

**LETTER TO THE EDITOR**

# Comment on "Parainfectious Anti-Glial Fibrillary Acidic Protein-Associated Meningoencephalitis"

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Dear Editor,

We enjoyed reading the case report by Joo et al.,<sup>1</sup> which reported parainfectious autoimmune encephalitis (AE) with glial fibrillary acidic protein (GFAP) antibodies. Autoimmune-mediated meningoencephalitis has been noted and actively studied in recent years. The clinical spectrum of anti-GFAP-mediated autoimmune disorders encompasses meningoencephalitis, myelitis, movement disorders, epilepsy, and cerebellar ataxia.<sup>2,3</sup> In the case report, Joo et al.<sup>1</sup> speculated that viral meningitis in the patient might trigger autoimmune processes to produce anti-GFAP autoantibodies, resulting in AE. Likewise, other researchers reported a case of autoimmune GFAP astrocytopathy after herpes simplex viral encephalitis.<sup>4</sup>

However, we need to consider another scenario in the case report regarding the clinical course and laboratory findings of the patient. We think that the patient might have just one etiology of anti-GFAP-associated meningoencephalitis. We have several reasons for this assumption. First, both the clinical course and laboratory findings, including serial cerebrospinal fluid (CSF) profiles of the patient, seem to be monophasic. Moreover, the patient's symptoms and CSF profiles showed considerable improvement with immunotherapy but not with antiviral therapy. Second, no responsible viral pathogen was identified, despite extensive CSF tests for various viral markers. The diagnosis of parainfection in autoimmune-mediated meningoencephalitis could be convincing only after confirming an infectious organism. Third, Yang et al.<sup>5</sup> recently reported that the symptoms in some GFAP-positive patients could mimic viral meningitis. Taken together, the case report can be regarded as one of the diseases in the spectrum of anti-GFAP-associated meningoencephalitis, which ini-

tially started from meningitis and evolved into encephalitis.

In conclusion, the manifestations in this case may be due to two possibilities. One is that viral encephalitis induced AE with anti-GFAP antibodies, as described by Joo et al.<sup>1</sup> in the manuscript. The other is that anti-GFAP meningoencephalitis could initially present as meningitis immediately followed by encephalitis.

**Ethics Statement**

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1975 Helsinki declaration and its later amendments or comparable ethical standards.

**Conflicts of Interest**

The authors have no financial conflicts of interest.

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**Author Contributions**

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