

Covid-19 Associated Impact on the Blood-Brain Barrier Integrity and Function

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Abstract

Covid-19 infection results from severe respiratory illness associated with increasing neurological manifestations. Some studies have recently highlighted the COVID-associated brain insult which could preferably involve the loss of integrity and function of the blood-brain barrier. Here, we present a short review in analysing the concrete involvement of the BBB breakage/leakage due to the recent pandemic surge.

Recent Developments

With an upsurge in Covid-associated illness there has been a moderate increase in neurological manifestations including ischaemic and hemorrhagic stroke, autoimmune disorders such as AIDP etc. Understandably, the blood-brain barrier (BBB) is a critical interface between the blood and brain parenchyma. It is the gateway of protection that could withhold COVID-induced insult if any. Recent preliminary studies have demonstrated the SARS-Cov-2 interaction with the BBB [1]. The pathogenesis associated could be a time-oriented breakdown of the BBB via influx of cytokine storm or hypoxia leading a recognised insult [2]. Moreover, the BBB leakage would further add-on to an increase virus entry and further developing neurological sequelae. Furthermore, CNS-associated Virus influx via disruption of the BBB or engulfing the peripheral nerves would certainly be a steppingstone towards initiation of the neuroinflammation [3]. Due to uncovered information in how the virus would implicate in the CNS makes the clinicians difficult to initiate a timely treatment. There could be various pathways including the above-mentioned BBB insult, SARS-CoV-2 could initiate DNA fragmentation which could further lead to cellular apoptosis, hence contributing to BBB leakage. Moreover, with presence of this foreigner could trigger reactive oxygen species leading to directly promote matrix-metalloproteinase (MMP) protein expression which in turn would lead to basement membrane degradation. This may further trigger Super-oxide anion levels in conjunction with accelerating NADPH levels. In relation to the mentioned factors associated with covid-BBB insult, inter-junction proteins such as Claudin-5 would most certainly reduce their capabilities to maintain the integrity of the BBB. These mentioned pathways could develop a new conduit in novel treatment measures to avoid development of a neurological pathogenesis.

Conclusion

With time, we do require further studies involving both clinical and molecular aspects of neurological insults provoked by Covid-19 infection. The BBB integrity and function could be assessed using human in-vitro triple culture (human brain microvascular endothelial cells, astrocytes and pericytes) models when exposed to mimicking the insult via virus inflation. We could further add-on with the timely release of various inflammatory cytokines.

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