Hepatic encephalopathy and prehepatic portal hypertension rat model

June 29 2009

Portal hypertension is responsible for severe and often lethal complications of cirrhosis. Another important syndrome is hepatic encephalopathy as a consequence of acute and chronic liver failure, which is characterized by a wide spectrum of neuropsychiatric abnormalities. A study group from Argentina analyzed the participation in these syndromes of glutamine synthetase and glutamate uptake in the hippocampus and frontal cortex, by using the prehepatic PH rat model.

A research article to be published June 21, 2009 in the World Journal of Gastroenterology addresses this question. The research team lead by Dr. Gabriela Beatriz Acosta, observed that the activity of GS was increased in the hippocampus in PH rats. There was a significant decrease in glutamate uptake in both brain areas, which was more marked in the hippocampus.

The decrease in glutamate uptake might be caused by deficient transport function and persistent glutamate activity, which is not metabolized. This leads to severe damage in the cells of the central nervous system (CNS), associated with the presence of moderate ammonia concentration in the blood, as observed in this model. These results demonstrated that partial stricture of the portal vein is able to modify normal function in important areas of the rat brain.

These results suggest that, in PH this pathology, there are differences between both regions of the brain, possibly caused by the toxic metabolic action of ammonia and glutamate, and perhaps glutamine, in

the brain, among other factors.

Using this model, it may be possible to understand more clearly the mechanism of toxicity and defense of the brain against the two toxic substances: ammonia and the excitatory neurotransmitter glutamate. The study is important for understanding of some of the mechanisms related to HE.

More information: Acosta GB, Fernández MA, Roselló DM, Tomaro ML, Balestrasse K, Lemberg A. Glutamine synthetase activity and glutamate uptake in hippocampus and frontal cortex in portal hypertensive rats. *World J Gastroenterol* 2009; 15(23): 2893-2899 www.wjgnet.com/1007-9327/15/2893.asp

Source: World Journal of Gastroenterology (news : web)