Sarcouncil Journal of Medical Series

ISSN(Online): 2945-3550

Volume- 03| Issue- 11| 2024

Letter to the Editor **Received:** 11-10-2024 | **Accepted:** 29-10-2024 | **Published:** 18-11-2024

Fronto-Orbital Cortex and Olfactory Bulb Atrophy are Unlikely Common Features of COVID-19 with Hyposmia

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Keywords: SARS-CoV-2 infection, cortical atrophy, hyposmia, complication, olfactory bulb.

LETTER TO THE EDITOR

We read with interest the article by Gezegen, *et al*., on a study of 36 patients with SARS-CoV-2 infection (SC2I) and hyposmia (SC2I-HO), 21 COVID-19 patients without hyposmia (SC2I-nonHO), and 25 healthy controls (HC) using a neurological examination, neuropsychiatric tests and sniffer stick tests *[Gezegen, H. et al., 2024]*. SC2I-HO patients had lower revised total and speech sub-scores, and performed worse on the sniffer stick tests, identification score, and composite score compared to HC *[Gezegen, H. et al., 2024]*. Left and right olfactory bulb (OB) volumes were reduced compared to HC. Furthermore, orbitofrontal cortex thickness was reduced in SC2I-HO patients compared to HC *[Gezegen, H. et al., 2024]*. The study is compelling but some points require discussion.

The first point is that no explanation has been provided as to why cortical atrophy, as described in the 36 patients, has not been reported in millions of patients infected with SC2I and suffering from hyposmia. How to explain this discrepancy? There is also no evidence in the literature of a connection between hyposmia and fronto-basal cortical atrophy.

The second point is that the pathophysiological mechanism underlying fronto-orbital cortex atrophy has not been explained. Was it due to invasion of the cortical neurons by SC2? Was there an inflammatory reaction that resulted secondarily in cortical atrophy? Was there evidence of hypoxia or virus particle in cortical neurons?

The third point is that the group sizes were small, making comparison between the three groups unreliable. The group sizes could be increased by organising a multicentre study.

The fourth point is that the clinical consequences of orbital frontal cortex atrophy have not been described. Did these patients develop cognitive impairment with memory deficit or were there indications for a drive disorders?

The fifth point is that the latency between the onset of SC2I and the time the MRI was performed was not included in the analysis. Considering that cortical atrophy is indeed a feature of hyposmia in SARS-CoV-2 infected patients, it is conceivable that cortical atrophy does not develop immediately after the onset of infection but takes some time to develop. Therefore, cortical atrophy may only be detectable until a certain period of time has elapsed between onset of anosmia and MRI.

The sixth point is that the results of comprehensive cerebral imaging were not compared between the three groups *[Gezegen, H. et al., 2024]*. We should know how many of the included SC2I-HO patients, SC2I-nonHO and HC had ischemic stroke, intracerebral bleeding, venous sinus thrombosis, encephalitis, meningitis acute disseminated encephalomyelitis, posterior reversible encephalopathy syndrome, or epilepsy, which are common CNS complications of SC2I and may be associated with cortical atrophy *[Nagubadi, R. et al., 2024]*.

In summary, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these weaknesses would strengthen the conclusions and could improve the study. Whether cortical atrophy is indeed a feature of COVID-19 patients with anosmia, remains to be confirmed in a multicentre study.

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Source of support: Nil;
Conflict of interest: Nil.

Cite this article as:
Finsterer, J. "Fronto-Orbital Cortex and Olfactory Bulb Atrophy are Unlikely Common Features of COVID-19 with Hyposmia." *Sarcouncil Journal of Medical Series* 3.11 (2024): pp 7-8.

Publisher: SARC Publisher