Pathophysiology of hepatic encephalopathy: exploratory study using $^{13}$N-ammonia PET

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Abstract

Increased blood ammonia in patients with liver cirrhosis is key factor to develop hepatic encephalopathy (HE). But, the pathophysiology of hyperammonemia-induced HE still do not have been fully understood. Nishiguchi et al. reported the evaluation of ammonia metabolism in the skeletal muscles of patients with cirrhosis using N-13 ammonia positron emission tomography (PET) before and after branched-chain amino acids (BCAAs) administration. Thus our aim is to clarify the regional cerebral ammonia metabolism before and after BCAAs administration. We are going to undertake N-13 ammonia PET of brain of cirrhotic patients before and after BCAA-enrich infusion, that are used as a treatment for hyperammonemia in Japan. Simultaneously, we are going to conduct neuropsychiatric tests, to consider the mechanism of HE by analysing the results and patient’s biochemical profiles.