MSJ Experimental Translational Clinical

Letter

Uveitis, optic neuritis and MOG

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Multiple Sclerosis Journal—Experimental, Translational and Clinical

April–June 2020, 1–2

DOI: 10.1177/2055217320925107

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A 59-year-old woman, an agricultural scientist, presented with blurring of vision in the right eye for 4 days, along with painful eye movements and decreased vividness of colours. There were no complaints of watering or redness of the eyes. Examination revealed a visual acuity of 6/18 in the right eye (which did not correct with pin hole) and relative afferent pupillary defect. Fundus showed mild temporal pallor. The rest of the neurological and systemic examination was unremarkable. In the past 3 months she had experienced two similar episodes (once in the right then in the left eye). She had been treated at an outside hospital with intravenous steroids (methylprednisolone, 1 g for one day) followed by oral prednisolone at a dose of 1 mg/kg body weight which was gradually tapered over a period of a month. She had attained complete recovery after administration of steroids in both these episodes. She had previously been diagnosed with anterior uveitis after being evaluated for multiple episodes of blurry vision, redness and watering of the eyes. Although a course of topical steroids led to complete recovery on every occasion, she continued to experience recurring episodes of uveitis. Evaluation for various etiologies of recurrent uveitis ranging from systemic immune-mediated causes to infective causes was negative and it was labelled idiopathic uveitis.

Based on the clinical history and examination findings a diagnosis of optic neuritis was made. Her complete blood picture, biochemical profile including kidney and liver function test were within normal limits. Magnetic resonance imaging of the brain with gadolinium contrast revealed enhancement of the right optic nerve. Cerebrospinal fluid examination was unremarkable with negative oligoclonal bands. The angiotensin-converting enzyme level was normal. The vasculitis profile and anti-aquaporin 4 IgG antibodies were negative. Cell-based immunoassay with immunofluorescence was strongly positive for anti-myelin oligodendrocyte glycoprotein (MOG) IgG antibodies. She was treated with intravenous steroids (methylprednisolone 1 g per day for 5 days) followed by 1 mg/kg body weight of oral prednisolone. She was also started on azathioprine as a steroid-sparing agent. She has not developed any further episodes of optic neuritis and uveitis since then.

Uveitis is associated with central nervous system demyelinating disorders such as multiple sclerosis.[1] Anti-MOG antibodies are strongly associated with bilateral and sequential optic neuritis. However, the association of MOG antibodies and uveitis is not well studied. Ramanathan et al. recently described four cases with uveitis and positive anti-MOG antibodies.[2] Similarly, in another series of 105 patients with multiple sclerosis, two of five patients who tested positive for MOG antibodies were found to have coexisting uveitis.[3] Shao et al. in their experiment on mice demonstrated that MOG peptide could induce uveitis.[4] Intermediate uveitis is the most common type of uveitis in multiple sclerosis. In the Ramanathan series, two out of three patients had bilateral anterior and intermediate uveitis and one had intermediate and posterior uveitis. As MOG is not present in the unmyelinated structures of the eye its contribution to the pathogenesis of uveitis is poorly understood.

It is possible that the patient's episodes of anterior uveitis were a part of MOG spectrum disorder. The literature pertaining to the possible role of anti-MOG antibodies in causing uveitis is growing. Indeed, there could be more to it than just a chance association. This unusual case of uveitis, optic neuritis and positive anti-MOG antibodies highlights that the

clinical spectrum of anti-MOG antibody-associated disease is evolving and uveitis could be a part of it.

Conflict of Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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