

Issues with Neurological Manifestation in COVID-19 Infected Patients

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Perspective

Coronavirus disease in children usually has a mild infectious illness course, but when both acute infection and associated phenomena such as the multisystem inflammatory syndrome in children are present, serious effects might occur (MIS-C). Adults have reported headaches, seizures, peripheral neuropathy, stroke, demyelinating disorders, and encephalopathy, among other neurological problems. COVID-19 symptoms, such as pulmonary and cardiac signs, manifest differently depending on age and underlying comorbidities. This information is based on literature reviews and primary data collected at New York Presbyterian Morgan Stanley Children's Hospital. It gives a quick overview of the neurodevelopmental disorders seen in the context of COVID-19, as well as potential mechanisms and long-term implications of COVID-19 in the paediatric population.

For neurological patients and their careers, the COVID-19 pandemic, which was caused by the SARS-CoV-2 virus, has left many unanswered questions. COVID-19 can produce severe symptoms in the elderly and immunocompromised individuals, and it can exacerbate symptoms of underlying neurological illness, particularly in those with significant bulbar and respiratory weakness or other neurologic dysfunction. Patients and clinicians may be concerned about the vaccination's theoretical hazards, such as vaccine safety and efficacy in the context of immunotherapeutic, as well as the possibility of inducing or aggravating neurological symptoms.

The present literature is summarised in this statement on behalf of the AAN Quality Committee, with a focus on COVID-19 infection in adults with neurological disease to highlight the risks and benefits of immunisation in this population. Based on the current data, neurologists should encourage their patients to get vaccinated against COVID-19. Immunotherapy patients should be aware of the importance of vaccine timing in relation to their medication and the danger of a compromised immune response. In December 2019, a disease outbreak driven by a novel coronavirus (2019-nCoV, later termed SARS-CoV-2) was reported in Wuhan, China. Coronavirus disease 2019 (COVID-19) quickly spread over the world, becoming a pandemic. COVID-19 symptoms include fever, dry cough, exhaustion, and respiratory discomfort.

SARS-CoV-2 infection can also affect both the central and peripheral nervous systems. These neurological changes could be caused by viral neurotropism, a hyperinflammatory and hypercoagulable disease, or even mechanical ventilation-related dysfunction. Hypoxia, endothelial cell damage, and the impacts of various ventilatory procedures may all increase stress and strain, resulting in an inflammatory response and a complex connection between the lungs and the brain. There hasn't been any investigation into the possibility of mechanical breathing having a secondary influence on brain repair and results. The purpose of this information is to present a current overview of the putative pathogenic pathways of COVID-19 neurological symptoms,

explain physiological concerns linked to brain-lung interactions, and propose strategies for enhancing respiratory support in critically ill patients.

There has been mounting evidence that SARS-CoV-2 infection is linked to a variety of neurological symptoms, including acute cerebrovascular events, since the coronavirus disease 2019 (COVID-19) pandemic, which was caused by infection with the Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) virus (i.e., stroke and cerebral venous thrombosis). These occurrences can occur before, during, or even after the onset of COVID-19's fundamental symptoms. Although the mechanisms underlying COVID-19-related cerebrovascular issues are unknown, hypercoagulability, inflammation, and altered Angiotensin, Converting Enzyme 2 (ACE-2) signalling in combination with SARS-CoV-2 could all play a role. ACE-2 is required for the proper functioning of the heart and brain.

As the number of published COVID-19 cases with peripheral vascular events grows, randomised trials would help gather more valuable insights into the pathophysiology of peripheral vascular events, effective therapies, and factors predicting poor functional outcomes related to such events in COVID-19 patients. In most situations, a severe acute respiratory infection in a child results in minor or no respiratory symptoms. The multisystem inflammatory syndrome in children is a dangerous illness that affects some paediatric patients and is linked to a high mortality rate (MIS-C). In both cases, neurological symptoms have been reported. There have been cases of children with major neurological abnormalities and a positive SARS-CoV-2 test at the same time. A literature search was carried out between March 2020 and May 2021.

Although SARS-CoV-2 is primarily focused on the respiratory tract, there is evidence that the virus can infect the Central Nervous System (CNS) and Peripheral Nervous System (PNS), causing a variety of neurological problems, including serious complications such as seizures, encephalitis, and loss of consciousness. We provide a thorough examination of SARS—currently CoV-2's recognised involvement, as well as a comprehensive list of all neurological problems described in COVID-19 case reports from around the world. The virus could enter the CNS by olfactory neurons or damaged endothelium in the brain microvasculature via the ACE2 receptor, which is potentiated by neuropilin-1, or through damaged endothelium in the brain microvasculature via the ACE2 receptor, which is potentiated by neuropilin-1 (NRP1) [1-5].

In certain COVID-19 patients, the most serious symptom appears to be spontaneous breathing cessation. This could indicate an issue with the cardiopulmonary regulatory centres in the brainstem. These ground breaking studies lay the groundwork for future in-depth basic and clinical research that will be required to demonstrate the role of SARS-CoV-2 infection in the neurodegeneration of critical brain regulatory regions.

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