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Letter to the Editor

Peripheral facial palsy due to SARS-CoV-2: Do not miss the differentials

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We read with interest the article by Paudel *et al.* on a 60-years-old female with peripheral facial palsy being attributed to an infection with SARS-CoV-2 ^[1]. In addition to facial palsy, SARS-CoV-2 infection manifested with fever and coughing ^[1]. The patient benefited from prednisolone (30mg/d), eye protection measures, and physiotherapy ^[1]. The study is excellent but has limitations that are cause of concern and should be discussed.

The main limitation of the study is that a causal relation between the mild SARS-CoV-2 infection and peripheral facial palsy remained unconfirmed ^[1]. SARS-CoV-2 was not confirmed in the cerebrospinal fluid (CSF) or other tissues. There was no documentation of a “dissociation cyto-albuminique” to rule out cranial nerve involvement in Guillain-Barre syndrome, and there was no proof that immunological parameters, such as cytokines, chemokines or glial factors were elevated in serum/CSF. The detection of SARS-CoV-2 in the CSF would support the assumption that the facial palsy was actually caused by SARS-CoV-2. Without evidence of the virus in the CSF or the immune response to the virus, a causal relationship between SARS-CoV-2 and facial palsy remains speculative.

Furthermore, it remains unclear why the patient was treated for ischemic stroke with acetyl-salicylic acid but was diagnosed with peripheral facial palsy 6 hours after admission. This discrepancy should be explained. We should whether there were any indications in addition to the initial clinical exam suggesting that the patient had experienced a vascular cerebral lesion. Was the patient a smoker, was there hyperlipidemia, or atrial fibrillation? We should be informed about the results of the initial blood tests and whether the patient was taking any regular medication. It should be also reported whether the patient had undergone follow-up cerebral imaging to eventually document an ischemic stroke in the brainstem or supratentorially.

A further limitation of the study is that neither cerebral imaging by means of magnetic resonance imaging (MRI) nor MRI of the cranial nerve roots with contrast medium had been done. If peripheral facial palsy is not due to pathology in the facial canal, it may be due to radiculitis of the cranial nerves. In case of cranial nerve radiculitis, the nerve roots in the MRI may be thickened or enhancing ^[2].

A further limitation is that no nerve conduction studies (NCSs) of the facial nerves were recorded. NCSs of the facial nerve are imperative to confirm that the lesion was indeed of the peripheral type and to monitor recovery over time.

There is no mention how differential etiologies of facial palsy were ruled. There is no mention of the results of the virus panel, measurement of antibodies against *Borrelia burgdorferi*, or of blood/CSF cultures. Since facial palsy is most commonly due to herpes or varicella viruses, it is crucial that these viruses have been ruled out alongside SARS-CoV-2.

Overall, the interesting study has limitations that put the results and their interpretation into perspective. Clarifying these limitations would strengthen the conclusions and could improve the study. Before attributing peripheral facial palsy to SARS-CoV-2 alternative etiologies must be thoroughly ruled out.

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Keywords: SARS-CoV-2, Facial Palsy, Peripheral Nervous System, Steroids

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