by hepatic encephalopathy (HE) type C, which worsens the prognosis of survival significantly [1]. Postmortem diagnosis of HE always requires the exclusion of other causes of brain failure and is yet to be developed [2]. Brain ammonia plays the key role in the morphogenesis of HE and instigates significant changes to the neurogliovascular unit (NVU) causing HE manifestations [3]. As has been shown previously [4], brain tissue ammonia level correlates with specific neuropathological changes, including Alzheimer type 2-astrocytosis [5]. For assessment of the predictive ability of postmortem brain ammonia levels for the presence of intravital manifestation 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 [6]) in the postmortem brain parietal cortex of 90 deceased patients with LC were analyzed for the presence or absence of HE Grade1-4 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 cortical tissue > 29.67 CUOD indicated a statistically significant probability (sensitivity = 65.45 %, specificity = 94.29 %, AUC=0.732, $p<0.001$) of the presence of clinical signs of HE during patient's lifetime. Based on the results obtained, the level of postmortem cortical tissue ammonia can be used for the retrospective diagnosis of the overt HE type C, although, final neuropathological diagnosis

requires evaluation of the additional morphological ammonia-associated changes of the brain NVU.

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