

Letter to the editor

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Neuro-COVID is a Common Complication of SARS-CoV-2 Infections and Merits Prospective Evaluation

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We read with interest the article by Tan et al. on a cross-sectional, observational, mono-center study of 156 COVID-19 patients [1]. Among these 156 in-hospital patients collected between July and December 2021, 23.7% had neurological complications from the infection, which included stroke, encephalitis, or encephalopathy [1]. Diabetic patients were at an increased risk for neuro-COVID. Unvaccinated patients had a 4.25 fold increased risk of developing neurological complications compared to vaccinated patients [1]. The risk of developing neurological complications from COVID-19 was increased by 18% among those with leucocytosis [1]. It was concluded that patients with diabetes, leucocytosis, and being unvaccinated are at increased risk of developing neuro-COVID. The study is excellent but has limitations that raise concerns and require discussion.

We disagree that muscle weakness and numbness are exclusively manifestations of ischemic stroke. Muscle weakness and sensory disturbances are not only due to central nervous system (CNS) disease but also involvement of the peripheral nervous system (PNS). We should know whether all patients with weakness and numbness also had a stroke on cerebral imaging. Did truly all patients undergo cerebral imaging when stroke was suspected?

It is not comprehensible why patients with seizures were delineated from those with status epilepticus. Status epilepticus is a seizure type, why these two entities should not be evaluated separately. Surprisingly, not a single patient with PNS disease was detected among the 156 COVID-19 patients. We should know the reason why no patients with Guillain-Barre syndrome plexitis, large or small fiber neuropathy, myasthenia, myopathy, myositis, or rhabdomyolysis was detected. Were the included patients not prospectively investigated for PNS disease?

A stroke mechanism not considered in the study is endotheliopathy. There are indications that SARS-CoV-2 infections can be complicated by endothelialitis, which increases the risk of thrombosis and vessel occlusion. Another pathophysiological mechanism not considered is arterial dissection. Supposing that SARS-CoV-2 causes damage of the vascular wall, it is conceivable that also dissection occurs. Several patients with dissection during a SARS-CoV-2 infection have been reported [2].

We disagree with the notion that critical illness polyneuropathy is a direct consequence of a SARS-CoV-2 infection [1]. Critical illness polyneuropathy is iatrogenic and therefore

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should be excluded from the evaluation. Leucocytosis is multifactorial. Therefore, we should know how alternative causes of the COVID-19 infection were ruled out as causes of leucocytosis. We should know how many of the included patients were smokers, received steroids, were under stress, had a chronic infection, colitis ulcerosa, a fungal infection, a parasitic infection, were pregnant, had an allergy, or had a malignancy.

A limitation of the study is that no definition of the term “encephalopathy” was provided. It should be explained whether encephalopathy means seizures, leukoencephalopathy, encephalitis, confusion, or neuropsychological deficits in the absence of morphological abnormalities on cerebral imaging. If encephalopathy is regarded as a post-ictal condition, it should be told if these patients were classified into both the “seizures” group and the “encephalopathy” group. If encephalopathy resulted from metabolic dysfunction, we should be informed what kind of metabolic derangement resulted in encephalopathy. How many of the included patients had diabetes or hyperglycemic crisis on admission?

Seizures in COVID-19 patients are commonly symptomatic manifestations of stroke, encephalitis, or venous sinus thrombosis (VST). Therefore, we should know how many of those with seizures also had morphological abnormalities on MRI.

The sentence “Anosmia and dysgeusia were less associated with neurological complications” is not comprehensible because anosmia and dysgeusia are classical neurological complication already.

Overall, the interesting study has limitations that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Neurological complications of SARS-CoV-2 infections are manifold and may be recognised only if an appropriate number of patients is investigated or if multi-center or multinational studies with a high number of patients are undertaken and if COVID-19 patients are prospectively and comprehensibly investigated for CNS and PNS disease having started after onset of COVID-19.

Keywords: SARS-CoV-2, COVID-19, complication, neuro-COVID, stroke, brain, nerve.

Declarations

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