Comment on 'MOG antibody-associated encephalomyelitis mimicking bacterial meningomyelitis following ChAdOx1 nCoV-19 vaccination: a case report'

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We read with interest the article entitled 'MOG antibody-associated encephalomyelitis mimicking bacterial meningomyelitis following ChAdOx1 nCoV-19 vaccination: a case report' by Escolà *et al.*[1] about a 43-year-old female who developed paraparesis, urinary retention, headache, spasticity, meningism, and fever with an Expanded Disability Status Scale (EDSS) score of 5, nine days after having received the first dose of the Astra Zeneca vaccine (AZV). Despite application of ceftriaxone, ampicillin, methylprednisolone, and plasma exchange, the condition progressed to coma and spastic tetraparesis and granulocytic pleocytosis further increased.[1] Since extensive search for infectious agents was negative but myelin oligodendrocyte glycoprotein (MOG) elevated in the cerebrospinal fluid (CSF), a MOG antibody-associated disorder (MOGAD) was diagnosed and the therapeutic regimen changed to meropenem, methylprednisolone, plasma exchange, and tocilizumab.[1] Under this regimen, the patient had partially recovered at the 3 months follow-up.[1] The study is appealing but raises concerns that need to be discussed.

We disagree that the index case is the first developing MOGAD following a vaccination with a vector-based SARS-CoV-2 vaccine.[1] In a recent study of 27 patients developing central nervous system (CNS) demyelinating disease after vaccination with AZV, five patients developed MOG-associated optic neuritis, three MOG-associated transverse myelitis, and two MOG-associated acute, disseminated encephalomyelitis (ADEM).[2] Furthermore, MOGAD has been also reported after mRNA-based anti-SARS-CoV-2 vaccines.[3]

We also disagree with the conclusions that mRNA-based SARS-CoV-2 vaccines should be considered in patients with vector-based SARS-CoV-2 vaccine associated MOG encephalomyelitis.[1] MOGAD has been also reported in patients who received mRNA-based vaccines.[3]

Although the authors claim that infectious agents have been excluded as cause of granulocytic pleocytosis, a culture of the CSF is missing. In addition, it was not reported whether tuberculosis was specifically excluded as the cause of the clinical presentation and the laboratory findings. Furthermore, we should know whether vasculitis, sarcoidosis, syphilis, lymphoma, Whipple disease, parasitosis, infectious foci outside the CNS (sepsis, endocarditis, malignoma), and HIV were appropriately excluded. These disorders may go along with granulocytic pleocytosis.[4]

Since the patient also received meropenem for granulocytic pleocytosis, it should be discussed whether the antibiotic rather than methylprednisolone, the plasma exchange, or tocilizumab were beneficial. We should be told for how long meropenem was applied.

Marked granulocytic pleocytosis is unusual in MOG-associated disorders.[5] In a study of 163 CSF samples from 100 patients with MOGAD, pleocytosis was present in more than half of the patients,[5] but it consisted mostly of lymphocytes and monocytes.[5]

Elevated MOG antibodies in the CSF are a non-specific finding and occur in other inflammatory central nervous system (CNS) disease as well.[6] In a recent study of 474 patients with suspected inflammatory demyelinating disease, elevated MOG antibodies were found in 19 patients with MOGAD, 9 patients with other inflammatory

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demyelinating disease, and 4 patients with multiple sclerosis.[6]

How do the authors explain that the patient initially progressed to cerebral involvement despite a 5-day cycle of methylprednisolone and one session of plasma exchange?

Overall, the interesting study has several limitations and inconsistencies that call the results and their interpretation into question. Clarifying these weaknesses would strengthen the conclusions and could improve the status of the study. Since elevated CSF MOG antibodies are nonspecific, MOGAD should be diagnosed not before exclusion of all disorders that can go along with elevated MOG antibodies. Granulocytic pleocytosis in MOGAD requires extensive work-up to exclude possible differentials.

Ethics approval and consent to participate

Ethics approval was in accordance with ethical guidelines. The study was approved by the institutional review board. Consent to participate was obtained from the patient.

Consent for publication

Consent for publication was obtained from the patient.

Author contributions

Josef Finsterer: Data curation; Formal analysis; Validation; Writing – original draft; Writing – review & editing.

Fulvio A Scorza: Formal analysis; Methodology; Software; Validation.

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Conflict of interest statement

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Availability of data and materials

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