• Letter to the Editor •

A case of post-infectious anti-myelin oligodendrocyte glycoprotein antibody-positive optic neuritis

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Dear Editor,

Myelin oligodendrocyte glycoprotein (MOG) is a minor component of myelin, expressed on the external surface of oligodendrocytes in the central nervous system (CNS)[1]. Anti-MOG antibodies (MOG-ab) have been implicated in the demyelinating process and are considered unique biomarkers for a group of heterogeneous autoimmune inflammatory CNS diseases known as MOG-associated disorder (MOGAD)[1]. MOGAD can present with a range of clinical manifestations, including optic neuritis, transverse myelitis, acute disseminating encephalomyelitis, and brainstem or cerebral encephalitis[1]. Optic neuritis is the most common clinical feature of MOGAD in adults, typically manifesting as steroid-sensitive, recurrent, bilateral optic neuritis with optic disc swelling[1].

We reported a case of MOG-ab-positive optic neuritis that developed following eczema herpeticum and complicated bacteremia in a patient with chronic atopic dermatitis.

Ethical Approval This study was approved by the Institutional Review Board (IRB; IRB number: 2206-014-115). Informed consent was obtained from the patient.

A 30-year-old man presented with decreased visual acuity and gaze-evoked eye pain in the left eye for three days. He had a medical history of atopic dermatitis for 25y and was taking oral methotrexate (10 mg per week). Two months prior to the onset of visual symptoms, he presented to the emergency department with fever and extensive vesicular and erosive skin lesions on his face, trunk, and upper limbs. He was clinically diagnosed with eczema herpeticum, complicated by secondary skin and soft tissue infections and *Staphylococcus aureus* bacteremia. Methotrexate was discontinued, and he was treated with systemic antiviral and antibiotic agents for three weeks. At the initial visit, his best-corrected visual acuity was 0.8 in the right eye and 0.04 in the left eye. Relative afferent pupillary defect was noted in the left eye. Visual field deficit was present in both eyes and was more prominent in the left eye. Color vision was impaired bilaterally. Fundus and slit-lamp examinations revealed no specific findings. Magnetic resonance imaging (MRI) revealed focal enhancement of the bilateral retrobulbar optic nerves (Figure 1). In laboratory tests, serum anti-aquaporin-4 antibodies was negative and anti-MOG antibody was positive. A diagnosis of bilateral optic neuritis was made, and the patient was treated with intravenous methylprednisolone (1000 mg/d) for three days, followed by a tapering course of oral prednisolone (1 mg/kg, tapered to 10 mg per week). One month later, visual acuity was restored to 1.0 in the right eye and 0.8 in the left eye.

Due to the characteristic steroid dependency and high relapse rate of MOG-ab-positive optic neuritis, a maintenance dose of 10 mg of prednisolone was maintained. Methotrexate was not restarted, as his skin lesions did not recur. Six months after the first attack, the patient returned with complaint of gaze-evoked eye pain and decreased visual acuity measuring 0.125 in the right eye. Inferior half and central visual field were deteriorated, and MRI showed that increased extent of focal enhancement at the right intraorbital optic nerve consistent with recurrent optic neuritis. High-dose steroid therapy was re-administered, resulting in an improvement of visual acuity in the right eye to 1.0. Azathioprine (25 mg per day) was initiated alongside the tapered steroid. At the final follow up, conducted three years after the first attack, no further episodes were observed. Prednisolone was continued at a maintenance dose of 10 mg, along with azathioprine 25 mg per day, up to the last follow up.

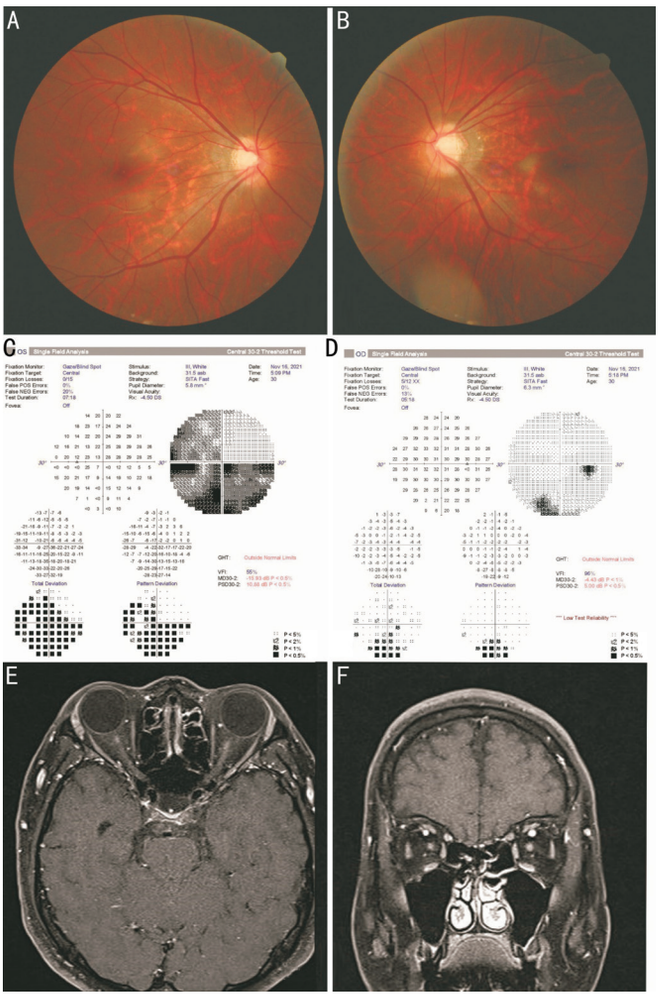

Figure 1 Initial clinical presentation of the patient A, B: Fundus photographs showed no specific findings; C, D: Bilateral visual field defect was observed which was prominent in the left eye; E, F: Bilateral optic nerve enhancement at T1-weighted image in magnetic resonance imaging.

Eczema herpeticum is characterized by skin rash with bullous lesions, typically occurring in individuals with preexisting dermatitis, such as atopic dermatitis. It is most often caused by herpes simplex virus (HSV) primarily type 1 and less commonly by type 2, in patients with inflammatory skin conditions. Eczema herpeticum can lead to life-threatening complications in some cases[2].

Post-infectious optic neuritis may follow various viral and bacterial infections, including infections caused by varicella-zoster virus, HSV, cytomegalovirus, paramyxovirus, Ebstein-Barr virus, and *Mycobacterium tuberculosis*[3]. The reported interval between infection and onset of optic neuritis is 19.5d (range, 14-30d), similar to the timeline observed in our patient [3]. This supports the notion that a delayed autoimmune response may play a role in post-infectious optic neuritis.

Two major mechanisms are believed to contribute to the onset and exacerbation of autoimmune diseases following infection. The first is bystander activation, where T cells not specific to the pathogens become activated. The second is molecular mimicry, in which infectious microorganisms possess antigens that cross-react with autoantigens[4]. Cross reactivity with MOG has been reported in both animals and human studies[5]. The pathogen of the infection in our patient could be attributed to cross-reactivity with MOG epitope in CNS could accelerate the infection-autoimmune link. Given the unique sequence of systemic infection and the onset of optic neuritis in our patient, it is plausible that the preceding eczema herpeticum or *Staphylococcus aureus* bacteremia contributed to the pathogenesis of MOG-ab-positive optic neuritis. Additionally, chronic atopic dermatitis which is a chronic inflammatory skin disease with immune dysregulation and barrier defect, could be one of the risk factors of post-infectious optic neuritis.

MOG-ab-positive optic neuritis occurred after the eczema herpeticum. It may offer insights into the pathophysiology of MOGAD. Considering the characteristic clinical features and steroid responsiveness, screening for MOG-ab-positivity should be considered in patients with post-infectious optic neuritis and proactive and aggressive therapeutic approaches is needed.

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