

Neuropsychiatric and Cognitive Sequelae of SARS-CoV2 Infection: Is there a Risk of Dementia?

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The infection originated in the city of Wuhan, China by the SARS-CoV2 virus, called by the World Health Organization (WHO) as COVID-19 and spread worldwide in the first pandemic of the 21st century, has reached to date the figure of 122 million people infected [1].

Although the symptoms are mainly respiratory, other symptoms have been reported in different body systems including the central nervous system: depression, anxiety, insomnia, headache, delirium, hypogeusia/dysgeusia, hyposmia/anosmia, seizures, encephalitis, myelitis, encephalopathy, generalized myoclonus, cerebrovascular events, and neurogenic respiratory failure, among others [2,3]. A recent meta-analysis showed that 65 to 67% of intensive care unit patients diagnosed with severe COVID 19 developed confusion and agitation and 33% had dysexecutive syndromes at discharge [4].

Based on infections caused by other strains of coronavirus, including Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome (MERS), different pathophysiological mechanisms have been proposed to explain neuropsychiatric and cognitive symptoms. Possible pathophysiological mechanisms includes direct lesions by the virus since it is neurotropic, exaggerated immune response, neuroinflammation and increased permeability of the blood-brain barrier; predilection of the virus for olfactory epithelium, bulb and vagal centers, forebrain, basal ganglia and hypothalamus, cerebral hypoxia, increased intra-cranial pressure, vascular congestion, vasodilation, cascade of the cytokine system, systemic inflammatory response syndrome, increased IL-15 release, as well as vascular and endothelial damage [5,6].

Brain imaging abnormalities on nuclear magnetic resonance such as parenchymal abnormalities, micro and macro subcortical hemorrhages, nonspecific changes in white matter and acute demyelinating lesions have been reported [7].

We are currently recognizing the acute neuropsychiatric effects caused by the virus, but we do not know the possible long-term sequelae. Based on data from other infections with Covid viruses, in which post-infection cognitive symptoms have occurred, it is possible to think that a neurodegenerative process take place in the brain of these patients. Also, we must consider that immune and neuronal cells can host latent Covid viruses, so long term symptoms can occur [8].

If some risk factors for dementia such as cardio and cerebrovascular pathology, delirium (present in up to 84% of ICU patients with severe Covid -19), small vessel disease, and brain atrophy, occur in the patients with SARS-CoV2 infection, it is not unreasonable to think that the disease could be a risk factor for post-infection cognitive decline.

In fact, the World Health Organization has created an international multidisciplinary consortium, with the participation of 30 countries, to study the short and long-term consequences of SARS-CoV2 in the central nervous system, with follow-ups at 6, 9 and 18 months after the discharge of the patients [7].

The enemy is here, maybe it will leave soon, but it seems that it's sequelae will remain for a long time in our patients.

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