Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Parkinsonism and Related Disorders 77 (2020) 20

Contents lists available at ScienceDirect

Parkinsonism and Related Disorders

journal homepage: www.elsevier.com/locate/parkreldis

Correspondence

May Parkinson's disease be a protective factor against CNS involvement by SARS-CoV2?

ARTICLE INFO

Keywords:
covid19
Coronavirus
Parkinson

We read with great interest the manuscript by Antonini and collaborators on the relationship of Parkinson's disease (PD) and Covid infection19. However, regarding the not yet well-defined relationship between these two diseases, we have developed the hypothesis that PD can hinder the virus's access to the central nervous system.

The pathophysiology of PD is linked to ascendant α-synucleins (α-syn) spreading in about 50% of cases. According dual-hit hypothesis the α-syn spreading starts in the olfactory bulb and enteric plexuses, migrating in a retrograde way through the vagus nerve to the bulb and, from there to the midbrain and other CNS structures [1].

SARS-CoV2 can reach the CNS via two main routes: through the bloodstream and by retrograde neuronal dissemination (Wu et al., 2020). After the systemic infection, the parasympathetic pulmonary terminations of the vagus nerve can be affected with further retrograde dissemination to the bulb, affecting ambiguous and solitary tract nuclei. In addition, there may also be an invasion of the olfactory bulb by the presence of the virus in contact with the olfactory mucosa [2].

The α-syn spreading in PD is followed by neuronal death of the affected structures, which is visible in studies of myocardial scintigraphy and volumetric MRI [3]. Thus, it can be assumed that the routes for SARS-CoV2 entry in CNS are reduced in patients with PD who have involvement of peripheral structures prior to motor symptoms (gut-first parkinsonism).

For events that do not depend on virus direct CNS invasion such as stroke (coagulation factors disturbance, D-dimer increasing, platelets redutcion) or decrease in level of consciousness (hypoxia, metabolic changes, sepsis, cytokine storm) [4], the natural course of the disease is expected. Still, patients with PD have a higher theoretical risk of evolving to a severe condition, and possibly death, after being infected by SARS-CoV2 due to advanced age, previous limitations and the presence of a greater number of comorbidities [5].

However, considering the above, it is postulated that, regardless of the severity of the systemic condition and possible higher mortality, in these patients there will be no virus spreading to the CNS due to the previous destruction of structures that would serve as their gateway.

Financial disclosures

The authors declare that there is no conflict of interest regarding the publication of this article. No author has received funding from any institution.

Funding sources for study

No funding

References

[1] H. Braak, U. Rüb, W.P. Gai, K. Del Tredici, Idiopathic Parkinson's disease: possible routes by which vulnerable neuronal types may be subject to neuroinvasion by an unknown pathogen, J Neural Transm (Vienna) 110 (5) (2003) 517–536.
[2] M. Desforges, A. Le Coupanec, P. Dubeau, et al., Human coronaviruses and other respiratory viruses: underestimated opportunistic pathogens of the central nervous system? Viruses 12 (1) (2019) 14.
[3] P. Borghammer, N. Van Den Berge, Brain-first versus gut-first Parkinson's disease: a hypothesis, J. Parkinsons Dis. 9 (s2) (2019) S281–S295.
[4] Y. Wu, X. Xu, Z. Chen, J. Duan, K. Hashimoto, L. Yang, C. Liu, C. Yang, Nervous system involvement after infection with COVID-19 and other coronaviruses, Brain Behav. Immun. (2020), https://doi.org/10.1016/j.bbi.2020.03.031 pii: S0889-1591(20)30357-3.
[5] A. Antonini, V. Leta, J. Teo, K.R. Chaudhuri, Outcome of Parkinson's Disease Patients Affected by COVID-19, Mov Disord, 2020 [published online ahead of print, 2020 Apr 29].

F. Spina Tensini*
Complexo Hospital de Clínicas da UFPR, Departamento de Clínica Médica, Unidade de Neurologia e Psiquiatria, Setor de Distúrbios do Movimento, Curitiba, PR, Brazil
E-mail addresses: ftensini@gmail.com, fernando.tensini@hc.ufpr.br.

T. Spina Tensini
Faculdade Pequeno Príncipe, Curitiba, PR, Brazil

G.L. Franklin, H.A.G. Teive
Complexo Hospital de Clínicas da UFPR, Departamento de Clínica Médica, Unidade de Neurologia e Psiquiatria, Setor de Distúrbios do Movimento, Curitiba, PR, Brazil

* Corresponding author. Hospital de Clínicas da Universidade Federal do Paraná, Rua General Carneiro, 181, Curitiba, PR, CEP 80060-900, Brazil.

https://doi.org/10.1016/j.parkreldis.2020.06.012
Received 1 June 2020; Received in revised form 8 June 2020; Accepted 14 June 2020
Available online 18 June 2020
1353-8020/