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Impact of Corona Virus on Psychiatric Disorders

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Abstract

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has been linked to 121 214 686 cases worldwide and 2 680 740 deaths as of March 18, 2021, according to World Health Organization and Johns Hopkins University reports. Since the virus is deeply neurotropic and neuroinvasive, it is generally accepted that this infection has a variety of effects on the nervous systems. To provide direction for the treatment of neurodevelopmental and neuropsychiatric conditions over the long term, it will be necessary to comprehend COVID-19's post-infectious manifestations.

Keywords: Acute respiratory syndrome; Neuroinvasive

Introduction

Anosmia, ageusia, central respiratory failure, stroke, acute inflammatory demyelinating polyneuropathy, toxic metabolic encephalopathy, headache, myalgia, myelitis, and ataxia are the most common clinical manifestations of COVID-19, according to published clinical papers and case reports. In the acute phase, COVID-19 may be a factor in neuropsychiatric manifestations like encephalopathy, psychosis, insomnia, and mood changes, as previously mentioned.

Both the viral infection itself and the host's subsequent neuroinflammatory response are responsible for the neuropsychiatric manifestations, which are attributed to: The activation of microglia An imbalance of central neurotransmitters like noradrenaline, epinephrine, and serotonin (which may be related to mental health issues); A breach in the blood–brain barrier (BBB) that causes immune cells from the peripheral blood to migrate into the central nervous system (CNS). The immunopathology and associated clinical symptoms of neuropsychiatric disorders and COVID-19 are the primary focus of this manuscript's review of fundamental critical aspects.

It is common knowledge that the SARS-CoV-2 virus-host receptor, specifically angiotensin-converting enzyme 2 (ACE-2), which is expressed in numerous human body tissues, including the CNS, is recognized and bound during viral infection. Glial cells and endothelial cells in the brain's blood vessels have been found to be infected in the central nervous system (CNS). It has been demonstrated that it can disrupt the BBB and increase permeability in brain blood vessels [1-4].

Discussion

CoV may enter the central nervous system (CNS) via synaptic routes of nerve cells, which appear to retrogradely infect the CNS via peripheral sensory nerves. On the other hand, the ACE-2 receptor that is found in olfactory epithelial cells appears to make it possible for direct CNS infection to spread through the olfactory nerve cells. Several pieces of evidence confirm that death following CoV infection can be caused by autophagy, apoptosis, pyroptosis, or elimination via innate immune cells. However, this mechanism does not have a complete consensus from various research groups. A non-specific immune mechanism involving activated macrophages, neutrophils, and natural killer cells and an adaptive immune mechanism with a relevant effector function mediated by dendritic cells and lymphocytes have been found to induce activation of both the innate and adaptive arms of the immune system toward an uncontrolled systemic response after SARS-CoV-2 infection. T helper cells (CD4), T cytotoxic cells (CD8), and B cells are all part of the adaptive mechanism. After that, these effector cells release more pro-inflammatory cytokines like interleukin (IL)-1b, IL-6, IL-10, IL-12, interferons (IFN)-alpha, IFN-gamma, tumor necrosis factor (TNF)-alpha, transforming growth factor beta, and chemokines (CCL This kicks off the so-called "cytokine storm," which is absolutely necessary for the multi-organ failure that results in the high lethality of the affected patient.

As a result of the cytokine storm and reactivation of immune cells, neuroinflammation caused by SARS-CoV-2 also becomes an additional effect of CoV infection. The strong immune response that humans experience as a result of being infected with SARS-CoV-2 has been linked to a number of different pathways in this context. During this infection, there was a significant decrease in the absolute count of T cells, monocytes, eosinophils, and basophils, with a primary impact on the absolute count of T cells, memory T helper, and regulatory cells [5-7].

There is also a significant increase in pro-inflammatory cytokines, such as IL-6, IL-2, IL-17, granulocyte-colony stimulating factor, and TNF, which co-occur in SARS-CoV-2 infection and affect the central nervous system. As they have been responsible for neurological manifestations such as encephalitis, CNS demyelination, and neuropsychiatric disorders, inflammatory conditions can also induce the increase of IL-1, IL-6, and TNF soluble mediators that may facilitate major BBB permeability.

Peripheral cytokines derived from the systemic host antiviral response may also transmigrate to induce neuropsychiatric symptoms through the neuro-inflammatory response and disruption of the BBB, even in the absence of SARS-CoV-2 CNS infiltration. Both local impairment of the neurotransmission system and the migration of effector immune cells into the central nervous system (CNS) could be to blame. Psychological conditions like post-traumatic stress disorder and depression have been linked to elevated levels of neurotransmitters like noradrenaline, epinephrine, and serotonin.

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The hypothesis that the cytokine storm is linked to the onset of psychiatric manifestations may be an indirect result of the hyperinflammation. Studies of respiratory virus pandemics in the past have shown that a variety of neuropsychiatric symptoms can occur during or after the acute infection, as evidenced by the presence of persistent cognitive deficits.

In the acute phase of COVID-19, it has been argued that, in addition to the psychosocial stressors, the infection that is underlying the pathophysiology of neuropsychiatric manifestations like encephalopathy, psychosis, insomnia, mood changes, post-traumatic stress disorder, panic attacks, and anxiety, which are mostly seen in health care workers and SARS-CoV infection survivors, is mostly due to the virus itself and is secondary to the immune response of the host. Direct viral infiltration of the central nervous system (CNS) can thus elicit an inflammatory response at the brain level that results in the activation of local microglia and the initiation of demyelinating processes, one of the primary causes of encephalopathy. Neuropsychiatric manifestations have been linked to a peripheral cytokine storm that causes an imbalance of neurotransmitters in the CNS in the absence of direct viral infiltration. Neurotransmission imbalances are brought on by the transmigration of immune cells from the peripheral immune system into the brain as a result of this cytokine storm's induction of a neuroinflammatory response that disrupts the BBB [8-10].

Conclusion

Due to general findings brought on by SARS-CoV-2 infection, it continues to be challenging for researchers to elucidate the true core mechanisms of COVID-19-associated neuropsychiatric complications. It may be challenging to differentiate this from encephalopathy caused by a systemic infection that does not affect brain tissue. Patients with COVID-19 have been shown to have a higher mortality rate due to an increase in IL-6, which is comparable to the rise in this cytokine seen in other neuropsychiatric disorders like depression and schizophrenia. Parallel to the neurological events, there is growing evidence for neuropsychiatric disorders that have been reported as secondary complications of SARS-CoV-2 infection. In a few years, this will become clear. In this context, COVID-19 has been described as causing an increase in mental health disorders like delirium, cognitive impairment, mood changes, and psychosis. 90% of COVID-19 cases result in delirium, and SARS-CoV-2 infection of the central nervous system is thought to directly contribute to cognitive disorders. With worsened scores on psychopathological measures in those with a history of psychiatric comorbidities, anxiety, depression, post-traumatic stress disorder, insomnia, and obsessive-compulsive symptomatology appear to be quite common in COVID-19 survivors and coworkers, primarily in females. Additionally, mental health impairment can result from hypoxemia, a common clinical sign of COVID-19. As a result, a worsening of attention, executive function, and verbal memory is a result of acute respiratory syndrome and relative hypoxia.

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