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Perspective

# Effect of COVID-19 Infections and its Repercussions on the Neurological Abnormalities

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## DESCRIPTION

The effects of COVID-19 and the scientific data supporting its involvement in the kidneys, intestines, lungs, and heart are extended to the brain, highlighting neurological problems and the disease's high rates of morbidity and mortality. In China, hospitalized COVID-19 patients were investigated at the start of the pandemic; 35% of these individuals had neurological symptoms. Understanding the SARS-CoV-2 pathophysiology with relation to contamination and damage to the nervous system is therefore essential for clinical and therapeutic methods that may lessen the disease's impact. In this regard, it's critical to emphasize that, in addition to the disease's direct impact on neurons; the inflammatory state caused by a dysregulation of the immune system due to infection is a major contributor to the disease's neurological symptoms.

SARS-CoV-2 can infect Nervous System (NS) through the bloodstream like other infections can. Although there is a barrier between blood flow and neural tissue, this defense is compromised by the viral infection's enhanced inflammatory response. The activation of metalloproteinase, which are enzymes capable of breaking down the proteins that make up the blood-brain barrier and thereby increasing its permeability, plays a crucial role in the recruitment of white blood cells, such as neutrophils, associated with the known cytokine storm, especially with the release of IL-6, interleukins, and other factors. Post-mortem microscopic observations in individuals with neurological symptoms and positive SARS-CoV-2 RT-PCR have already shown that the virus migrates from endothelium to neuronal cells concurrently with the presence of viral particles within neural cytoplasmic vacuoles. The cerebral microcirculation is a facilitating factor in the coupling of the glycoprotein S (spike) to the functional ACE-2 receptor, allowing penetration into neurons and glial cells and resulting in neurological infection. Systemic circulation plays a fundamental role in viral dissemination in the neurological context. ACE-2 receptors are more prevalent in the alveolar epithelium of the lungs, but it has been determined that SARS-CoV-2 can also infect brain tissue through these same receptors, expanding the COVID-19 symptom spectrum to include neurological signs.

Viral infection through the nose can also enter the brain *via* the cribriform plate of the ethmoid bone. This method takes use of the brain's abundant ACE-2 receptors, which enable the virus to spread after entering through the olfactory nerve. Additionally, hyposmia, an early symptom reported in the majority of infected individuals, is explained by the infection pathway through this nerve. Due to the olfactory changes in COVID-19, the olfactory bulb, a portion of the central nervous system not covered by the dura mater, is unquestionably a contemplated route.

It's important to keep in mind that, in addition to the processes that cause direct brain tissue destruction, infected patients are increasingly reporting cerebrovascular episodes that are linked to the disease's hypercoagulability. Being a fearsome and well-known complication of conditions like sepsis, which can also be developed with the progression of the disease, the prothrombotic state appears to be directly tied to the systemic inflammation brought on by the infection. The hypercoagulable state observed in COVID-19 may be caused by activation of the coagulation cascade, dysregulation of natural anticoagulant systems, including the protein C system and fibrin disintegration, as well as the presence of pro-inflammatory cytokines and the endothelium lesion. All of these imbalances cause thrombin to be produced and endogenous fibrinolysis to be reduced, which causes clots to form and remain in the microvasculature. As the condition progresses, these clots may grow in size and become more significant. Since the production of inflammatory mediators triggers the process of coagulation and this state encourages the maintenance of the inflammatory process, the development of Disseminated Intravascular Coagulation (DIC) may be caused by the feedback that exists between the processes of coagulation and inflammation. The moment this vicious cycle starts, it intensifies.

## CONCLUSION

Because of this, it is feasible to see that many COVID-19 patients have elevated D-dimer levels that increase with disease severity as well as delayed prothrombin and activated partial thromboplastin times. Most critically ill individuals with the Severe Acute Respiratory Syndrome (SARS) appear to be more

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susceptible to cerebrovascular accidents and cerebral venous thrombosis when these parameters change. This proves that patients with severe infections or numerous comorbidities are more likely to have hypercoagulation and the high inflammatory response brought on by the viral presence in the host organism.

We emphasize that the amount of drugs administered in intensive care units can also interfere with neurological conditions and any comorbidities the patient may have, even though it is still unclear whether the impairment of the nervous system is a result of direct infection by SARS-CoV-2 or of a diffuse inflammatory process affecting various organs and systems and generating multiple manifestations, including those of the nervous system. In order to enhance the patients' prognosis, it is important to evaluate hospitalized patients with neurological abnormalities brought on by COVID-19, change protocols, and teach healthcare staff.