Letter to the Editor
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# Response to "Brain Lesions in Liver Cirrhosis May Not Only Be Due to Hepatic Encephalopathy"

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We thank Dr. Josef Finsterer for taking the time to read our article (1) and for providing his comments. In the letter to the editor, Dr. Josef Finsterer highlighted seven issues for discussion. The content pointed out by the author of the letter can be roughly divided into two parts: the first four issues concern the patient group, and the latter three concern the analysis. As the title of his letter suggests, his main point was "Brain lesions in liver cirrhosis may not only be due to hepatic encephalopathy." We agree with this proposition. Readers interested in this topic may agree with the inevitability of the question, "Can MRI differentiate brain lesions from hepatic encephalopathy in cirrhotic patients?" When the brain MRI of a cirrhotic patient who visits the emergency room with changes in consciousness shows symmetric regional cerebral edema (SRCE), how can a radiologist write an interpretation? As Dr. Josef Finsterer pointed out, patients with liver damage are very sensitive to organ failure of the kidneys, brain, respiratory system, and circulatory system, as well as blood coagulation failure. If genetic and infectious diseases are also considered, the image analysis becomes virtually meaningless. An accurate diagnosis of a patient's brain lesion depends only on the clinician's clinical diagnosis, including history-taking and blood laboratory tests. Moreover, a few cases of hepatic encephalopathy show high T2 or diffusion signal intensities in the basal ganglia, mimicking extrapontine myelinolysis (EPM), a form of osmotic demyelination syndrome, or uremic encephalopathy. EPM is regarded as a disease occurring in alcoholic and malnourished patients with chronic renal failure or hepatocellular dysfunction (2, 3). In chronic liver dysfunction, decreased urea cycle activity causes astrocytes to increase cerebral glutamine synthesis, which plays an important role in uremic encephalopathy.

Chronic liver disease causes various changes including impaired protein synthesis, alterations in glial fibrillary acidic protein gene expression, and increased sensitivity to electrolyte changes, which are different underlying causes of encephalopa-

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thy (4). To elucidate the causality between such metabolic changes and the SRCE phenotype, it is important to classify the SRCE patterns. Some brain regions, such as the red and dentate nuclei, are well known to have close interconnectivity and may be an important factor in classifying brain lesions. Nevertheless, the authors acknowledge that 98 patients were insufficient to classify the types of hepatic encephalopathy by examining 12 anatomical regions. In the 98 patients who did not include patients with heart failure, genetic disease, brain infection, or immune-related brain disease, there was a group mainly affected at the tegmentum or tectum, a group affected at the striatum and showing a form similar to EPM, and a group affected at the red and dentate nuclei. These can be divided into the affected groups.

The recent acceptance of new clinical concepts, such as "acute-on-chronic liver failure (ACLF)," has led to changes in the clinical view of hepatic encephalopathy. One of the important concepts in ACLF is organ failure, and both the Chronic liver failure-sequential organ failure assessment (CLIF-SOFA) score and the CLIF consortium organ failure score define West-Haven criteria III and IV as "cerebral failure" (5). Although identifying the specific cause through imaging is crucial, understanding the patients' overall condition and predicting their prognosis based on clinical concepts are equally important tasks for radiologists. Vasogenic edema and astrocytic cytotoxicity have been frequently observed in the autopsies of patients with cirrhosis and in animal models (6, 7). To investigate the two types of cerebral edema, we attempted to analyze lesion patterns using lesion connectivity in T2-weighted images and diffusion-weighted images. Although it is well known that brain edema occurs in ACLF, we acknowledge that radiologists currently lack clear diagnostic criteria for this condition. SRCEs were lesions on brain MRI in patients with cirrhosis, defined based on these considerations.

## Conflicts of Interest

The author has no potential conflicts of interest to disclose.

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