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Neuroradiology

A little consideration for “Rare presentations of COVID-19: PRES-like leukoencephalopathy and carotid thrombosis”

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Dear Editor:

We are grateful to Doo FX et al. for their research, which greatly helped us understand clinical manifestations of posterior reversible encephalopathy syndrome (PRES), a rare neurological disease in the COVID-19 patient.¹ However, in this letter, we want to put forward a different view on the pathogenic mechanism of PRES in the patient.

On the one hand, previous studies have concluded that transient acute kidney injury creates an environment of cerebral hyperperfusion, which may be involved in formation of PRES. At the same time, the posterior part of the cerebral hemisphere is more susceptible to high blood perfusion to form vasogenic edema because the sympathetic nervous system of the vertebrobasilar artery is weaker than that of the internal carotid artery system. The posterior circulation is more prone to vasodilation than the anterior circulation.^{2–3} It is consistent with the symmetrical parieto-occipital white matter hyperintensity found in the magnetic resonance imaging (MRI) of the PRES patient.

On the other hand, immunomodulatory drugs can cause vascular endothelial dysfunction, which may trigger the occurrence of PRES.² The patient was given two doses of tocilizumab due to his serious illness. With the use of these drugs, the vascular endothelium was destroyed by the presence of exogenous toxins, making the presence of the vasoconstrictor substance in the vascular endothelium released, and then vasospasm occurs, which destroys the stability of the blood-brain barrier and creates conditions for the emergence of PRES.^{2–3}

It is worth noting that both acute kidney injury and the use of tocilizumab before epilepsy symptoms associated with PRES. Judging from the order of time, this seems to support the two mediation of formation of PRES. More interestingly, there are also several similar cases reported

in the COVID-19 pandemic. Some patients with SARS-CoV-2 infection, who have suffered acute kidney injury or have used interleukin inhibitors such as anakinra and tocilizumab, were finally confirmed to have typical vasogenic edema by brain MRI, which is consistent with PRES.^{4–6} This suggests that the two may be potential risk factors for the development of PRES.

Therefore, we believe that combined effects of three factors including acute kidney injury, use of immunomodulatory drugs such as tocilizumab, and invasion of COVID-19 co-created the environment for the arrival of PRES, rather than simply caused by the direct effects of SARS-CoV-2 infection in the case.

Declaration of competing interest

The authors declare that they have no competing interests.

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Statistical analysis

No statistical analysis.

Authors' contribution statements

FT designed and supervised the overall research. DS wrote the first

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