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Letter to the Editor

In Cases of Atypical Acute Flaccid Myelitis, Search for Causes Other than the Common Viral Infections should be Initiated

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Letter to the Editor

We read with interest the article by Wang *et al*. about four patients (Patient 1: 22 months old, female; Patient 2: 11.5 years old, female; Patient 3: 12.5 years old, male; Patient 4: 14 years old) with atypical acute flaccid myelitis (AFM) [1]. Three patients showed no viral prodromal phase and had marked sensory symptoms; one patient presented with symmetrical weakness [1]. All suffered from bowel and bladder dysfunction, one had pleocytosis, and all showed grey matter involvement on imaging [1]. Two patients had severe motor neuron damage, and a short hospital stay was associated with better functional outcomes [1]. The study is promising, but some points require discussion.

First, the term "acute flaccid myelitis" should no longer be used. Myelitis cannot be "flaccid." Only muscle weakness can be flaccid. Flaccidity refers to muscle tone and not to inflammation of the spinal cord.

Second, patient 4 showed involvement of the cranial nerves (nerves IX and X) [1], which casts doubt on the diagnosis of AFM. Brainstem imaging in patient 4 showed involvement of the ventral medulla oblongata. Similarly, pure AFM cannot be diagnosed in patient 1 because the dorsal pons was also affected [1]. Due to the mild pleocytosis in patient 1, brainstem encephalitis with myelitis is more likely than pure AFM.

Third, the cerebrospinal fluid (CSF) was only tested for poliovirus, enterovirus, parechovirus, echovirus, and rhinovirus [1]. The CSF of all patients should have been additionally tested for herpes simplex virus, rabies virus, Zika virus, West Nile virus, HIV, Japanese encephalitis virus, SARS-CoV-2, coxsackievirus, echovirus, and measles.

The fourth point is that immune myelitis/encephalitis was not considered and correctly ruled out. Particularly in patients 3 and 4, in whom no virus could be detected, it would have been important to test for antibodies against autoimmune encephalitis. In these two patients also ADDEM, MOGAD and NMO should be ruled out.

Fifth, no pulmonary function tests were performed in patients 1–3. Since AFM can affect the respiratory muscles [2], as in patient 4, it would have been essential to examine all patients for involvement of the primary and secondary respiratory muscles. Respiratory muscle involvement is particularly likely in patients 1 and 4; however, both patients also exhibited brainstem involvement. Regarding the respiratory insufficiency in patient 4, it should be clarified whether this was due to involvement of the medulla oblongata, the spinal cord from C1 to T5, or both.

Finally, there is a discrepancy between the description of patient 4, according to which the spinal MRI showed T2 hyperintensity between the ventral medulla oblongata and T8, and Table 1, which describes spinal cord involvement between C1 and T5. This discrepancy should be resolved.

In summary, atypical manifestations of AFN should prompt investigations into alternative viral or immunological causes. To improve treatment outcomes for these patients, it is essential to identify the underlying cause and treat it appropriately.

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