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Letter to the Editor

Acute, henorrhagic leucoencephalitis not necessarily represents neuro-COVID

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We read with interest the article by Benevides *et al*. about a 50 years-old female with acute, hemorrhagic leucoencephalitis (AHLE) who was admitted for two tonic clonic seizures, severe headache, psychomotor agitation, myalgia, vomiting, and fever since 3 days [1]. Upon cerebral MRI, cerebrospinal fluid (CSF) investigations, and electroencephalography, (EEG), AHLE was diagnosed and the patient received intravenous methyl-prednisolone during five days resulting in almost complete recovery until the one-month follow-up after dismissal [1]. The study is appealing but has several limitations that raise concerns and should be discussed.

We disagree with the notion that AHLE was triggered by SARS-CoV-2 [1]. Only a few viral agents have been ruled out as possible trigger of AHLE. Because AHLE is most commonly associated with previous bacterial or viral disease, we should know the results of a comprehensive CSF viral panel.

We also disagree with the statement that AHLE may be reported more frequently with COVID-19 than other viral illnesses [1]. No systematic studies have been conducted on the prevalence of AHLE in COVID-19 patients. Therefore, the statement remains unsupported by evidence and is thus a speculation.

The information about the status of the cerebral arteries is missing. We should know the results of magnetic resonance arteriography and computed tomography angiography. Particularly, arterio-venous malformations, aneurysm formation, cerebral vasculitis, and venous sinus thrombosis (VST) need to be appropriately ruled out. There is also a need to rule out reversible, cerebral vaso-constriction syndrome (RCVS). Ruling out vascular compromise is crucial as the index patient had severe headache at onset of the disease which remained unexplained [1].

How can the authors sure that there was no evidence of systemic vasculitis [1]? No information about arteries on imaging or biopsy was provided. No data about the anti-nuclear and anti-neutrophil cytoplasmic antibodies were provided.

There is a discrepancy between the description that the patient had apathy and psychomotor agitation at the same time. This discrepancy should be solved. There is also a discrepancy between the statement that multiorgan clinical examination was normal and the extensive neuropsychological deficits described on clinical neurologic exam. This discrepancy should be addressed.

The PCR for SARS-CoV-2 was negative in the CSF two times. We should know if cytokines, chemokines, neurofilaments, or glial factors were elevated in the CSF Elevation of some of these parameters would be an argument in favour of a causal relation between SARS-CoV-2 and AHLE, as these factors have been reported to be elevated in neuro-COVID [2].

The erythrocyte count was elevated two times in the CSF [1]. We should know if elevated CSF erythrocytes were due to traumatic lumbar puncture, to subclinical subarachnoid bleeding (SAB), due to hemorrhagic meningitis, or due to AHLE. Missing is also an explanation for the elevated CSF protein.

A shortcoming of the study is that the treatment the patient received for COVID-19 was not provided.

Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. A causal relation between SARS-CoV-2 and AHLE in the index patient remains unsupported.

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References

1. Benevides ML, Elias S, Fagundes DA, Martins RF, Dutra MM, Rodrigues de Oliveira Thais ME, *et al*. Acute Hemorrhagic Leukoencephalopathy Triggered by COVID-19 Infection. Neurohospitalist. 2022; 12(3):524-528.
   Doi: 10.1177/19418744221077735.
2. Gigli GL, Vogrig A, Nilo A, Fabris M, Biasotto A, Curcio F, *et al*. HLA and immunological features of SARS-CoV-2-induced Guillain-Barré syndrome. Neurol Sci. 2020; 41(12):3391-3394.
   Doi: 10.1007/s10072-020-04787-7.