CLINICAL COMMENTARY ON THE BROADENING SPECTRUM OF MYELIN OLIGODENDROCYTE GLYCOPROTEIN ASSOCIATED DISORDER (MOGAD)

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Myelin oligodendrocyte glycoprotein associated disorder (MOGAD) manifests with variable clinical phenotypes.[1] Its clinical and radiological features have recently been defined as a distinct phenotype of the neuromyelitis optica spectrum disorder (NMOSD).[2] Therefore, MOGAD is often considered in the differential diagnosis of other demyelinating conditions, such as atypical multiple sclerosis, acute disseminated encephalomyelitis, and Aquaporin 4-antibody-associated NMOSD.

This case report highlights a potentially broadening phenotype of MOGAD by describing an elderly patient with bilateral orbital inflammatory changes causing optic neuritis. This atypical presentation of bilateral optic neuritis would normally entertain other inflammatory conditions within the differential diagnosis such as sarcoidosis, lymphoma, IgG4-related disease.[3]

Furthermore, even if the typical age of onset for MOGAD tends to be childhood-young adulthood,[4] this case highlights that elderly patients are not excluded from being affected (>70 yrs old).[5] Therefore, in the elderly, after ruling out other causes of subacute optic neuropathy, MOG-IgG antibodies could be tested.

Although MOGAD relapses tend to recover well, patients may still be left with clinical disability. Interestingly this case reports lack of visual recovery. The authors postulate that a superimposed ischaemic optic neuropathy may have contributed to this lack of recovery. In patients of advancing age, high dose corticosteroid treatments (and plasmapheresis) may also have greater complication risks, hence immunosuppressive treatment decisions may require more careful consideration. Future studies could determine if perineuritic or orbital inflammatory involvement are negative prognostic factors for recovery.

In conclusion, MOGAD is a disorder whose boundaries are still evolving, and future vigilance will enrich its recognized clinical spectrum. Meanwhile, clinicians should not limit MOG-IgG antibody testing to the differential diagnosis of multiple sclerosis or other mimics but could extend this test to patients presenting with visual disturbances suggestive of atypical optic nerve involvement.

References:

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