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LETTER TO THE EDITOR

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SARS-CoV-2 complicated by transverse myelitis

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Abstract

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1 | LETTER TO THE EDITOR

With interest we read the article by Shahali et al. about a 63yo male with COVID-19 who experienced transverse myelitis 4d after clinical onset of an infection with SARS-CoV-2 (1 . The patient presented with a transverse syndrome T8 with flaccid lower-limb paraparesis, sensory disturbances below T8, and urinary retention(1. Transverse myelitis did not resolve upon application of methyl-prednisolone intravenously during 3d which is why immunoglobulins were initiated with success (1. The report is appealing but raises the following comments and concerns.

Transverse myelitis as a neurological complication of an infection with SARS-CoV-2 is not unique but increasingly recognised as a neurological complication of COVID-19. In a recent case report and review 11 cases with transverse myelitis were listed, including details about clinical, imaging, and biochemical findings and the therapeutic management of these patients [2]. Since other patients with SARS-CoV-2 associated myelitis have been reported since then, at least 20 patients with this manifestation of neuro-COVID are currently available.

There is a discrepancy between the statement in the introduction, that currently no "definite treatment" of COVID-19 is available and the fact that the patient was nonetheless treated with hydrochloroquine, azithromycin, and ritonavir. Additionally, these compounds are potentially neurotoxic [3], why they should be avoided or given with caution if indicated. Not only should be discussed that currently widely applied therapeutic agents for COVID-19 are applied only as symptomatic measures and are usually beneficial for secondary damage of the virus but also that some of them are potentially toxic and can explain the neurological manifestations in some cases.

A further shortcoming is that no reference limits were provided in table-1, making it difficult for the

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reader to assess what is normal or abnormal. Missing is a PCR test for SARS-CoV-2 in the cerebro-spinal (CSF) fluid. Missing are cytokine levels in the CSF. Occasionally, virus particles have been detected in the CSF (6% of patients undergoing a spinal tap) [4 why it is crucial to test the CSF for virus-RNA. Missing is an axial MRI image of the spinal cord showing the extension of the lesion in the horizontal plane.

We should be told about the outcome of the urinary retention. Did the patient require a disposable catheter or a permanent catheter? Did urinary retention completely resolve upon application of steroids and immunoglobulines?

Missing are nerve conduction studies to rule out neuropathy as the cause of weakness and sensory disturbances.

Missing are follow-up MRIs of the spinal cord to confirm that not only symptoms and signs had resolved but also the underlying lesion in the spinal cord.

Whether SARS-CoV-2 reactivates poliomyelitis or herpes viruses in spinal cord neurons, remains elusive. Single reports indicate that SARS-CoV-2 weakens the cellular and humoral immune response why exacerbation of herpes labialis may occur (5.

Overall, the interesting report has several limitations which should be accomplished before drawing final conclusions. A causal relation between myelitis and SARS-CoV-2 can be established only by documentation of virus RNA in the CSF or the myelin. Otherwise, a causal relation can be suspected but remains unconfirmed

Declarations

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Data availability: not applicable

Consent to participate: not applicable

Consent for publication: not applicable

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