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Letter to the Editor

Acute, disseminated encephalomyelitis incidentally or causally related to SARS-CoV-2

Josef Finsterer
Neurology & Neurophysiology Center, Vienna, Austria

Corresponding Author: **Josef Finsterer**

We read with interest the article by Yen *et al*. about a 59 years-old male with the diagnosis SARS-CoV-2 related acute disseminated encephalomyelitis (ADEM) and concomitant critical ill polyneuropathy (CI-PNP) who was admitted for rehabilitation after prolonged hospitalisation during 50 days [1]. The patient profited significantly from the applied programs regarding motor and cognitive functions as assessed 9 months after onset [1]. The study is appealing but raises concerns that should be discussed.

We disagree with the diagnosis SARS-CoV-2 related ADEM [1]. The latency between onset of COVID-19 and diagnosis of ADEM was 43 days [1]. Such a latency is too long to convincingly establish a causal relation. Although a causal connection cannot be definitively ruled out, such a relation is highly unlikely.

We also disagree with the diagnosis ADEM [1]. ADEM was diagnosed upon the clinical presentation and the cerebral magnetic resonance imaging (MRI) findings. To diagnose ADEM, it is crucial to investigate the cerebrospinal fluid (CSF) but these results were not provided [1]. CSF findings in ADEM include pleocytosis, elevated protein, elevated IgG index, and positive oligoclonal bands.

Because the patient experienced a sepsis during hospitalisation, it is crucial to rule out sepsis associated encephalopathy (SAE). SAE is clinically characterised by focal neurological deficits and pathophysiologically by inflammation with endothelial/microglial activation, increased permeability of the blood-brain barrier (BBB), hypoxia, imbalance of neurotransmitters, glial activation, and neuronal loss [2]. SAE may mimic ADEM clinically, on imaging, and regarding the CSF findings. CSF findings in patients with SAE may show elevation of neurofilament light chain (NFL) levels and elevation of soluble triggering receptor expressed on myeloid cells 2 (sTREM2) [3].

Another differential not appropriately ruled out is cerebral vasculitis. Cerebral vasculitis has been reported as a complication of SARS-CoV-2 infections [4], why it is essential to rule out vasculitis by determination of a vasculitis panel, CSF investigations, and magnetic resonance angiography (MRA), black blood sequences, respectively conventional digital subtraction angiography (DSA). Ruling out these differential diagnoses is crucial as the prognosis and outcome may differ significantly between them.

Because ADEM is per definition affecting the spinal cord and appropriate MRI is warranted to rule out or confirm a spinal cord lesion.

The patient was diagnosed with steroid myopathy [1]. We should be informed upon which criteria this diagnosis was established and if the patient had a myopathic needle electromyography (EMG) or a muscle biopsy compatible with a mitochondrial myopathy.

Overall, the interesting study has some limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Before diagnosing SARS-CoV-2 related ADEM, a causal relation between the virus and the cerebral abnormalities needs to be established and various differential diagnoses of ADEM need to be ruled out.

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