Int Clin Neurosci J. 2020 Autumn;7(4):162-163 Letter to Editor

doi:10.34172/icnj.2020.19

Novel Coronavirus Characteristic Cerebrovasculopathic Effects

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Published online 28 September, 2020

Dear Editor,

Coronavirus beta 2 started infecting people from china in fall 2019. It did not take a long time to spread worldwide and take many victims and cause many deaths.

As it is known, beta coronaviruses (SARS-Cov-2), which cause the severe acute respiratory syndrome, COVID-19 pandemic, have developed neurological manifestations in addition to primary respiratory symptoms in a significant number of patients.[1,2] The occurrence of anosmia and hyposmia is evidence of this claim.

Stroke incidence of 0.9%-2% in especially young COVID-19 patients, is reported.[2-5] The worst issue about this incidence is the high mortality rate despite even optimal treatment.[6,7] Since the number of patients is as much as the World Health Organization (WHO) could declare the disease pandemic, it is important to review the pathophysiology in order to estimate the risk and guide the decision making. This letter aims to review the mechanism of virus pathophysiology and the following cerebrovascular presentations reported until now.

SARS- CoV-2 virus blocks the angiotensinogen-converting enzyme 2 (ACE2), which plays a critical role in the autoregulation of cerebral perfusion. It has also reported that it induces a hypercoagulable state (same as other flu viruses but with more mortality) and a hyperinflammatory response with cytokine storm characterization.[8,9] Inhibiting ACE2, neuroinvasion, and neurovirulation, and hypercoagulopathy induction are the pathophysiologic characteristics of the COVID-19.[10-16]

In some patients, the cerebrovascular attack was noticed as the early manifestation with no severe presentations. The vascular occlusion had seen in both arterial and venous sides; most of them occurred in large vessels. There are unusual manifestations reported in recent studies that there are young patients without previous history or risk factors, represented acute ischemic stroke, sinus thrombosis, tandem occlusion (internal carotid artery and middle cerebral artery M1 occlusion), carotid T occlusions, M1 and A2 occlusion, central retinal artery occlusion.[17]

The mean age of the patients was lower than historical non-COVID stroke patients. There was a noticeable number of patients who presented the cerebrovascular stroke as the early manifestation of COVID-19. There is a report of a 9-day lag period between the COVID-19 symptoms and the cerebrovascular event occurrence.[16] There are a few interpretations for the mechanism of COVID-19 vasculopathy incidence: epitheliopathy, i.e., a direct vasculopathic effect, immune-mediated platelet activation, potentiation of prothrombic surrounding, cardiac arrhythmias caused by infection, and also dehydration.[12-15,18]

It is vital in pandemic break-outs to raise awareness to improve decision-making, risk estimation, and health care workers' protection. We should watch patients and novel manifestations precisely and report the new findings orderly for this issue.

Conflict of Interest

The authors declare that they have no conflict of interests.

Ethical Statement

Not applicable.

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Citation: Zandpazandi S, Shahmohammadi MR. Novel Coronavirus Characteristic Cerebrovasculopathic Effects. Clin Neurosci J. 2020;7(4):162-163. doi:10.34172/icnj.2020.19.