

COVID-19 and Neurologic System

Firooz Salehpour¹ and Ata Mahdkhah^{2*}

¹Department of Neurosurgery, Tabriz University of Medical Sciences, Tabriz, Iran

²Department of Neurosurgery, Urmia University of Medical Sciences, Urmia, Iran

***Corresponding Author:** Ata Mahdkhah, Department of Neurosurgery, Urmia University of Medical Sciences, Urmia, Iran.

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Like the severe acute respiratory syndrome coronavirus (SARS-CoV) and Middle East respiratory syndrome coronavirus (MERS-CoV), another extremely harmful coronavirus named SARS-CoV-2 (previously known as 2019-nCoV) spreaded in China and rapidly expanded around the world. It results in acute, seriously lethal pneumonia coronavirus disease 2019 (COVID-19) with clinical manifestations similar to those mentioned for SARS-CoV and MERS-CoV.

Study of the genome sequences of COVID-19, SARS-CoV, and Middle East Respiratory Syndrome coronavirus (MERS-CoV) showed that COVID-19 has a better sequence identity with SARS-CoV compared to MERS CoV. But, the amino acid sequence of COVID-19 is not similar to other coronaviruses particularly in the regions of 1ab polyprotein and surface glycoprotein or S-protein. While some animals have been considered to be a reservoir for COVID-19, no animal reservoir has been previously defined. COVID-19 causes COVID-19 disease that has parallel manifestations as SARS-CoV.

Studies propose that the human receptor for COVID-19 may be angiotensin-converting enzyme 2 (ACE2) receptor like SARS-CoV. The nucleocapsid (N) protein of COVID-19 has nearly 90% amino acid sequence identity with SARS-CoV. The N protein antibodies of SARS-CoV may cross react with COVID-19 but may not provide cross-immunity. In a related to SARS-CoV, the N protein of COVID-19 may take part in a key function in suppressing the RNA interference (RNAi) to affect the host resistance.

The most distinguishing manifestation of patients with COVID-19 is respiratory distress. Moreover, some patients with COVID-19 presented with neurologic manifestations, such as headache, nausea, vomiting, anosmia and even loss of consciousness. Increasing evidence shows that coronaviruses are not always limited in the respiratory system and that they may reach the central nervous system. The infection of SARS-CoV has been reported in the brains from both patients and experimental animals, where the brainstem was invaded. Additionally, some coronaviruses have been confirmed able to spread via a synapse-connected route to the medullary cardiorespiratory center from the mechanoreceptors and chemoreceptors in the lung and lower respiratory airways.

Based on the relationship between SARS-CoV and SARS-CoV2, it remains to make clear whether the expansion of SARS-CoV2 is partially responsible for the acute respiratory failure of patients with COVID-19. Future studies will be an implication for the prevention, diagnosis and treatment of the SARS-CoV-2-induced respiratory disease [1-4].

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