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Letter response to the brain after COVID-19: Compensatory neurogenesis or persistent neuroinflammation?

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After the outbreak of COVID-19, we conducted a DTI-related research to explore the cerebral micro-structural changes in recovered COVID-19 patients. According to our results, we supposed that the regional volumetric enlargement was caused by neurogenesis and hypertrophy, and remyelination possibly existed in the white matter pathways of these patients [1]. E. Goldberg, et al. proposed persistent neuroinflammation as an alternative explanation.

In our study, the COVID-19 and non-COVID-19 patients were matched for age and sex. The patients with premorbid neurocognitive diseases were excluded at the beginning. Accordingly, no volunteers with such conditions were recruited. Thus, the impact of underlying neurocognitive conditions was ruled out. We agree that the result could be more convincing if the GMV (grey matter volume)-related factors including education, physical activity and BMI, were matched or adjusted [2,3].

The regional increased GMV and decreased MD/AD values could result from neuroinflammation in the acute or chronic stage. The inflammatory response to neurotropic coronavirus infection was reported to reach peak within the first month and could last for 2–3 months [4]. Concerning the fact that the patients in our study were collected 3 months after recovery (twice PCR negative tests, mean duration from the onset to the date of MRI scans, 97.46±8.01 days), we thought it was hard to attribute the GMV enlargement to persistent inflammation.

However, our sample size was quite limited. It is encouraged to explore the dynamic cerebral changes in COVID-19 patients and their relationship with neurological manifestations and cognitive behaviors in the future with larger samples.

Declaration of Competing Interest

The authors have nothing to declare.

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