



Long-term Effects on the Nervous System of COVID-19 Patients

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DESCRIPTION

The effects of COVID-19 and the scientific data supporting its involvement in the kidneys, intestines, lungs, and heart are extended to the brain, highlighting neurological problems and the disease's high rates of morbidity and mortality. In China, 214 hospitalized COVID-19 patients were investigated at the start of the pandemic, and 78 (36.4 percent) of these patients had neurological symptoms [1]. Understanding the SARS-CoV-2 pathophysiology with relation to contamination and damage to the nervous system is therefore essential for clinical and therapeutic methods that may lessen the disease's impact. In this regard, it's critical to emphasize that, in addition to the disease's direct impact on neurons; inflammation brought on by immune system dysregulation brought on by infection is a major contributor to the disease's neurological symptoms.

There are currently six species of coronaviruses that can infect humans, four of which are known to cause cold syndromes and mild upper respiratory tract infections in immunocompetent patients of any age, and two of which are known to cause major respiratory syndromes: SARS-CoV, the coronavirus that causes Severe Acute Respiratory Syndrome, and MERS-CoV, the coronavirus that causes Middle Eastern Respiratory Syndrome. The SARS-CoV-2 is the sixth coronavirus that is currently identified, and it is not just the SARSCoV-2 that exhibits neurotropism. In addition to the recently reported cases of myelitis, other members of the family, such as SARS-CoV and MERS-CoV, are also capable of affecting the Nervous System (NS) and causing illnesses similar to those already reported in COVID-19 patients, such as encephalitis, strokes, infectious toxic encephalopathy, and Guillain-Barre syndrome [2].

There are various disease-related elements that have an impact on the neurological repercussions. In addition to the tropism of SARS-CoV-2 by ACE-2 receptors, the virus's presence in the bloodstream, its capacity to directly enter the Nervous System (NS) through peripheral nerves, and the blood-brain barrier's thinning all contribute to the appearance of brain manifestations that are linked to metabolic issues in the autoimmune processes brought on by viral clinical condition [3]. Sars-Cov-2 can infect NS through the bloodstream like other infections can. Although there is a barrier between blood flow and neural tissue, this defense is compromised by the viral infection's enhanced inflammatory response. The activation of metalloproteinase, which are enzymes capable of breaking down the proteins that make up the blood-brain barrier and thereby increasing its permeability, plays a crucial role in the recruitment of white blood cells, such as neutrophils, associated with the known cytokine storm, especially with the release of IL-6, interleukins, and other factors. Post-mortem microscopic results in individuals with neurological symptoms and positive SARS-CoV-2 RT-PCR have already shown that the virus migrates from the endothelium to neuronal cells concurrently with the presence of viral particles within neural cytoplasmic vacuoles. The cerebral microcirculation is a key factor in the coupling of the glycoprotein S (spike) to the functional ACE-2 receptor, allowing penetration into neurons and glial cells and resulting in neurological infection [4]. Systemic circulation plays a fundamental role in viral dissemination in the neurological context. The brain tissue is known to be a target for SARS-CoV-2 through these similar receptors, permitting the expansion of the clinical spectrum of COVID-19 to neurological indications. ACE-2 receptors are present in greater abundance in the alveolar epithelium of the lungs.

CONCLUSION

We emphasize that the amount of drugs administered in intensive care units can also interfere with neurological conditions and any comorbidities the patient may have, even though it is still unclear whether the impairment of the nervous system is a result of direct infection by SARS-CoV-2 or of a diffuse inflammatory process affecting various organs and systems and generating multiple manifestations, including those of the nervous system. In order to enhance the patients' prognosis, it is important to evaluate hospitalized patients with neurological abnormalities brought on by COVID-19, change protocols, and teach healthcare staff.

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REFERENCES

- Mao L, Jin H, Wang M, Hu Y, Chen S, He Q, et al. Neurologic Manifestations of Hospitalized Patiens with Coronavirus Disease 2019 in Wuhan, China. JAMA Neurol. 2020;77: 683-689.
- 2. Wu Y, Xu X, Chen Z, Duan J, Hashimoto K, Yang L, et al. Nervous system involvement after infection with COVID-19 and other

coronaviruses. Brain Behav Immun Health. 2020;87: 18-22.

- 3. Berger JR. COVID-19 and the nervous system. J Neurovirol. 2020;26: 143-148.
- Netland J, Meyerholz DK, Moore S, Cassell M, Perlman S. Severe acute respiratory syndrome coronavirus infection causes neuronal death in the absence of encephalitis in mice transgenic for human ACE2. J Virol. 2008;82: 7264-7275.