Int. j. adv. multidisc. res. stud. 2025; 5(4):489-490

Received: 28-06-2025
Accepted: 08-07-2025

International Journal of Advanced Multidisciplinary Research and Studies

ISSN: 2583-049X

Letter to the Editor

The diagnosis of SARS-CoV-2 associated transverse myelitis despite normal imaging makes the diagnosis questionable

Josef Finsterer
Department of Neurology, Neurology & Neurophysiology Center, Vienna, Austria

DOI: https://doi.org/10.62225/2583049X.2025.5.4.4634
Corresponding Author: **Josef Finsterer**

Letter to the Editor

We were interested to read the article by Hamaguchi *et al*. about a 59-year-old man with a 4.5-year history of ulcerative colitis (UC) who developed transverse myelitis (TM) concurrently with SARS-CoV-2 infection (SC2I) [1]. The patient was treated with molnupiravir and methyl-prednisolone and partially recovered at 6- and 12-month follow-up [1]. The study is noteworthy, but several points should be discussed.

The first point is that the diagnosis of TM is not confirmed [1]. Although the patient presented with a sensory level at Th5 and with paraparesis and paraspasticity, he had no urinary bladder or bowel dysfunction and no deep sensitivity disorder. In addition, magnetic resonance imaging (MRI) of the spinal cord showed no lesion that could explain the lower limb paraparesis and sensory level at Th5. MRI of the spinal cord was normal not only at admission but also on day 11 after admission [1]. TM due to SC2I is usually visible as a hyperintense lesion on short T1 inversion sequences (STIR), on T2 and on T1 or T2 with contrast [2, 3].

The second point is that a causal relationship between TM and SC2I is uncertain. Several arguments speak against a causal relationship. 1. the virus was not detected in cerebrospinal fluid (CSF). 2. the TM occurred at the same time as the SC2I outbreak, indicating that pathogens other than SARS-CoV-2 were probably the cause. 3. alternative causes for TM could not be sufficiently ruled out. Although infection with influenza and 14 other unspecified infectious agents were ruled out, the results of a comprehensive viral panel, including HIV, HSV, HZV, EBV, polio, Coxsackie, West Nile, Echo, Zika, hepatitis, mumps, and CMV in CSF, were not reported. We should also know the results of CSF culture, fungal culture of CSF and Löwenstein culture and whether stroke and venous congestion of the spinal cord had been carefully excluded. As the patient had a reduced CSF glucose and a history of long-term immunosuppression, a chronic tuberculous infection, clinically manifested by SC2I, must be excluded. The antibodies against neuromyelitis optica (NMO) should also have been determined.

The third point is that the detailed treatment of UC was not reported [1]. It was only stated that he received several types of treatments, including a TNF-alpha blocker [1]. However, the exact treatment when paraparesis occurred was not mentioned. Since chronic immunosuppression can impair immunocompetence [4], it cannot be excluded that immunosuppression may have promoted secondary infection in addition to SC2I. We should also know whether the treatment of UC was discontinued with the detection of SC2I and the onset of paraparesis.

The fourth point is that the immunogenic nature of pleocytosis has not been studied in depth. Of the cytokines, only IL-6 has been measured, but it has also been reported that other cytokines such as IL-1B, IL-4, IL-8, and IL-9 may be elevated in the CSF of patients with CNS complications after SC2I. Other factors that have been shown to be elevated are chemokines, 14-3-3, neurofilament light chains and tau protein [5]. If CSF is still available, these parameters should be determined. It would also have been useful to determine CSF antibody levels associated with autoimmune encephalitis.

In summary, the diagnosis of transverse myelitis is questionable if imaging is normal. Before attributing lower limb paraparesis to SC2I, all other possible causes must be thoroughly ruled out.

Declarations
Ethical approval: Not applicable.

Consent to participation: Not applicable.

Consent for publication: Not applicable.

Funding: None received.

Availability of data and material: all data are available from the corresponding author.

Completing interests: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Author contribution: JF was responsible for the design and conception, discussed available data with coauthors, wrote the first draft, and gave final approval. xx: contributed to literature search, discussion, correction, and final approval.

Acknowledgements: None.

Keywords: SARS-CoV-2 Infection, Transverse Myelitis, Paraparesis, Spinal Cord, Ulcerative Colitis

References
1. Hamachi T, Fujita-Nakata M, Shojima Y, Uchida N, Nakanishi M, Itoh T, *et al*. Acute transverse myelitis following SARS-CoV-2 infection during treatment for ulcerative colitis with anti-TNFα therapy. Clin Neurol Neurosurg, May 2025; 252:108854. Doi: 10.1016/j.clineuro.2025.108854
2. Gemici Yİ, Çelik F, Göz ZZ, İşisağ A, Ovalı GY, Zileli M, *et al*. Anaplastic Ependymoma and Atypical Refractory Longitudinal Expansive Transverse Myelitis Due to Immune Reaction After COVID-19: A Case Discussion That Raises Many Unknown Questions About Covid-19. Noro Psikiyatr Ars, Nov 21, 2023; 61(4):370-373. Doi: 10.29399/npa.28464
3. Tapia-Fonseca CV, Cortés-Enríquez OD, Raya-Garza LP, Gutiérrez-Cuellar DM. COVID-19 associated transverse myelitis: Case report. Bol Med Hosp Infant Mex. 2024; 81(3):191-194. Doi: 10.24875/BMHIM.23000179
4. Hussain Y, Khan H. Immunosuppressive Drugs. Encyclopedia of Infection and Immunity, 2022, 726-740. Doi: 10.1016/B978-0-12-818731-9.00068-9.
5. Sasaki LMP, Canellas-de-Castro ME, Fernandes GM, Motta F, Araújo Júnior DA, Schulte HL, *et al*. Systemic and cerebrospinal fluid immune mediators coordinate a dichotomic microenvironment in parturients with acute or convalescent phases of COVID-19. Immunol Lett. 2025 Aug; 274:106979. Doi: 10.1016/j.imlet.2025.106979