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The Repercussions of Corona Virus-Related Disorders in Women Having a Neurologic Impact

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Abstract

SARS-CoV and MERS-CoV's effects on the central nervous system & implications for SARS-CoV-2, multiple viruses have been proven to penetrate the CNS, despite the fact that few viruses are particularly neurotropic in nature. Neuroinvasive qualities and neurovirulent features are two ways viruses might be categorized. SARS-CoV-2 caused illness is most commonly associated with respiratory symptoms, there is growing evidence that the SARS-CoV-2 virus, like other known human coronaviruses (SARS-CoV and MERS-CoV), can cause neurological symptoms as a result of both neuroinvasive and neurovirulent mechanisms. However, case studies and series have provided the majority of evidence on the neurological effects of SARS-CoV-2 infection. Although the pathogenesis of COVID-19's neurological effects is unknown, numerous possibilities have been proposed, including direct viral infection of the nervous system, autoimmune sequalae, hypoxia-mediated injury and sequined-mediated harm. Evidence with other human coronaviruses, such as those implicated in the Severe Acute Respiratory Syndrome (SARS) and Middle East Respiratory Syndrome (MERS) pandemics, provides preliminary evidence for the possibility of SARS-CoV-2 having a neurological impact. SARS is a coronavirus that is prevalent in the CNS and is linked to a variety of neurological illnesses. It is caused by a coronavirus that has a high genetic similarity to SARS-CoV-2. It should be highlighted, however, that case studies with a small sample size are also used to study the neurological implications of the SARS virus.

Keywords: Neurovirulent mechanisms; Neurological implications; Neurodegenerative disorders; Severe acute respiratory syndrome

Abbreviations: SARS: Severe Acute Respiratory Syndrome; MERS: Middle East Respiratory Syndrome; SARDS: Severe Acute Respiratory Illness; PD: Parkinson's Disease; CSF: Cerebrospinal Fluid; ACE2: Angiotensin-Converting Enzyme 2

Introduction

According to intriguing findings, COVID-19 infection not only influences the clinical spectrum of manifested neurological disorders, but it also plays a critical role in the development of future diseases as long-term consequences. Intention is to investigate the sensitivity of neurological diseases to SARS-CoV-2 infection and the formation of COVID-19. Neuropathology, rather than age, may be the decisive factor in neuroimmunology disorders like multiple sclerosis. Age is more relevant in neurodegenerative disorders like Parkinson's disease [1]. Coronavirus 2 causes Severe Acute Respiratory Illness (SARDS) SARS-CoV2 is an invasive virus that infects the upper respiratory tract of humans and causes significant illness, including pneumonia and neurological problems [2].

According to multiple studies, SARS-CoV2 has been related to exacerbating the symptoms of Parkinson's Disease (PD), with the potential to increase fatality rates in those with advanced disease. SARS-CoV2 has been linked to the possibility of causing Parkinson's disease due to the virus's ability to penetrate the brain, where it can stimulate cellular processes involved in neurodegeneration [3]. The potential for SARS-CoV2 to worsen and cause specific neurological illnesses, such as Parkinson's disease, will be discussed in this study. We'll then look into its effects on the brain as well as its pathways and mechanisms of action. The current review stated that Nano based drug delivery systems are effective for Neuro Degenerative disorders in women, in severe acute respiratory illnesses caused by the coronavirus [4]. Nanotechnology is a new and promising branch of study that employs nanostructures and nanophases in a wide range of fields, including nanomedicine and nano-based drug delivery systems [5]. Nanomaterials are materials that are 1 to 100 nanometres in size. Nanoparticles are small nanospheres that are designed at the atomic or molecular level. Nanoscale particles have unique structural, chemical, mechanical, magnetic, electrical,

and biological capabilities, and nanotechnology helps to improve the pharmacological and therapeutic properties of traditional medications in a variety of ways [6].

Drug targeting, controlled drug release, therapeutic payload protection and enhanced bioavailability are all advantages. Researchers can now deliver drugs for longer periods of time with less frequent dose and greater precision and penetration in hard-to-reach areas by manipulating molecule size and surface features [7]. Overall, despite significant hurdles, a well-designed nano system can make a significant contribution to human health improvement. Several studies have found that Covid-19 patients with previous neurological illnesses have worsened neurological symptoms, which are regarded comorbidities linked to a higher risk of death in Covid-19 patients [8] Coronaviruses have long been recognised to be found in the central nervous system of people with Parkinson's disease, Alzheimer's disease and multiple sclerosis. SARS-COV-2 enters the brain through the olfactory nerve and the blood-brain barrier [9]. During the acute period of infection, the virus may induce neurological abnormalities that last beyond recovery. Earlier studies found SARS-CoV-1 particles in the Cerebrospinal Fluid (CSF) and in the brain, almost exclusively in the neurons, leading to the hypothesis that SARS-CoV-2 has the ability to penetrate and infect the brain [10]. Several investigations using transgenic mice found that intranasal injection of SARS-CoV resulted in virus penetration into the brain via the olfactory nerves, with fast dissemination to particular brain regions such as the thalamus and brain stem [11,12]. Furthermore, particles were discovered only in the brains of infected mice with modest concentrations of MERS-CoV virus infection in the brain was more important for the infected mice's high mortality than the infection in the lung, indicating that the infection in the brain was more important for the infected mice's high mortality [13]. SARS-CoV-2, like SARS-CoVs, enters target cells primarily through the ACE2 receptor. SARS-CoV-2 infects cells via interacting with the ACE2 receptor via its spike glycoprotein (S). Protein S is made up of two subunits: S1 and S2. S1 aids viral attachment to the ACE2 receptor, while S2 is essential for membrane fusion [14]. Protein S must be cleaved by transmembrane serine protease for this interaction to occur (TMPRSS2).

ACE2 receptors are found in the cardiorespiratory regions of the brain stem, cerebral cortex, posterior hypothalamic area, striatum and dopamine neurons of the substantia nigra [15]. The presence of the ACE2 receptor in the striatum and substantia nigra, as well as its expression with dopamine decarboxylase, an enzyme that converts L-dopa to dopamine, suggests that SARS-CoV-2 is involved in the pathogenesis of PD caused by viral infection [16,17]. When the virus was detected in the brain tissue of infected patients, it was also reported to have neuroinvasive properties, indicating that the virus was neurotropic. SARS CoV-2 was found in CSF samples from some Covid-19 patients with encephalitis or demyelinating disease, according to reverse transcription polymerase [18] chain reaction (RT-PCR).

These findings imply that Covid-19's severe acute respiratory syndrome is linked to SARS-entry CoV-2's into the brain, causing symptoms to aggravate and neurological abnormalities to develop. Currently, there are no cures for neurodegenerative diseases, and the treatments available only treat the symptoms or slow the progression of the disease [19]. Nanotechnology-based techniques have provided several strategies to cross the blood-brain barrier and increase drug moieties' bioavailability in the brain. Polymeric nanoparticles, lipidic nanoparticles, pegylated liposomes, microemulsions, and nanogels are examples of materials that have been studied in Parkinson's, Alzheimer's and Huntington's disease models [20]. Overall, the findings demonstrate that DDS has a lot of promise as a treatment for NDs.

An autopsy in multiple SARS patients revealed the presence of the virus in the brain and hypothalamus, as well as enema and neuronal degeneration, according to a case series. According to a case series, an autopsy in many SARS patients revealed the presence of the virus in the brain and hypothalamus, as well as edoema and neuronal degeneration [21]. The SARS virus was also found in the cerebrospinal fluid of a patient who had convulsions after contracting SARS. SARS virus was also detected in the cerebrospinal fluid of a patient who developed seizures in the context of SARS infection [22]. Adding to the evidence that coronaviruses may have an effect on the central nervous system. MERS is a coronavirus that has been shown to affect CNS function and illness. In individuals with MERS, a case series revealed neurological implications, including altered awareness, ataxia, and focal motor impairments [23].

SARS-CoV-2 is genetically related to SARS-CoV and MERS-CoV, as well as other coronaviruses. This group of human coronaviruses belongs to the β corona viruses, which are linked to serious human disease [24] SARS-CoV-2 and SARS-CoV have a genetic resemblance of 79.5 percent while MERS-CoV has a genetic similarity of 50 percent provide a detailed analysis of the viral structure and infection processes of SARS-CoV, SARS-CoV-2, and MERS-CoV. MERS-CoV attaches to dipeptidyl-pepitidase 4 to enter human cells, whereas SARS-CoV and SARS-CoV-2 bind to Angiotensin-Converting Enzyme 2 (ACE2) as a cell entrance receptor [25]. According to new research, the CoV spike glycoprotein that binds SARS to the cell membrane is longer in the SARS-CoV-2 virus, implying that the SARS-CoV-2 virus binds the ACE2 receptor with a higher affinity. This idea has been put forward to explain SARS-increased CoV-2's infectiousness and is proposed to help the virus have a greater neuroinvasive capability than previous CoV viruses [26]. This is seriously considering the broad expression of ACE2 in the brain, implying that SARS-CoV-2 could infect neurons and glial cells throughout the CNS. In comparison to SARS-CoV, neurological symptoms tend to manifest earlier in the course of illness in SARS-CoV-2 albeit the processes behind these reported differences will require more investigation [27,28].

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Acute cerebrovascular damage and decreased consciousness were among the neurologic signs of severe illness. In an attempt to characterise and better understand the neurological syndromes that presented after patients developed COVID-19, the University College London Queen Square National Hospital for Neurology and Neurosurgery COVID-19 Study Group identified five categories of neurological disorders: encephalopathies (n14 10), inflammatory CNS syndromes, including encephalitis and acute disseminated encephalomyelitis (ADEM; n14 12), ischemic strokes (n14 8), peripheral [29]. According to research of hospitalised COVID-19 patients conducted in France, eight patients with encephalopathy of unclear cause displayed leptomeningeal augmentation, whereas patients with perfusion scans demonstrated bilateral frontotemporal hypoperfusion [30].

Conclusions

In neurological disorders, the most important clinical lesson we've learnt from COVID-19 is that it's a completely unpredictable sickness. We discovered that Parkinson's disease neuropathology may protect against SARS-CoV-2 infection, whereas risk factors include age and disease development. Patients with multiple sclerosis may be more susceptible to severe COVID-19 results than those with neurodegenerative illnesses. In the neurodegenerative population, however, further neurodegeneration as a long-term consequence of COVID-19 infections is possible. A large cohort of neurological patients with severe acute respiratory syndrome is needed to corroborate our early findings.

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