Novel Therapeutic Strategies for the Management of Neuropathology and Neuroprotection in COVID-19

The COVID-19 pandemic has caused a strong impact across all sectors of society. The virus is known as severe acute respiratory CoV-2 (SARS-CoV-2) and involves neurotropism and several central and peripheral nervous system structures that express the receptor Angiotensin-converting enzyme 2 (ACE2) employed by SARS-CoV-2. It has been observed that this disease not only affects the lungs of patients but also other tissues that also trigger cell damage processes. Several studies have shown that neurological symptoms are present in infected patients with a difference in intensity and chronicity. It has also been shown that ACE2 is expressed in different regions of the central nervous system, in addition to the fact that SARS-CoV-2 can cross the blood-brain barrier, so it can infect these tissues, partially explaining the short-term neurological symptoms ranging from headache, epilepsy and even cerebral vascular events. Particularly, two conditions, the long-term gliosis secondary to inflammation and the release of free radicals, plus hypoxia due to the pathophysiology of COVID-19, where the first organ affected by oxygen deprivation is the brain, prompt the necessity of employing neuroprotective strategies to minimize chronic damage. Unfortunately, neuroprotection is not contemplated in current treatment schemes for COVID-19; for this reason, this issue is intended to demonstrate that there are feasible options for neuroprotection in these patients. Hernández-Ochoa et al. propose as a target for neuroprotection the administration of G6PD since deficiency of this molecule has been seen in patients with COVID-19, which has been related to greater damage in different tissues, including the brain [1]. Sixto-López and Correa-Basurto propose the employment of HDAC inhibitors (HDACi) due to their neuroprotective effect through down-regulation of the proinflammatory cytokines and the inhibition of viral entry to neuronal tissues as well as decreasing the virus replication [2]. Cortes-Altamirano et al. consider the treatment with cannabis as a feasible option for neuroprotection in patients with COVID-19 because the cannabinoid system has been related to a decrease in viral entry, viral replication, and hyperinflammation in the brain tissues [3]. Quintero-Fabián et al. have established the mechanisms by which vitamin D acts as an important neuroprotector, which should be taken into consideration for determining its accessibility, safety and its immunoregulatory functions. It has been shown that this vitamin crosses the blood-brain barrier efficiently, so they proposed it as a neuroprotective agent that could be administered to patients with COVID-19, not only during the disease but for a long period of time [4]. In conclusion, this issue establishes, first, the need for neuroprotection in all patients with COVID-19 because the nervous tissue can be directly affected by the interaction of the virus with its receptor and by the hypoxia that may occur; second, it considers alterations that in the long term can be related to neurodegeneration due to neuroinflammation and release of free radicals. Lastly, feasible neuroprotection alternatives are proposed. We are encouraging the scientific community to propose observational and experimental studies to culminate in adding neuroprotection as a fundamental element in the therapeutic management of all patients with COVID-19.

REFERENCES

Cindy Bandala
(Guest Editor)
Instituto Nacional de Rehabilitación LGII,
Escuela Superior de Medicina, Instituto Politécnico Nacional
Mexico City, Mexico
Tel: +52 5513383955
E-mail: dra.cindy.bandala@gmail.com

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