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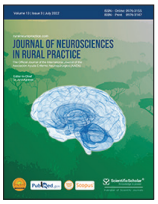

Letter to the Editor

Before a diagnosis of recurrent osmotic demyelination syndrome is made, all possible differential diagnoses must be ruled out

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Dear Sir,

We were interested to read the article by Varoglu *et al*. about two patients who suffered from recurrent "attacks of central nervous system (CNS) demyelination" after BNT162b2 vaccination.[1] Patient 1 was a 37-year-old man, who suffered an isolated right-sided oculomotor palsy attributed to a T2/fluid-attenuated inversion recovery (FLAIR) and diffusion-weighted imaging (DWI)-hyperintense and apparent diffusion coefficient (ADC)-hypointense pontine lesion that regressed under methyl prednisolone and recurred 4 months later.[1] Patient 2 was a 57-year-old man with hand and foot numbness, disorientation, and confusion that began 2 weeks after a BNT162b2 vaccination.[1] Magnetic resonance imaging showed T2 hyperintensity with contrast enhancement.[1] He also benefited from methyl-prednisolone but relapsed after 6 months.[1] The study is convincing, but several issues should be addressed.

First, the diagnosis of pontine myelinolysis in patient 1 is not confirmed.[1] The lesion was described as T2/FLAIR and DWI hyperintense and ADC hypointense.[1] This constellation suggests cytotoxic edema rather than vasogenic edema, as would be expected in pontine myelinolysis. It is therefore essential to rule out an ischemic stroke as the cause of the oculomotor palsy. In particular, we should know what kind of risk factors for cerebrovascular disease the index patient had. Was there a history of smoking, arterial hypertension, diabetes, hyperlipidemia, or atrial fibrillation? Was there a history of a previous cerebrovascular event? Another strong argument against pontine myelinolysis in patient 1 is that the serum sodium was normal. Pontine myelinolysis is often associated with hyponatremia.[2]

The second point is that various other differential diagnoses have not been sufficiently ruled out. Of particular importance is the exclusion of cerebral manifestations of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (SC2I).[3] Were both patients tested for SC2I on admission and during hospitalization? SARS-CoV-2 vaccinations (SC2Vs) do not necessarily protect against SC2I.

The third issue refers to the examination of the cerebrospinal fluid (CSF) which was inadequate. We should know whether there was pleocytosis, positive oligoclonal bands, a disruption of the blood-brain barrier or autochthonous immunoglobulin G production. What was the CSF culture result and the results of the virus panel, including SARS-CoV-2?

The fourth point is that no evidence has been provided as to why the reported lesions are actually areas of demyelination.[1] Glucocorticoids are not only effective in pontine myelinolysis but also in a number of other CNS disorders associated with SC2V. These include acute disseminated encephalomyelitis, acute hemorrhagic leukoencephalitis, acute hemorrhagic necrotizing encephalopathy, multiple sclerosis, myelin oligodendrocyte glycoprotein antibody-associated disease, autoimmune encephalitis, and neuromyelitis optica spectrum disorder.

The fifth point is the discrepancy between the discussion, which mentions that the ADC maps were isointense, and the legend of Figure 1, which states that the ADC map was hyperintense.[1] Hyperintense DWI and hypointense ADC suggest cytotoxic edema rather than vasogenic edema. This discrepancy should be clarified. One limitation in this regard is that the DWI and ADC sections shown in the figure do not always match.

In summary, alternative causes must be carefully excluded before attributing cerebral lesions to a complication of SC2V.

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