CLINICAL SIGNIFICANCE OF HEPATIC ENCEPHALOPATHY IS VERY HIGH

EXPERT INTERVIEW WITH DR. HENNING PFLUGRAD · CLINIC FOR NEUROLOGY AT THE HANNOVER MEDICAL SCHOOL (MEDIZINISCHE HOCHSCHULE HANNOVER)

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The hepatic encephalopathy is among the most frequent and severe complications for patients with liver failure. It makes the prognosis worse and affects the patient's quality of life. If diagnosed promptly, the neurological disorder can be treated well.

Dr. Pflugrad, what is the significance of hepatic encephalopathy (HE) today in medical practice? What role does minimal hepatic encephalopathy (mHE) play?

The clinical significance of hepatic encephalopathy is very high, because there are so many patients with chronic liver failure. It is estimated that approx. 35–45 % of these patients experience HE episodes. The frequency of minimal HE is estimated to be about 30-80 % among patients with chronic liver failure. The large range with mHE can be traced back to the impairments not being determinable in clinical investigation and therefore the psychometric test procedures are required. As a result, the estimated number of affected patients is definitely high. The significance of mHE is however, not to be underestimated because it can restrict the performance and health-related quality of life of the patient.

What influence do comorbidities, like metabolic disorders or neurological diseases often found with cirrhosis, have on the development of an HE?

Factors such as infections, medication, metabolic disorders, electrolyte lapses or traumas can trigger HE or substantially influence the course. Identification of these triggering or accompanying factors is a great challenge, but always of immense significance because the treatment or removal of these factors is the first step in treating HE.

How is HE diagnosed and treated?

HE is always a diagnosis by exclusion because there are no symptoms or biomarkers that unequivocally demonstrate HE. If a patient with liver cirrhosis develops cognitive disorders, the causes are initially sought after. The first steps consist of laboratory-chemistry diagnostics and cerebral imaging. The significance of the ammonia levels is controversially discussed. High ammonia levels can support the diagnosis of HE, however it does not exclude other causes of cognitive disorders, for example a bleed on the brain.

The best confirmation for the presence of an HE is the patient's response to an ammonia-lowering treatment.

The therapeutic concept for HE arises after the exclusion of other causes of a cognitive disorder, initially from the identification and treatment of precipitating factors. The pharmacological treatment targets the reduction of the ammonia levels. The most important medication is lactulose, which reduces the intestinal ammonia resorption and rifaximin, intestinal-selective antibiotics, as well as L-Ornithine-L-Aspartate, which supports the degradation of ammonia in the liver, brain and muscles.

Cognitive disorders often occur after a liver transplant. Until recently, it was believed that the transplant not only treated the underlying liver disease, but also the hepatic encephalopathy that developed from it. Are there any new findings today?

After the transplant, the cirrhosis is healed as the cause of HE, thus HE cannot arise anymore. Metabolic changes in the brain of patients with HE recede within months after a transplant. In the long term, we have seen that HE-related cognitive changes no longer play a significant role five years after the transplant. Therefore, approx. 30 % of patients exhibit neurological symptoms in the first few weeks after the transplant, which are frequently caused by metabolic changes. Here, we speak of a post-transplant encephalopathy. The causes are unclear. HE-related metabolic changes in the brain and neurotoxic side-effects of the immunosuppressive medication are being discussed.

MANY THANKS DR. PFLUGRAD FOR THIS INTERESTING INTERVIEW.