

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

Yuki Watanabe<sup>1</sup>, Kei Sahara<sup>1</sup>, Takayosi Oikawa<sup>1</sup>, Hiroya Takahashi<sup>1</sup>, Kazuyuki Suzuki<sup>1</sup>  
Akinobu Kato<sup>1,2</sup>, Tosihaki Sasaki<sup>3</sup>, Kazunori Terasaki<sup>3</sup> and Kouichiro Sera<sup>3</sup>

<sup>1</sup>Iwate Medical University, Department of Internal Medicine, Division of Gastroenterology and Hepatology  
19-1 Uchimaru, Morikoka, Iwate 020-8505, Japan

<sup>2</sup>Morioka Municipal Hospital  
15-1 Motomiya-aza-Koyasiki, Morioka, Iwate 020-0866, Japan

<sup>3</sup>Cyclotron Research Center, Iwate Medical University  
348-58 Tomegamori, Takizawa, Iwate 020-0173, Japan

### **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.