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The spectrum of neuro-COVID is broader than anticipated

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With interest we read the review article by Zhang et al. about the neurological implications and manifestations of an infection with SARS-CoV-2 (neuro-COVID) [1]. The authors intended to summarise and discuss the significance of the central and peripheral nervous system (CNS, PNS) invasion by the virus and to improve the understanding of how to manage neuro-

COVID [1]. We have the following comments and concerns.

The comprehensive review lacks discussing a number of CNS and PNS manifestations of neuro-COVID, which should be additionally considered by those involved in the management of COVID-19 patients. There is increasing evidence that that the infection with SARS-CoV2 may also cause CNS disorders such as myoclonus-ataxia syndrome, acute, disseminated encephalomyelitis (ADEM), limbic encephalitis, psychosis, intracerebral bleeding, sinus venous thrombosis, vasoconstriction syndrome, optic neuritis, or cerebral vasculitis [2]. Ischemic stroke may not only be due to hypercoagulability or sinus venous thrombosis, but also due to heart failure or arrhythmias, particularly atrial fibrillation. PNS disorders not addressed in the review were myasthenia, single or multiple cranial nerve palsies, and trigeminal neuralgia.

Missing is a discussion about myositis and dermatomyositis as a manifestation of neuro-COVID. Though there is mentioning that the skeletal muscle can be damaged by the virus, it is not addressed that the infection may cause viral myositis, which may clinically manifest as muscle weakness, myalgia, generalised fatigue, or exercise intolerance. Mentioning myositis is crucial as it has been reported to occur in 11-50% of the cases [3] and requires analgesic, anti-inflammatory, or immune-modulating treatment and physiotherapy.

Missing in the review are the neurological complications of drugs given to treat the viral infection. A number of agents applied for the treatment of COVID-19 are neurotoxic. Differentiating primary manifestations of the viral infection from side effects of the treatment is crucial as most of these neurological side effects disappear with discontinuation of the causative agent. Neurotoxic drugs frequently applied for the treatment of COVID-19 include chloroquine, remdesivir/lopinavir, azithromycin, and tocilizumab. From chloroquine it is known that it can trigger myopathy or myasthenic syndrome [4]. Remdesivir, lopinavir, and ritonavir may occasionally trigger myopathy or rhabdomyolysis. Azithromycin may induce rhabdomyolysis. Tocilizumab is known for muscle aching and muscle cramps as rare side effects. Steroids may induce mitochondrial myopathy [4]. Patients requiring ICU treatment may develop critical ill neuropathy or myopathy.

Missing in the review are the neurological complications of primary involvement of the heart and vessels in the SARS-CoV-2 infection. It is well appreciated that SARS-CoV-2 may go along with myocarditis [5]. Complications of myocarditis may not only be heart failure but also conduction defects or arrhythmias, including atrial fibrillation. From heart failure or

arrhythmias it is known that they may be complicated by cardio-embolic events, including ischemic stroke. Thus it is crucial to investigate patients with COVID-19 cardiologically if CNS abnormalities, particularly embolic stroke, occur.

There is also no discussion about neurological manifestations as the initial manifestation of COVID-19. Knowing that COVID-19 can start with CNS/PNS disease prior to manifestations in the lungs or other organs is crucial not to overlook the ongoing infection but also to prevent the further spreading of the pandemic.

Overall, the review has a number of limitations, which should be addressed to further strengthen the results and discussion. Neurological involvement in COVID-19 is evidently broader than anticipated and neurological manifestations may be the initial manifestation of the infection in some cases.

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