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Severe Acute Respiratory Influence on Adult Human Neurogenesis

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Abstract

Infection with SARS coronavirus 2 is linked to the onset of neurological and psychiatric symptoms during and after the acute phase of illness. SARS-CoV-2-induced inflammation and hypoxia affect brain regions important for fine motor function, learning, memory, and emotional responses. The mechanisms underlying these central nervous system symptoms are mostly unknown. We investigated how SARS-CoV-2 affects neurogenesis while looking for the causes of neurological deficits. In this study, we compared a control group to a group of COVID-19 patients. Neurogenesis marker expression analysis revealed a decrease in the density of neuronal progenitor cells and newborn neurons in the SARS-CoV-2 group. Microglial activation was found to be higher in COVID-19 patients than in the control group.

Keywords: Adult human neurogenesis • SARS-CoV-2 • COVID-19 • Microglial

Introduction

People have been dealing with the COVID-19 pandemic, which is caused by the severe acute respiratory syndrome coronavirus 2. Subsequent mutations have resulted in COVID-19 infection waves with widely disparate symptoms, prognoses, and transmissibility levels. To date, reports have focused on coronavirus structural proteins, specifically the receptor binding domains of spike -proteins, rather than other nonstructural and accessory proteins. According to the World Health Organization, SARS-CoV-2, also known as the Delta variant, is a variant of concern due to increased transmissibility and disease severity. The Omicron variant, on the other hand, appeared to be more infectious than the Delta variant and more likely to result in vaccine breakthrough infections; it has also been reported to cause milder symptoms in most patients.

According to WHO statistics, over 655 million people have been infected with SARS-CoV-2, and approximately 6.6 million have died; however, the true figure is much higher. Because of the global spread of new SARS-CoV-2 variants, the coronavirus disease pandemic continues. COVID-19 symptoms include anosmia and ageusia, as well as fever, a dry cough, and shortness of breath. COVID-19 is a neurotropic virus associated with neurological manifestations in up to 36% of patients, with cerebrovascular events being the most commonly reported manifestation, followed by altered mental status.

Neurological symptoms can range from a mild headache or "brain fog" to more serious complications like Guillain-Barre syndrome, encephalitis, and arterial and venous strokes. The SARS-CoV-2 vaccine is still in development, and no specific drug is available at this time. Many antiviral drugs have been used to treat SARS-CoV-2 infection, but none have been effective. The rapid development of effective drugs for COVID-19 therapy is a difficult task because the traditional drug development process takes a long time and costs billions of dollars. A comparative genomics-based approach with previously identified human CoVs may result in a breakthrough in COVID-19 therapeutics [1].

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Description

The angiotensin-converting enzyme (ACE)-2 receptor, which has been found to be expressed by airway epithelia, lungs, choroid plexus, and various brain cells, including endothelial cells of the cerebral microvascular system, is how SARS-CoV-2 enters the human body. The primary targets of SARS-CoV-2 infection are ciliated cells of the nasal mucosa that express angiotensin-converting enzyme 2 and transmembrane serine protease 2. Infection with SARS-CoV-2 causes respiratory tract infection, which frequently dominates the clinical course. In the early stages of SARS-CoV-2 infection, the olfactory tract appears to be the primary entry route into the CNS. Furthermore, a significant number of patients, possibly as many as 33%, continue to experience neuropsychiatric symptoms after being discharged from the hospital, including a dysexecutive syndrome characterised by inattention, disorientation, and poor movement coordination.

Postmortem human neuropathological findings in COVID-19 include hypoxic damage, microglial activation, astrogliosis, leukocytic infiltration, and microhemorrhages, indicating that the CNS experiences neuropathological sequelae associated with hypoxia and neuroinflammation in at least some cases. Neuroimaging studies in postacute COVID-19 patients show disruption of fractional anisotropy and diffusivity, implying microstructural and functional changes in the hippocampus, a brain region critical for memory formation and part of a conserved subcortical network involved in anxiety and stress responses. The neurobiological bases of COVID-19 neuropsychiatric symptoms are largely unknown at this time. During COVID-19 infection, the blood-brain barrier may be disrupted and tight junctions may be damaged. The BBB is critical in protecting the brain's hemodynamic function [2].

The BBB's interconnected nature of brain capillary endothelial cells, pericytes, neurons, astrocytes, and microglia strongly suggests that this is a path of SARS-CoV-2 viral entry into the brain and a contributor to neuroinflammatory events. Isolated spike proteins can cross the BBB, according to evidence from in vitro models. While all of the regions studied, including the olfactory bulb, cortex, hippocampus, and medulla oblongata, showed some degree of BBB disruption, the hippocampus was the most affected. Recent research has found that people with COVID-19 have a significantly increased risk of receiving a new diagnosis of Alzheimer's disease within 360 days of receiving their initial COVID-19 diagnosis, particularly those over the age of 85. The hippocampus is one of two areas in the brain where new neurons are formed. Adult human neurogenesis has been identified in two

locations in the adult human brain: the dentate gyrus and the subventricular zone. As a result, we wanted to investigate how SARS-CoV-2 affects adult human neurogenesis. Neurogenesis is the process by which progenitor cells proliferate and differentiate, as well as newly formed neurons migrate and mature. Erickson described the first direct evidence for the presence of neurogenic processes in the adult human brain in 1998. He discovered new neurons growing in the dentate gyrus and subventricular zone [3,4].

The migration of newborn neurons from the SVZ to the olfactory bulb (OB). The fate of newborn neurons in the DG is strictly determined topographically, and they do not migrate toward neocortical areas of the brain. They only contribute to the constant replenishment of the dentate pool of new granule cells. Three types of transcriptionally active cells were found in the DG: glia-like neural stem cells, cells without processes, and neuroblasts. The SVZ contains three types of transcriptionally active cells: GFAP-positive neural stem cells, progenitor cells, and neuroblasts. DCX and NeuN, which label migrating neuroblasts and immature neurons, as well as GFAP, which labels astrocytic stem cells, are markers of early adult human neurogenesis phases [5].

Conclusion

The COVID-19 group showed neuropathological changes in the subarachnoid space and around blood vessels in the parenchyma, as well as microbleeds/petechial haemorrhages and hemosiderin deposits, indicating previous petechial haemorrhage. Other neuropathological changes have been described in the literature as well. Postmortem human neuropathological findings in COVID-19 include hypoxic damage, microglial activation, astrogliosis, leukocytic infiltration, and microhemorrhages, indicating that the CNS experiences neuropathological sequelae associated with hypoxia and neuroinflammation in at least some cases. The hippocampus and subventricular zone are adult brain neurogenic areas that contain neural stem cells. The hippocampus is a functional region of the limbic system of the brain that contributes to neuroregeneration, long-term potentiation, learning, memory formation, and emotion regulation. Every day, approximately 700 new neurons are formed in the hippocampus of a healthy adult. Hippocampal structure and function defects caused by ageing and neurological illnesses have been linked to emotional disorders and memory loss. Impaired neurogenesis has been identified as a possible cause of cognitive decline and progressive memory loss in ageing and neurodegenerative diseases such as Alzheimer's, Parkinson's, and Huntington's disease.

Acknowledgement

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Conflict of Interest

There are no conflicts of interest by author.

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