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SARS-CoV-2 infection complicated by neuro- or psycho-COVID

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To the Editors

We read with interest the review article by Czarnowska et al. on the effects of SARS-CoV-2 on the nervous system [1]. The pathophysiology of neuro-COVID has been explained by a systemic immune reaction against the virus (platelet activation, thrombocytopenia, leukocyte activation, overproduction of inflammatory pro-aggregating cytokines, endothelial dysfunction, complement system activation, mast cell activation), microglial activation, cerebral hypoxia, or neuro-invasion of the virus via the olfactory tract, invasion of the virus into the central nervous system via haematogenous pathways, and disruption of the blood brain barrier allowing leukocytes carrying SARS-CoV-2 to enter the brain (the so-called 'Trojan horse' mechanism) [1]. The authors concluded that cerebrovascular disease is the most common neurological complication of SARS-CoV-2 infection [1]. The review article is excellent, but we feel it has limitations and raises concerns that should be discussed.

Specifically, we disagree with the subheading 'Neurological symptoms' and the heading given to Table 2: 'Neurological symptoms during acute/post-acute stage of infection in large analyses of patients hospitalised due to COVID-19' [1]. Contradicting these captions, this section and this Table actually discuss neurological disorders rather than neurological symptoms.

With regard to neurological diseases, the spectrum of neuro-COVID is much broader than discussed in the review. The neurological complications of SARS-CoV-2 infection not included in the review are: immune encephalitis, cerebellitis, acute disseminated encephalomyelitis (ADEM), acute, haemorrhagic, necrotising encephalitis (AHNE), pontine myelinolysis, cerebral vasculitis including giant cell arteritis, ventriculitis, hypophysitis, intracerebral bleeding, demyelinating disorders (multiple sclerosis, neuromyelitis optica (NMO) spectrum disorder, myelin-oligodendrocyte glycoprotein (MOG) associated disease), reversible, cerebral vasoconstriction syndrome (RCVS), Wernicke encephalopathy, transverse myelitis, mono- or polyneuritis cranialis including optic neuritis, Parsonage-Turner syndrome, lumbosacral plexitis, myasthenia gravis, myositis, and rhabdomyolysis [2].

Since the review article is entitled 'Impact of SARS-CoV-2 on the nervous system', we contend that psychiatric sequelae of SARS-CoV-2 infection should also have been included and discussed. In addition to 'altered mental state', 'anxiety', 'sleep disorders', and 'decreased mood', patients suffering a SARS-CoV-2 infection may also, or instead, develop delirium, isolated hallucinations, mania, akinetic mutism, psychosis, eating disorders, or autism spectrum disorders [3].

We disagree with the notion that cerebral hypoxia occurs due to SARS-CoV-2 infection [1]. Hypoxic brain damage has rarely ever been documented by cerebral MRI in COVID-19 patients, and where it does occur it is usually in patients with severe COVID-19 requiring prehospital bystander resuscitation. In-hospital COVID-19 patients with respiratory failure are usually given timely oxygenation by non-invasive

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means or invasive means by mechanical ventilation. Furthermore, cerebral hypoxia does not explain peripheral nervous system complications after SARS-CoV-2 infection.

In summary, this interesting study has some shortcomings that call into question the results and their interpretation. Addressing these weaknesses would strengthen the conclusions and thereby improve the study.

The spectrum of neuro- and psycho-COVID is broader than has been commonly assumed. Discussing the effects of SARS-CoV-2 on the central and peripheral nervous systems requires taking into consideration the full spectrum of neurological and psychiatric sequelae in order to shed more light on the possible underlying pathophysiological mechanisms.

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