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SARS-CoV-2 Related Immune Cerebellitis Requires Evidence Through Appropriate Imaging

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LETTER TO THE EDITOR

We read with interest Watanabe *et al*'s. article about a 56 year-old female who developed cerebellar symptoms and signs approximately two weeks after a mild infection with SARS-CoV-2 [Watanabe, T. *et al*., 2023]. Magnetic resonance imaging (MRI) revealed bilateral, hyperintense cerebellar lesions on diffusion-weighted imaging (DWI) and fluid attenuated inversion recovery (FLAIR). Antibodies against the glutamate receptor were detected in the cerebrospinal fluid (CSF) and therefore immune cerebellitis was diagnosed [Watanabe, T. *et al*., 2023]. After two three-day treatments with methyl-prednisolone, the MRI changes disappeared [Watanabe, T. *et al*., 2023]. The study is impressive, but some points require discussion.

The first point is that the MRI was performed without contrast agent [Watanabe, T. *et al*., 2023]. In order to document cerebellitis using imaging, the administration of contrast medium is absolutely necessary. Normally, immune encephalitis, including cerebellitis, is characterised by marked enhancement, particularly of the leptomeninges, on T1-sequences after contrast administration [Fadakar, N].

The second point is that cerebellar ischemia could not sufficiently be ruled out. Because the cerebellar lesions were hyperintense on DWI, it is imperative to report apparent diffusion coefficient (ADC) maps to determine whether the DWI lesions were hypo-, hyper- or iso-intense on ADC. DWI hyperintensity along with ADC hypointensity would suggest cytotoxic oedema and therefore ischemia. Vasogenic oedema, as occurs in immune encephalitis, would manifest as DWI hyperintensity along with ADC hyperintensity.

A third point is that the patient did not undergo single voxel proton MR-spectroscopy (MRS). Cerebellitis in the acute phase can occur in MRS with low N-acetyl-aspartate (NAA)/creatine (Cr) ratio but normal choline/Cr ratio [Guerrini, L. *et al*., 2002]. MRS would also be able to measure glutamate concentrations in the CNS [Tkáč, I. *et al*., 2023].

A fourth point is that the CSF was not tested for immune activation and not for neuronal or glial markers, which are known to be elevated in patients with central nervous system (CNS) involvement in SARS-CoV-2 infection [Kanberg, N. *et al*., 2023]. CSF studies in patients with SARS-CoV-2 infection with CNS involvement often show the absence of ongoing viral replication but signs of immune reactivity and elevation of neuronal and glial factors [Kanberg, N. *et al*., 2023]

A fifth point is that whole-body computed tomography is not sufficient to rule out malignancy. Has the patient undergone a gastroscopy, colonoscopy, mammography, and gynaecological examination?

The study has several limitations: CSF was not examined a second time after recovery; the CSF was only examined for a limited number of encephalitis-associated antibodies [Watanabe, T. *et al*., 2023]. Tests for NMDA, LGI1, GABA1, GABAb, AMPAR, CASPR2, DPPX, and amphyphysin antibodies are missing; the CSF was only tested for herpes simplex and herpes zoster viruses, but not for the entire virus panel [Watanabe, T. *et al*., 2023]. Several other viruses, including SARS-CoV-2, may have been causative; the vaccination status is missing. Was the patient adequately vaccinated before the cerebellar symptoms appeared? If vaccinated, it should be discussed whether CNS complications could have been less serious than without SARS-CoV-2 vaccination.

In summary, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these weaknesses would strengthen the conclusions and could improve the

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study. Before cerebellar dysfunction can be attributed to SARS-CoV-2-related cerebellar involvement, various differential diagnoses must be thoroughly ruled out.

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