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Letter to the Editor

Post-Acute Neuropsychological Deficits after SARS-CoV-2 Infections Vary Considerably Depending on Multiple Influences

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We read with interest the article by Prabhakaran *et al*. reporting on a study of the post-acute neuropsychological deficits in 205 SARS-CoV-2 positive patients using an electronic questionnaire [1]. Tests performed included the Neuropsych Questionnaire-45 (NPQ-45), the Medical Outcome Survey (MOS), and the posttraumatic stress disorder (PTSD) civilian checklist (PCL-C17) [1]. Three different clusters of neuropsychological deficits were identified ("normal cognition", "memory speed impaired", "dysexecutive") [1]. Recovery outcomes varied between the three groups, but all improved at 6 months [1]. It was concluded that there are multiple post-acute neurophenotypes of COVID-19, with distinct etiological pathways and recovery outcomes. The study is excellent but has limitations that should be discussed.

The major limitation of the study is that the neuropsychological tests were conducted through an electronic platform rather than in person. It is therefore conceivable that it was not the patient himself who filled out the form, but a relative, friend, caregiver, or child. In addition, no guarantee can be given that the questions have been answered correctly, carefully, and conscientiously.

Another limitation is that the cognitive status before infection was not checked in the included patients. Although the history of severe neurocognitive disorders was negative [1], this does not rule out the possibility that neurocognitive deficits were present on detailed neuropsychological tests.

A third limitation of the study is that the current medication that the included patients were taking at the time the test was performed was not included in the analysis. Because several drugs can severely impair neurocognitive function, it is important to know how many of them were taking sedatives, hypnotics, anxiolytics, antidepressants, antipsychotics at the time of the study. There is also no mention of how many of the included patients used illicit drugs on a regular basis.

A fourth limitation is that neurological and psychiatric abnormalities occurring during the acute SARS-CoV-2 infection were not included in the evaluation. There is no mention how many of the included patients experienced stroke, cerebral bleeding, venous sinus thrombosis (VST), reversible cerebral vasoconstriction syndrome (RCVS), meningitis, infectious or immune encephalitis, ventriculitis, acute, disseminated encephalomyelitis (ADEM), acute, hemorrhagic, necrotizing encephalitis (AHNE), cerebral vasculitis, multiple sclerosis, neuromyelitis opitca (NMO)-related disorders, myelin-oligodendrocyte glycoprotein (MOG) associated disease (MOGAD), pontine myelinolysis, posterior reversible encephalopathy syndrome (PRES), or pituitary apoplexy during the acute stage of the infection. There is also no mention how many of the included patients experienced psychiatric disease during the acute stage of the infection, such as "altered mental state", anxiety, sleep disorders, depression, delirium, isolated hallucinations, mania, akinetic mutism, psychosis, eating disorders, or autism spectrum disorders.

Overall, the interesting review has limitations that call the results and their interpretation into question. Addressing these issues would strengthen the conclusions and could improve the status of the study. Neuropsychological deficits within 12 weeks after SARS-CoV-2 infection are highly dependent on whether neurological and psychiatric disorders develop in the acute stage of infection. Neuro-COVID, psycho-COVID, current medication, and comparison with pre-infection status are required to assess the damage to neuropsychological abilities caused by the infection.

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