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Letter to the Editor

The spectrum of neuro-COVID is broader than is often propagated

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We read with interest the review article by Brola *et al.* about the neurological side effects of SARS-CoV-2 infections ^[1]. The authors included and discussed the most relevant neurological complications after an infection with SARS-CoV-2 which were reported in the literature ^[1]. It was concluded that neurological manifestations of SARS-CoV-2 infections can be the initial clinical manifestation of the disease, can even dominate the clinical presentation in the later disease courses, and can be the dominant abnormality in long-COVID syndrome ^[1]. The study is appealing but raises concerns that should be discussed.

The main limitation of the study is that a number of neurological complications of SARS-CoV-2 infections were not mentioned without explaining why not the entire spectrum of neurological SARS-CoV-2 associated disease was included ^[1]. Central nervous system (CNS) diseases that were not mentioned or discussed in the review include subarachnoid bleeding ^[2], opsoclonus myoclonus syndrome ^[3], acute, hemorrhagic, necrotising encephalopathy (AHNE) ^[4], acute, hemorrhagic, leucoencephalitis (AHLE) ^[5], reversible cerebral vasoconstriction syndrome (RCVS) ^[6], pituitary apoplexy ^[7], Tolosa-Hunt syndrome (THS) ^[8], cerebellitis, brainstem Bickerstaff encephalitis (BBE) ^[9], rhomb-encephalitis ^[10], limbic encephalitis ^[11], cerebral vasculitis ^[12], neuromyelitis optic spectrum (NMO) spectrum disorders ^[13], and microbleeds ^[14]. Peripheral nervous system (PNS) disorders following a SARS-CoV2 infection and not mentioned in the review are immune plexitis, also known as Parsonage Turner syndrome (PTS) ^[15], small fiber neuropathy ^[16], dermatomyositis ^[17], myositis ^[18], and rhabdomyolysis ^[19]. SARS-CoV-2 may not only deteriorate a pre-existing neurological condition, such as myasthenia or multiple sclerosis, but may also trigger new onset myasthenia or multiple sclerosis ^[20].

A further limitation is that the term “encephalopathy” was commonly used without providing a definition of this term. We should know whether the authors mean epilepsy, cognitive impairment, disorientation, or others with this term.

Missing is a discussion of SARS-CoV_2 associated immune, thrombotic thrombocytopenia (ITTP) as the cause of venous sinus thrombosis (VST). There is also no mentioning of cardiac involvement in a SARS-CoV-2 infection which can secondary cause embolic stroke due to thrombus formation in the atria, ventricles or peripheral veins in case of a foramen ovale.

Overall, the interesting study has limitations that call the results and their interpretation into question. Addressing these limitations could further strengthen and reinforce the statement of the study. A review about the neurological complications of SARS-CoV-2 infections should be compulsory and comprehensive to demonstrate the broad spectrum of complications and to guide clinicians on how to approach the diagnostic and therapeutic management of these complications.

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