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Post-Acute-COVID-19-Illness Neuropsychiatric Sequelae

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Editorial
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Introduction

Direct viral invasion, neurodegeneration, microvascular thrombosis, neuroinflammation, and severe systemic inflammation can be the causes of post-acute-COVID-19-illness neuropathology [1-4], supported by brain parenchyma and vessel changes of possibly driven inflammation in neurons, supportive cells, and brain vasculature in COVID-19 autopsy series [5,6]. A role in persistent brain effects of SARS-CoV-2 (COVID-19) may be played by an accumulation of memory T cells, a biomarker of immunosenescence in tissue injury and aging, accompanying with the decreased ability to respond to new antigens that are demonstrated in chronic low-level brain inflammation [7]. Cognitive-behavioral changes directly associated with the levels of immune activation [8]. Passive diffusion and axonal transport via the olfactory complex, viral invasion in the extracellular spaces of olfactory epithelium and dysfunctional lymphatic drainage from circumventricular organs [9,10]. Elevated peripheral blood levels of neurofilament light chain, a biomarker of brain injury with a more sustained increase in severe infections has been identified in post-acute-COVID-19-illness phase. Post-traumatic-stress disorder (PTSD) or deconditioning may be mechanisms that are hypothesized in critically ill COVID-19 patients with post-acute-COVID-19-illness brain fog whereas dysautonomia may be the cause of post-acute-COVID-19-illness brain fog in previously mild-COVID-19 patients [11-15]. Approximately, 20 %-40 % of patients with previously critical-COVID-19 illness demonstrated long-term cognitive impairment [16]. Non-restorative sleep, depressive symptoms, diffuse myalgia, post-viral syndrome of chronic malaise late-onset headaches ascribed to high cytokine levels, and migraine-like headaches (frequently refractory to traditional analgesics have been reported in post-COVID-19 survivors. Around 38 % of post-acute-COVID-19-illness patients had ongoing headaches after 6 weeks [17-22]. At up to 6 months follow-up, approximately, 10 % of post-acute-COVID-19-illness survivors may persist loss of smell and taste [23-26]. Ischemic or hemorrhagic stroke hypoxic-anoxic brain damage, posterior reversible encephalopathy syndrome and acute disseminated myelitis [27-30], may contribute to required-extensive-rehabilitation permanent or lingering neurological deficits.

Conclusion

In conclusion, further studies are urgently needed to identify the exact mechanisms and biomarkers of the neurological post-acute-COVID-19-illness sequelae.

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