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Letter to the Editor

Diagnosing infectious, SARS-CoV-2 related encephalitis/myelitis requires documentation of the virus in the cerebrospinal fluid

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We read with interest the article by AlHendawi *et al*. on a 2 years-old male who was diagnosed with SARS-CoV-2 related encephalitis and transverse myelitis based on the clinical presentation and the findings on cerebral magnetic resonance imaging (MRI) [1]. Initially the patient presented with a series of generalised seizures for which he required intubation and mechanical ventilation for two days [1]. He was empirically treated with ceftriaxone, vancomycin, and acyclovir, and later received intravenous immunoglobulins (IVIGs), methylprednisolone, and plasmapheresis [1]. He recovered completely within >10 days on the pediatric intensive care unit (PICU) [1]. The study is excellent but has limitations that raise concerns and require discussion.

We disagree with the diagnosis encephalitis for several reasons. First, there was no pleocytosis (1 cell/mm3) [1]. Although pleocytosis is occasionally absent in patients with encephalitis at least at the beginning, its presence would be a strong argument in favour of the diagnosis. Missing in this respect are repeated CSF investigations, as pleocytosis may develop with progression of the disease. Second SARS-CoV-2 was not confirmed by RT-PCR in the cerebrospinal fluid (CSF). Documentation of the virus in the CSF is mandatory to diagnose SARS-CoV-2 related encephalitis. Third, the MRI findings could be due to several other causes, which need to be thoroughly ruled out before attributing the cerebral lesions to SARS-CoV-2 encephalitis. Differential causes of these lesions include seizures, embolic stroke, acute disseminated encephalomyelitis, (ADEM), acute, hemorrhagic necrotising encephalitis (AHNE), acute, hemorrhagic leucoencephalitis (AHLE), posterior reversible encephalopathy syndrome (PRES), multiple sclerosis (MS), neuromyelitis optica (NMO) spectrum disorders, and MOG-associated disorders (MOGAD). We should also know if oligoclonal bands were positive or negative, and if aquaporin-4 antibodies, MOG antibodies, were elevated or normal. This is why control MRI after discontinuation of seizures is mandatory. Fourth, there is not mention that there was enhancement of the cerebral lesions upon application of contrast medium. Enhancing lesions are the hallmark of encephalitis on cerebral imaging. Fifth, the cerebral lesion could be pre-existent and could be present already prior to the SARS-CoV-2 infection. Sixth, there was no determination of antibodies for autoimmune encephalitis, which has been reported as a complication of SARS-CoV-2 infections [2]. Seventh, CSF was not investigated for cytokines, chemokines, 14-3-3, and glial factors, which can be elevated with SARS-CoV-2 associated CNS disease [3].

Regarding epilepsy, we should know the results of cerebral MRI, EEG, and eventually CSF after febrile seizures at age 1y. Did the patient experience any seizures between ages 1 and 2? There is no mention of the family history. We should know if any of the first-degree relatives had epilepsy.

A limitation of the study is that no reference limits were provided, making it difficult to assess what is normal and what is abnormal. Another limitation is that the MRI images were not provided, making it difficult to assess the type, nature, etiology and pathophysiology of the lesions.

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. To diagnose SARS-CoV-2 related encephalitis and transverse myelitis, documentation of pleocytosis and documentation of SARS-CoV-2 within the CSF is mandatory. Furthermore, all differentials of the described MRI lesions should be considered and thoroughly ruled out.

Declarations

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