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Before Permanent Hemifacial Pain can be attributed to SARS-CoV-2, Alternative Etiologies must be Ruled Out

***Walter Strobl*[1] *and Josef Finsterer*[2]**

[1]*MD, Dpt. of Health Sciences, Medicine and Research, Danube University Krems, and MOTIO, Vienna, Austria*

[2]*MD, PhD, Neurology Dpt., Neurology & Neurophysiology Center, Vienna, Austria, Orcid: 0000-0003-2839-7305*

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LETTER TO THE EDITOR

We read with interest the article by O'Neill, *et al*., about a 58 year-old female who was diagnosed with trigeminal neuralgia (TN) following a SARS-CoV-2 infection (SC2I) 18 months previously [O'Neill, F. *et al*., 2023]. The patient did not benefit from duloxetine, but only from three intradermal applications of Botulinum toxin at three-month intervals between injections [O'Neill, F. *et al*., 2023]. The case is impressive, but some points require discussion.

We disagree that the patient had acute SC2I 18 months previously at symptom onset. Presumptive acute SC2I was diagnosed exclusively by determining antibodies against the spike-protein [O'Neill, F. *et al*., 2023]. SC2I was not reported to be diagnosed by positive PCR, which is the gold standard for the diagnosis of SC2I. Spike-protein antibody titers decrease over time and their measurement at a specific time point does not allow determination of the onset of SC2I [Pérez-Juárez, H. *et al*., 2023]. The index patient may have had a SC2I months before onset of facial pain. We therefore disagree with the assumption that there was a causal relationship between TN and the previous SC2I [O'Neill, F. *et al*., 2023]. The strongest argument against a causal relationship is that acute SC2I was diagnosed solely by measuring titers of antibodies directed against the spike-protein.

We also do not believe that the patient had TN. TN is characterized by touch-related, unilateral, brief, shock-like paroxysmal pain in one or more divisions of the trigeminal nerve, lasting from a few seconds to a few minutes [Maarbjerg, S. *et al*., 2017]. In addition to the paroxysmal pain, some patients also experience persistent pain [Maarbjerg, S. *et al*., 2017]. Arguments against TN in the index patient are that the patient did not have paroxysmal pain necessary for the diagnosis of TN and that the facial pain was not caused by touch [Maarbjerg, S. *et al*., 2017]. Persistent pain alone, as in the index patient, without paroxysmal pain does not allow a diagnosis of TN.

Furthermore, we disagree with the statement that SC2I is only associated with neuropathy of cranial nerves II, III, IV, and VII, as mentioned in the introduction [O'Neill, F. *et al*., 2023]. All cranial nerves can be affected during SC2I either as an isolated cranial nerve neuropathy or as polyneuritis cranialis in variable combination with other cranial nerves [Finsterer, J. *et al*., 2022].

A limitation of the study is that various alternative causes of symptomatic TN were not sufficiently ruled out. Although the patient had undergone cerebral MRI with contrast medium [O'Neill, F. *et al*., 2023], this does not rule out meningeal carcinosis, glaucoma, small neurinomas, Guillain-Barre syndrome (GBS) with cranial nerve involvement, or compression of the nerve root entry zone by a pulsating arteria cerebelli superior or inferior. Because a causal relationship between facial pain and SCI has not been proven, alternative pathophysiologic explanations should be considered and discussed. Missing In this regard, cerebrospinal fluid (CSF) studies to determine whether carcinosis, meningitis, or GBS are lacking. It is also recommended that the patient undergoes a CIS-3D MRI study to determine whether there was anatomical proximity between the root entry zone of the trigeminal nerve and the superior or inferior cerebellar arteries. It is also important to rule out giant cell arteritis by determining the blood sedimentation rate, an ophthalmologic examination, and finally a biopsy of the temporal artery.

The patient also had abducens palsy homolateral to the TN, but there is no discussion as to whether the causes of abducens palsy and TN were the same or different. There is also no description of the clinical course of abducens palsy. Since polyneuritis cranialis is a subtype of GBS, it would

***Corresponding Author:** Josef Finsterer

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have been imperative to rule out GBS through appropriate studies.

Since corneal confocal microscopy revealed reduced corneal nerve fiber length, it is also conceivable that pain and numbness in the right facial area were simply a manifestation of small fiber neuropathy.

In conclusion, the excellent study has limitations that should be addressed before drawing final conclusions. Clarifying the weaknesses would strengthen the conclusions and could improve the study. Before 18 months of persistent hemifacial pain can be attributed to SC2I, alternative etiologies must be ruled out.

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