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Brain Lesions in Liver Cirrhosis May Not Only Be Due to Hepatic Encephalopathy

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We read with interest the article by Lim & Lee (1), who reported a retrospective study on the utility of cluster analysis of the symmetric regional cerebral edema (SRCE) score for predicting the development of brain failure among 98 patients with hepatic encephalopathy (HE). The study concludes that SRCE patterns could predict hepatic preservation and the occurrence of brain failure in patients (1). However, several points necessitate further discussion.

Firstly, patients with liver cirrhosis may suffer not only from liver disease but also from involvement of other organs. Cirrhosis is frequently associated with cardiac conditions such as heart failure or arrhythmias, which are significant risk factors for cardiovascular and cerebrovascular events (2). Cirrhotic cardiomyopathy can also develop in these patients (3). Therefore, it is crucial to ascertain how many of the 98 included patients had concomitant heart failure, left or right ventricular systolic dysfunction, pulmonary hypertension, atrial fibrillation, or malignant ventricular arrhythmia.

Furthermore, cirrhosis can lead to multisystem complications, potentially causing embolic stroke or watershed infarctions that may mimic HE. It is essential to consider comorbidities among the study cohort to better understand the complexities of liver cirrhosis. Alcoholic cirrhosis, for instance, can predispose patients to hyponatremia, which may provoke epileptic seizures presenting with MRI features resembling HE. Moreover, cirrhosis-related complications such as hepatorenal syndrome can lead to kidney failure, which in turn may result in cerebral complications (2, 3).

Secondly, there was no mention of how nontoxic liver cirrhosis was comprehensively ruled out. Liver cirrhosis not associated with alcoholism, intoxication, or viral infection has been reported in myotonic dystrophy type (4). Additionally, genetic liver cirrhosis can occur in carriers of POLG1 variants, presenting phenotypically as Alpers Huttenlocher syndrome or Leigh syndrome (5). Were there patients with genetic

liver disease among the 98 patients?

Thirdly, infectious or immune encephalitis has not been completely ruled out as a cause of cerebral lesions. Patients with liver cirrhosis are vulnerable to immunological and infectious diseases due to immune system effects. It is essential to determine how many included patients underwent cerebrospinal fluid examinations for infectious encephalitis and how many tested positive for antibodies associated with immune encephalitis.

Fourthly, diagnoses of drug-induced hepatitis and hepatitis of unknown etiology were not reported. In these 12 patients, were liver biopsies performed for diagnosis or was diagnosis based solely on imaging?

Fifthly, no definition of SRCE has been provided. Edema typically appears hyperintense on T2/fluid-attenuated inversion recovery but may also appear hyperintense on diffusion-weighted imaging and apparent diffusion coefficient. Additionally, there is no definition of "cerebral failure."

Sixthly, it is not reported how many of the included patients underwent liver transplantation and whether cerebral imaging abnormalities resolved in transplanted patients.

Seventhly, Table 1 presents serum values of several parameters without reference limits (1). To assess which parameters were outside the normal range, it is crucial to know the reference limits for all analyzed parameters. Notably, many included patients had elevated ammonia levels.

In summary, this study has limitations that should be addressed before firm conclusions can be drawn. Clarifying these points will strengthen and improve the conclusions. Before attributing cerebral lesions to HE in patients with liver cirrhosis, all differential cerebral complications associated with liver cirrhosis should be rigorously excluded.

Conflicts of Interest

The author has no potential conflicts of interest to disclose.

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