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Letter to the Editor

MOGAD Should be Diagnosed Based on Established Criteria, and a Stroke Should be Attributed to it Only After Alternative Causes have been Ruled Out

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Letter to the Editor

We read with interest the article by Leung *et al.* regarding a 58-year-old female patient who suffered a hemorrhagic stroke presenting with left-sided hemianopia, left-sided neglect, and left-sided hemiparesis of unclear etiology [1]. Two weeks after discharge, she developed bilateral amaurosis attributed to bilateral optic neuritis in the context of MOG antibody-associated disease (MOGAD) [1]. Vision improved partially under glucocorticoid therapy [1]. Although the study is interesting, several points warrant discussion.

A first point is that the criteria used to diagnose MOGAD in the index patient were not reported [1]. MOGAD is typically diagnosed in accordance with international consensus guidelines [2], which require the presence of three key components: a core clinical demyelinating event, the detection of IgG anti-MOG antibodies, and the exclusion of all alternative diagnoses. Core clinical events include optic neuritis, transverse myelitis, acute disseminated encephalomyelitis, cerebral mono- or polyfocal deficits, and brainstem or cerebellar deficits. Were all differential diagnoses indeed thoroughly ruled out? The most common differential diagnoses for MOGAD include multiple sclerosis, acute neuromyelitis optica spectrum disorders (NMO-SD) with elevated aquaporin antibodies, disseminated encephalomyelitis (ADEM, AHLE), chronic inflammatory demyelinating polyneuropathy, neuroborreliosis, neurosyphilis, neurosarcoidosis, Behçet's disease, cerebral vasculitis, central nervous system lymphoma, posterior reversible encephalopathy syndrome (PRES), and Leber's hereditary optic neuropathy [3]. To rule out these differential diagnoses, an examination of the cerebrospinal fluid and the intracerebral arteries using appropriate methods would have been necessary. Vasculitis has only rarely been described as a manifestation of MOGAD [4].

A second point is that the detection of anti-MOG antibodies in the index patient could represent a false-positive result [5]. False-positive results have been reported particularly in patients with a low pre-test probability of MOGAD or with low antibody titers [5]. Therefore, the specific test used to detect anti-MOG antibodies, whether the test was repeated, and the test-retest reliability should be reported. The gold standard for detecting anti-MOG antibodies is a cell-based assay (CBA), with a titer >1:100 considered a positive result [6]. Low-positive or borderline titers (1:10 to <1:100) require at least one supporting clinical or MRI feature (such as specific patterns of nerve or brain lesions) to confirm MOGAD [6].

A third point is that the presumed causal link between MOGAD and the hemorrhagic stroke remains questionable [1]. There is currently no evidence in the literature indicating that ischemic or hemorrhagic stroke is a clinical manifestation of MOGAD. Furthermore, the index case provides no plausible pathophysiological explanation for how the formation of anti-MOG antibodies could have caused a hemorrhagic stroke. Based on these considerations, it is unlikely that MOGAD was responsible for the hemorrhagic stroke in the index patient.

The fourth point is that the index patient was not sufficiently evaluated regarding the cause of the hemorrhagic stroke or whether it was a primary or secondary form. Major causes of primary hemorrhagic stroke include arterial hypertension, aneurysm formation, arteriovenous malformations, head trauma, anticoagulant medication, amyloid angiopathy, and hereditary or acquired coagulopathies. Given the patient's history of arterial hypertension, information regarding blood pressure levels at admission and the quality of blood pressure control shortly before admission would be important.

The fifth point is that no follow-up imaging of the stroke was provided. Was the blood completely resorbed, or did a structural lesion remain? Was the hemorrhagic stroke classified as primary or secondary? What did the residual morphology of the hemorrhage look like? Was there any evidence of an ischemic stroke with secondary hemorrhage?

Finally, no reference ranges were provided for the laboratory parameters ^[1]. This makes it difficult to assess which values fell within the normal range and which deviated from it. What was the blood pressure at admission?

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