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VERSITA

AMMONIA: THE NEGLECTED RISK FACTOR

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Abstract

The letter deals with the article published in Translational Neuroscience 2011, 2 (4), 360-362. It emphasizes the importance of the authors' findings in relation to the role of ammonaemia in diagnosing hepatic encephalopathy.

Keywords

• Ammonia • Hepatic encephalopathy • Dementia • Delirium • Cognitive impairment

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The case report by Boban and Malojčić (entitled "Young-onset dementia and MRI changes in a patient with subclinical liver cirrhosis due to chronic hepatitis C" published in Translational Neuroscience, 2011, 4, 360-362) is extremely interesting, especially because of its clinical implications.

The first point to be remarked is that a subject with obvious clinical and biochemical signs of cirrhosis with portal hypertension was referred to a dementia outpatient clinic due to cognitive decline. In such a situation, hepatic encephalopathy (HE) should have been suspected immediately. In addition, even when an otherwise healthy individual with cognitive dysfunction is investigated to confirm/exclude a neurodegenerative disorder all metabolic, reversible causes of brain dysfunction should be searched for, and treated first.

HE, far from being a rare disorder, can be detected, at least in its mild form, in a minimum of 30% of patients with cirrhosis [1]. The presented case was one of overt HE, as the patient was disoriented in both time and space. The considerable improvement in clinical status (MMSE 18/30 to 25/30) – we assume, over a much shorter period than the 7 months in between the two neurological evaluations – suggests that the episode was one of mild delirium rather than dementia, as is the case for bouts of HE.

The relevant observation made by the authors is that hyperammonaemia was a crucial element for the diagnosis, despite the fact that measurement of plasma ammonia levels is not recommended by the American Academy of Neurology. Ammonia, regardless of its role in the pathophysiology of HE, is an excellent marker of the presence of portal-systemic shunting and the reduction in hepatic clearance of gut-derived neurotoxins. Therefore, the existence of hyperammonaemia, even if it does not indicate the existence of HE, is proof of the existence of the pre-requisite conditions, which make it possible, or even likely, for HE to occur.

The lack of emphasis on the role of ammonia measurement in the management of patients with HE is probably related to the notion that a close correlation between ammonia levels and the clinical signs of HE does not exist [2]. However, this is somewhat irrelevant. As Boban and Malojčić clearly demonstrate, the clinical condition of delirium requires some degree of differential diagnosis, even in a patient with cirrhosis. Indeed, patients with cirrhosis are susceptible to HE, but also to other metabolic encephalopathies, such as those related to infection, malnutrition, electrolyte or glucose imbalance, etc. These need to be differentiated, and treated accordingly. There is no question that ammonia measurement helps in this process.

References

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