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LETTER TO THE EDITOR

Miller Fisher syndrome mimicking myasthenia gravis after Epstein-Barr Virus

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Miller Fisher syndrome (MFS) is an acute immune-mediated neuropathy within the anti-GQ1b antibody spectrum and is classically characterized by ophthalmoplegia, ataxia, and areflexia.[1,2] Although preceding infections are commonly reported, atypical presentations may complicate early recognition.

We describe an 18-year-old previously healthy female who presented with a four-day history of sore throat followed by progressive headache, vertigo, bilateral ptosis, binocular diplopia, and gait imbalance. She was initially evaluated by the Department of Otorhinolaryngology due to vertigo, where no peripheral vestibular disorder was identified. Neurological examination demonstrated preserved deep tendon reflexes in all extremities. The patient additionally described worsening of ocular symptoms toward the evening, which initially raised suspicion for myasthenia gravis (MG).

Initial laboratory evaluation demonstrated lymphocytosis (63.1%) and elevated liver enzymes consistent with acute hepatitis (aspartate aminotransferase [AST] 272 U/L, alanine aminotransferase [ALT] 586 U/L, and gamma-glutamyl transferase [GGT] 226 U/L). Acute Epstein-Barr virus (EBV) infection was supported by positive viral capsid antigen (VCA) immunoglobulin (Ig) M and positive early antigen (EA) IgG.

Brain magnetic resonance imaging and magnetic resonance venography revealed no acute abnormalities. Lumbar puncture performed on the second hospital day demonstrated normal cerebrospinal fluid protein concentration and normal cell counts. Since ocular MG remained among the differential diagnoses, acetylcholine receptor antibodies and anti-muscle-specific kinase (MuSK) antibodies were evaluated and were negative.

Additional immunological evaluation demonstrated antinuclear antibody (ANA) positivity (1:120) together with anti-Ro positivity in the absence of sicca symptoms or clinical findings suggestive of connective tissue disease. Serum anti-ganglioside analysis demonstrated positivity for anti-GQ1b IgG and anti-GT1a IgG antibodies.

Taken together, the antecedent EBV infection, neurological findings, anti-ganglioside

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antibody profile, and clinical course supported the diagnosis of MFS within the anti-GQ1b spectrum. Intravenous immunoglobulin therapy was initiated. Clinical improvement became evident by treatment day 2, and near-complete recovery of ptosis, diplopia, and gait imbalance was achieved by day 5. A written informed consent was obtained from the patient.

This clinical course raised several practical points. First, preserved reflexes and normal early cerebrospinal fluid findings early in the disease course did not exclude MFS, consistent with previous observations.[2,3] Second, fluctuating ocular symptoms created substantial diagnostic overlap with MG. One unexpected finding was concurrent ANA and anti-Ro positivity during the acute phase of illness. Since the patient had no sicca symptoms or additional findings supporting connective tissue disease, these serological findings were interpreted cautiously and were not considered sufficient to establish an independent systemic autoimmune diagnosis. EBV-associated anti-GQ1b-positive MFS has rarely been reported in the literature.[4]

In conclusion, this case illustrates that preserved reflexes, fluctuating ocular symptoms mimicking myasthenia gravis, and concurrent transient autoantibody positivity may further complicate early diagnosis.

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