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Perspective

Neurological Manifestation Issues in Patients with COVID-19 Infection

Edward Smith*

Department of Neurology, University of Arkansas for Medical Sciences, Little Rock, Arkansas, USA

PERSPECTIVE

Coronavirus disease in children normally results in a moderate infectious illness course, but substantial consequences can emerge when both acute infection and related phenomena such as the multisystem inflammatory syndrome in children are present (MIS-C). Adults have reported neurological symptoms ranging from mild headaches to seizures, peripheral neuropathy, stroke, demyelinating diseases, and encephalopathy. Neurological problems, like pulmonary and cardiac signs of COVID-19, emerge differently depending on age and underlying comorbidities. From literature reviews and principal data collected at NewYork-Presbyterian Morgan Stanley Children's Hospital, this information provides a concise overview of the neurodevelopmental disorders seen in the sense of COVID-19, as well as potential mechanisms and long-term implications of COVID-19 in the paediatric population.

The COVID-19 pandemic, which was caused by the SARS-CoV-2 virus, has left many unsolved problems for neurological patients and their caregivers. COVID-19 can cause severe symptoms in the elderly and immunocompromised people, and the virus can exacerbate symptoms of underlying neurological illness, especially in those with considerable bulbar and respiratory weakness or other neurologic dysfunction. Emerging SARS-CoV-2 vaccines provide significant protection against symptomatic infection, but both patients and providers may be concerned about the vaccine's theoretical risks, such as vaccine safety and efficacy in the context of immunotherapeutic, as well as the possibility of precipitating or exacerbating neurological symptoms. The current literature in this statement on behalf of the AAN Quality Committee, focusing on COVID-19 infection in adults with neurological disease, in order to highlight the risks and advantages of immunisation in these individuals. Neurologists should advise their patients to get vaccinated against COVID-19 based on the available data.

Patients receiving immunotherapies should be aware of the importance of vaccination scheduling in relation to treatment and the risk of a weakened immune response. An outbreak of disease caused by a novel coronavirus (2019-nCoV, later dubbed SARS-CoV-2) was reported in Wuhan, China, in December 2019. Coronavirus disease 2019 (COVID-19) spread rapidly over the world, quickly becoming a pandemic. Fever, dry cough, tiredness, and respiratory distress are common symptoms of COVID-19. In addition, SARS-CoV-2 infection can impair both the central and peripheral neurological systems. Viral neurotropism, a hyperinflammatory and hypercoagulative condition, or even mechanical ventilation-related dysfunction could be the source of these neurological alterations. Hypoxia, endothelial cell injury, and the effects of various ventilatory methods may all cause greater stress and strain, thereby increasing the inflammatory response and resulting in a complicated interaction between the lungs and the brain. To yet, no research has looked into the potential for mechanical ventilation to have a secondary effect on brain repair and outcomes. This information is intended to provide a current overview of the potential pathogenic mechanisms of COVID-19 neurological manifestations, discuss physiological issues related to brain-lung interactions, and propose strategies for improving respiratory support in critically ill patients with SARS-CoV-2 pneumonia.

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, there has been mounting evidence that SARS-CoV-2 infection is linked to a variety of neurological symptoms, including acute cerebrovascular events (i.e., stroke and cerebral venous thrombosis). These incidents can happen before, during, or even after COVID-19's basic symptoms appear. Although the mechanisms behind COVID-19-related cerebrovascular problems have yet to be fully understood, hypercoagulability, inflammation, and altered Angiotensin-Converting Enzyme 2 (ACE-2) signalling in conjunction with SARS-CoV-2 may all play a role. ACE-2 is essential for maintaining heart and brain equilibrium. Randomized 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, as the number of published COVID-19 cases with peripheral vascular events grows.

In most cases, severe acute respiratory infection in children results in mild respiratory symptoms or no symptoms at all. The multisystem inflammatory syndrome in children, which affects some paediatric patients and is associated with a high mortality rate, is a serious condition (MIS-C). There have been reports of neurological symptoms in both cases. The instances of juvenile patients who had serious neurological problems and a positive

Correspondence to: Edward Smith, Department of Neurology, University of Arkansas for Medical Sciences, Little Rock, Arkansas, USA; E-mail: edwardsmith1@gmail.com

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SARS-CoV-2 test at the same time. Between March 2020 and May 2021, a literature search was conducted. In terms of scale and magnitude, the current outbreak of SARS-CoV-2 infections that produce coronavirus-induced disease in 2019 (COVID-19) is the defining and unparalleled global health disaster of our time. Although SARS-CoV-2 is primarily focused on the respiratory tract, substantial evidence suggests that the virus may also infect the Central Nervous System (CNS) and Peripheral Nervous System (PNS), resulting in a variety of neurological problems, including significant complications such as seizures, encephalitis and loss of consciousness are two symptoms of encephalitis.

We give a complete analysis of SARS-currently CoV-2's known role, as well as a list of all neurological disorders documented in COVID-19 case reports from around the world. The virus could enter the CNS via a trans-synaptic route via olfactory neurons or through damaged endothelium in the brain microvasculature via the ACE2 receptor, which is potentiated by neuropilin-1 (NRP-1). The most dangerous symptom appears to be spontaneous respiratory cessation in some COVID-19 individuals. This could be a sign of a problem with the brainstem's cardiopulmonary regulatory centres. These ground breaking investigations provide a solid platform for future in-depth fundamental and clinical research that will be necessary to confirm the role of SARS-CoV-2 infection in the neurodegeneration of important brain regulatory areas.