



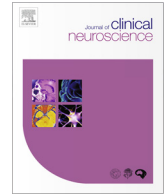
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## Journal of Clinical Neuroscience

journal homepage: [www.elsevier.com/locate/jocn](http://www.elsevier.com/locate/jocn)

## Correspondence

**COVID-19 and molecular mimicry: The Columbus' egg?***Dear Editor,*

It is now evident that COVID-19 (induced by the severe acute respiratory syndrome coronavirus 2, commonly abbreviated SARS-CoV-2) is not only a "respiratory" disease, at least in a subset of patients. Thrombosis and disseminated intravascular coagulation (DIC) prompted many clinicians, including neurologists, to add heparin to the therapeutic arsenal used in the fight against the virus. However, this drug could resolve the epiphenomenon but not the disease.

Reflecting on what induces thrombosis, DIC and other clinical signs attributable to autoimmunity, I was strongly surprised by the fact that no published paper so far suggested molecular mimicry against endothelial cells as the cause of the multi-organ failure that irreversibly aggravates the conditions of these patients [1].

Recently, this Journal published a report about Guillain-Barré syndrome associated with COVID-19 infection [2]. It is well known that molecular mimicry has a role in the pathogenesis of Guillain-Barré [3]. In my opinion, it would be appropriate if this Journal would stimulate the scientific community on the fact that molecular mimicry phenomena can occur in SARS-CoV-2, as well as it was described in another SARS [4]. In fact, it could explain both the acute pulmonary embolism and the multi-organ microvascular thrombosis that some patients experience [5]. Therefore, it is also urgent to start the search for human epitopes that turn into autoantigens, and to remind this risk to all those who are currently working on vaccines.

**Declaration of Competing Interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

**Appendix A. Supplementary data**

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.jocn.2020.05.015>.

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Received 27 April 2020

Accepted 4 May 2020