World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(CASE REPORT)

MOG antibody-associated optic neuritis: A case report

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World Journal of Advanced Research and Reviews, 2025, 26(03), 1652-1658

Publication history: Received on 30 April 2025; revised on 11 June 2025; accepted on 13 June 2025

Article DOI: https://doi.org/10.30574/wjarr.2025.26.3.2271

Abstract

The spectrum of Myelin Oligodendrocyte Glycoprotein Antibody Diseases (MOGAD) is a new entity in the spectrum of autoimmune inflammatory pathologies of the central nervous system. We report the case of 28 year old male, who presented for headache and retro orbital pain increased on eye movement. Examination of the fundus after dilatation revealed a bilateral disc swelling. Brain and optic tract MRI revealed edema of both optic nerves, associated with a T2 hypersignal with enhancement after gadolinium injection. The etiological work-up revealed positive anti-MOG antibodies. MOGAD-related optic neuritis often presents with bilateral involvement, severe visual loss, and optic disc swelling. MRI findings and serologic testing for MOG-IgG are essential for accurate diagnosis. This case highlights the importance of considering MOGAD in patients with atypical optic neuritis, especially when bilateral and associated with disc swelling.

Keywords: MOGAD (Myelin Oligodendrocyte Glycoprotein Antibody Disease); MOG (myelin oligodendrocyte glycoprotein); Anti-MOG antibody; Myelitis; Optic neuritis (ON); Case report

1. Introduction

The spectrum of Myelin Oligodendrocyte Glycoprotein Antibody Disease (MOGAD) is a new entity in the spectrum of autoimmune inflammatory pathologies of the central nervous system. It differs from multiple sclerosis (MS) and neuromyelitis optica spectrum disease (NMOSD). In fact, although they share some clinical features, these 3 diseases differ in terms of pathophysiology, evolution and treatment[1].

Anti-MOG antibody-associated disease (MOGAD) is an autoimmune disorder where the body's immune system attacks a protein called myelin oligodendrocyte glycoprotein (MOG), which is found on the surface of myelin in the central nervous system (CNS). MOG is a key component of the myelin sheath, which insulates nerve fibers and allows for the efficient transmission of nerve signals[2].

2. Case description

We report the case of 28 year old male, who presented for headache and retro orbital pain increased on eye movement since a week. On examination, relative afferent pupillary defect (RAPD) was negative. The rest of the anterior segment was without abnormality. Examination of the fundus after dilatation revealed a bilateral disc swelling with multiple flaming hemorrhages around the optic disc (figure 1-2). The macula was healthy on both eyes. There were no other infectious or inflammatory signs, no signs of vasculitis, no chorioretinal foci. Visual acuity was 10/10 on both eyes with dyschromatopsia, and the rest of the ophthalmological examination was unremarkable. His neurological signs were otherwise normal. An optical coherence tomography (OCT) scan of the optic nerve was performed, revealing a mean optic fibre height (RNFL) of 201µm in the right eye, and 136µm in the left, confirming bilateral disc swelling (figure 1-2).

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The patient was admitted to the neurology department, where a lumbar puncture with cerebrospinal fluid (CSF) pressure measurement was performed and returned normal.

The patient reported rapidly worsening visual blur within 48 hours, with visual acuity of 2/10 in both eyes. A visual field was performed showing a deficit (figure 3).

Brain and optic tract MRI revealed edema of both optic nerves, associated with a T2 hypersignal with enhancement after gadolinium injection (figure 4). This hypersignal extended from the papilla to the cisternal portion of the optic nerve, but respected the optic chiasm. There was no signal abnormality or contrast enhancement in the brain.

This led to the diagnosis of inflammatory optic neuropathy (ION).

The infectious workup was negative, and the patient was given a corticosteroid bolus. The etiological work-up revealed positive anti-MOG antibodies.

The patient was seen again at the end of their bolus treatment and then again after one month. There was a complete disappearance of orbital pain, an improvement in visual acuity, measured at 10/10 in the right eye and 9/10 in the left eye. Color vision was normal, with a decrease of disc swelling at the fundus. After several months the visual field also showed improvement(figure 5). Fundus examination showed disappearance of disc swelling. OCT revealed multiple optic fiber deficits in the temporal and nasal regions, as well as damage to the ganglion cell complex. (Figure 6-7)

After a peer discussion, the neurologists decided to start immunosuppressive treatments with rituximab to prevent a possible recurrence.

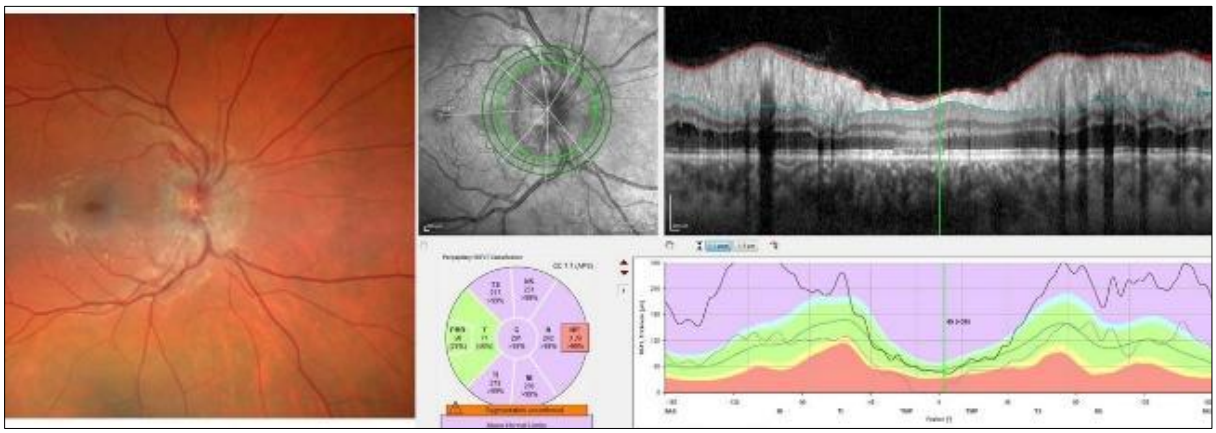

Figure 1 Fundus photograph of the right eye showing disc swelling with flaming hemorrhages around the optic disc confirmed by OCT focused on optic nerve with a mean RNFL fiber height of 201 microns

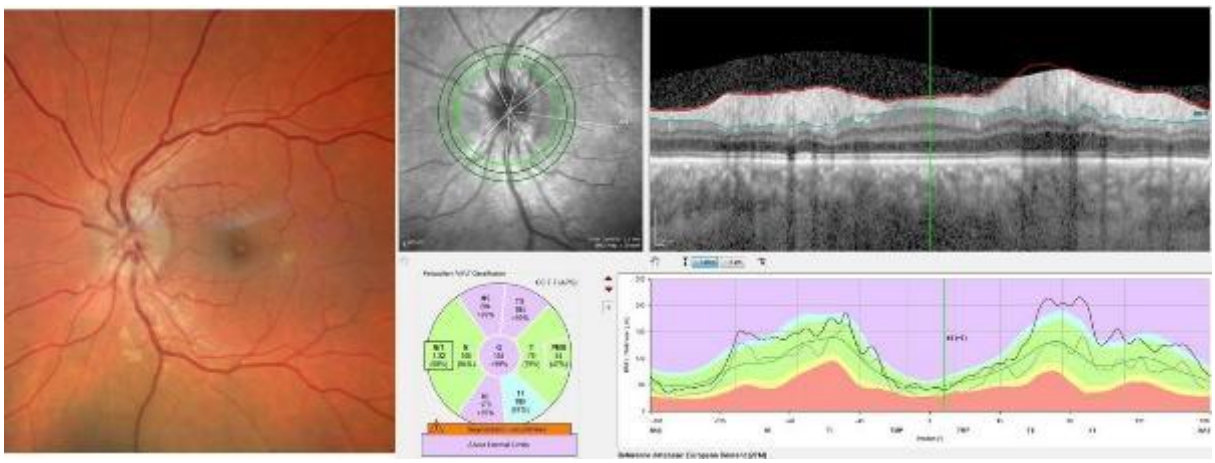

Figure 2 Fundus photograph of the left eye showing disc swelling confirmed by OCT focused on optic nerve with a mean RNFL fiber height of 136 microns

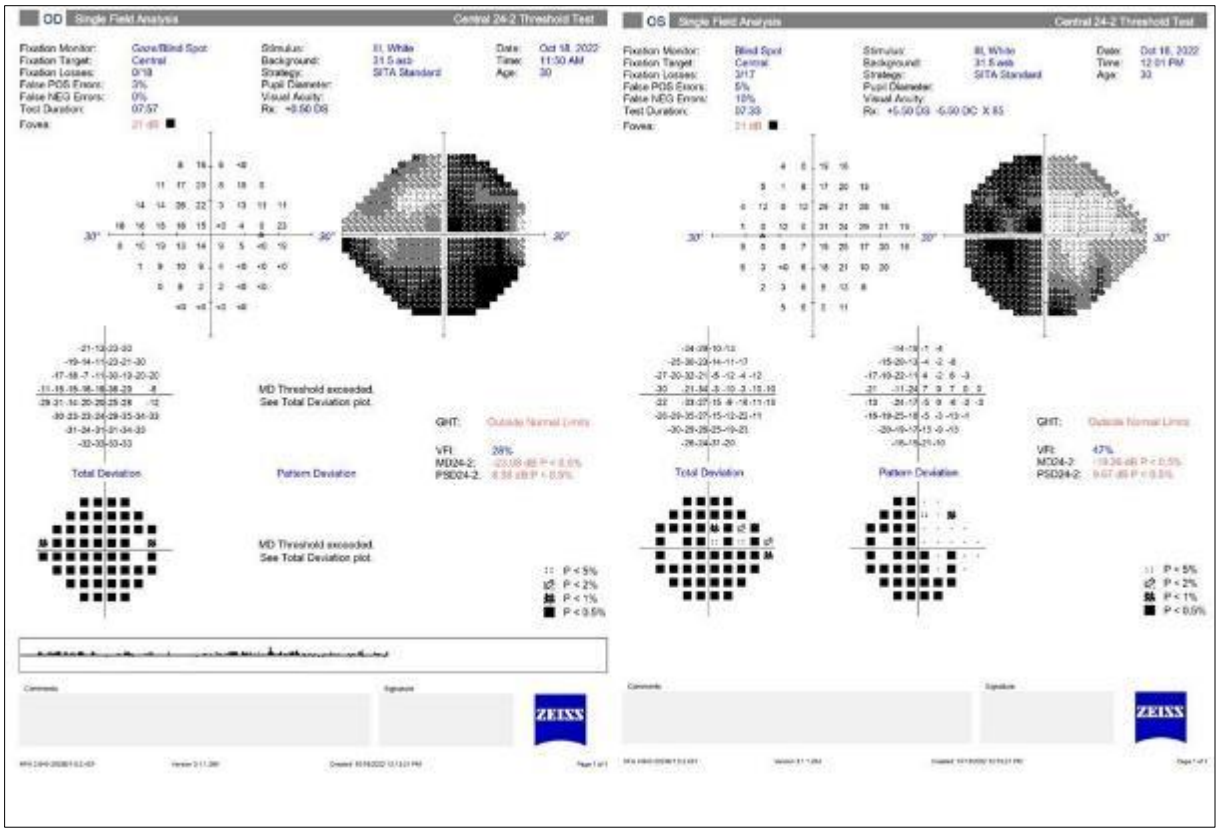

Figure 3 Initial visual field showing diffuse defects

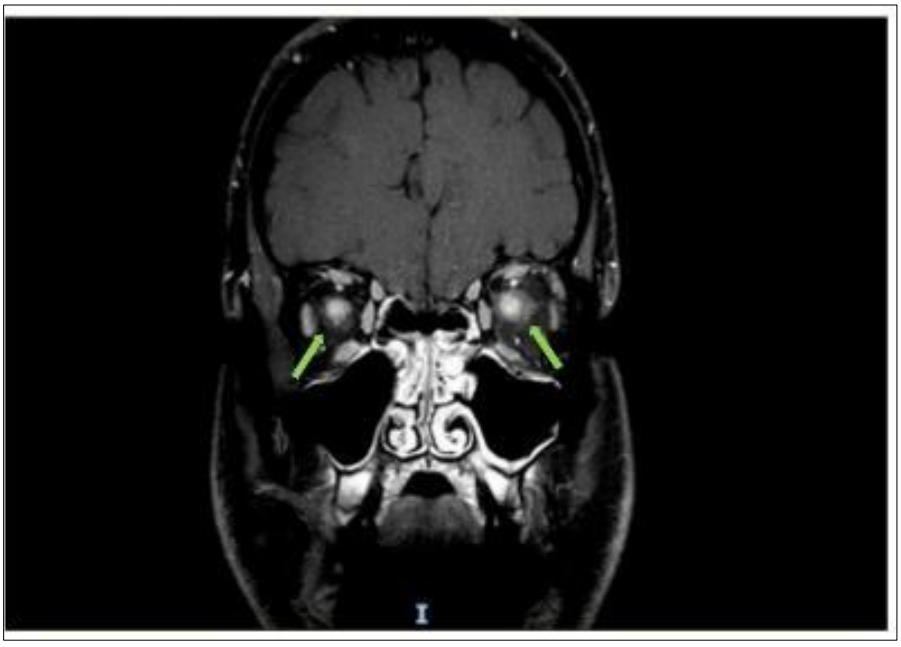

Figure 4 Brain and orbit MRI frontal section T1 sequence after gadolinium injection: enhancement after injection of both optic nerves

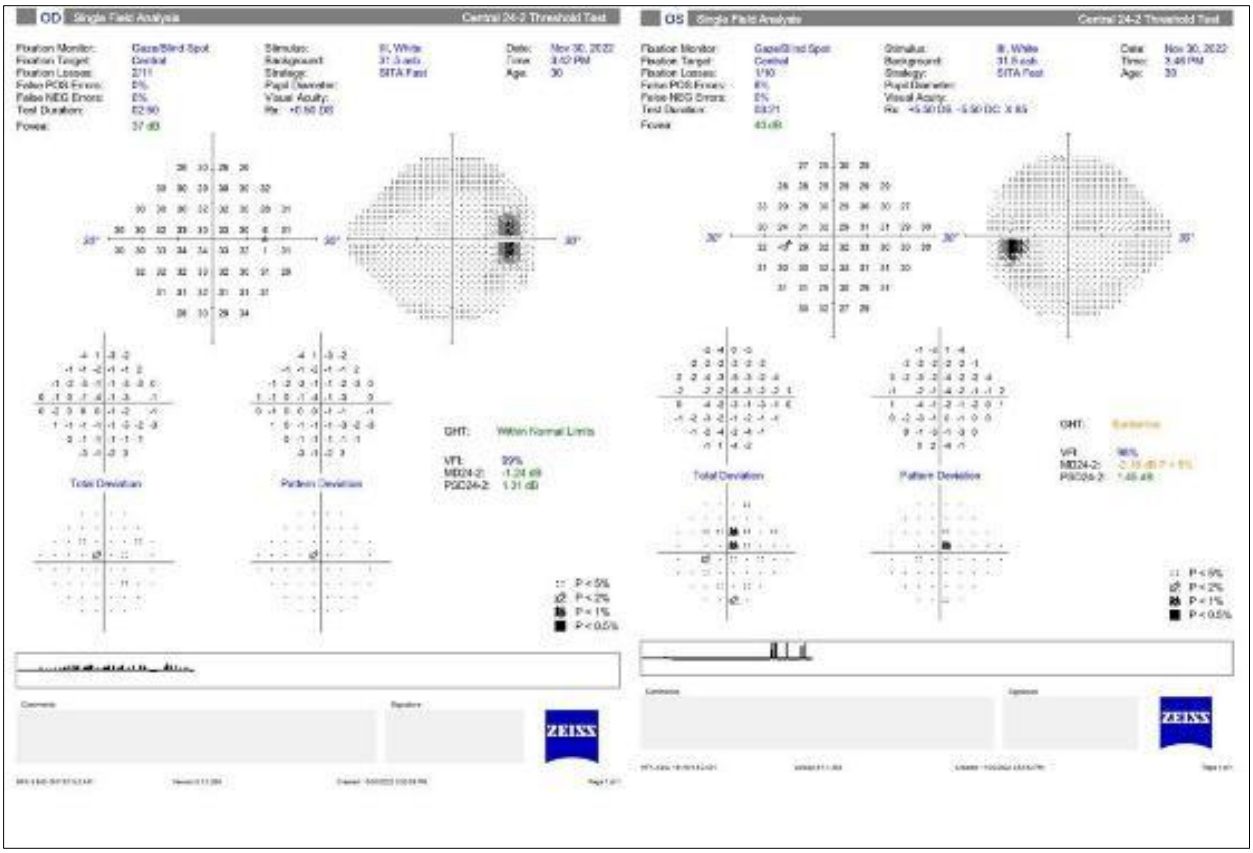

Figure 5 Control visual field showing clear improvement

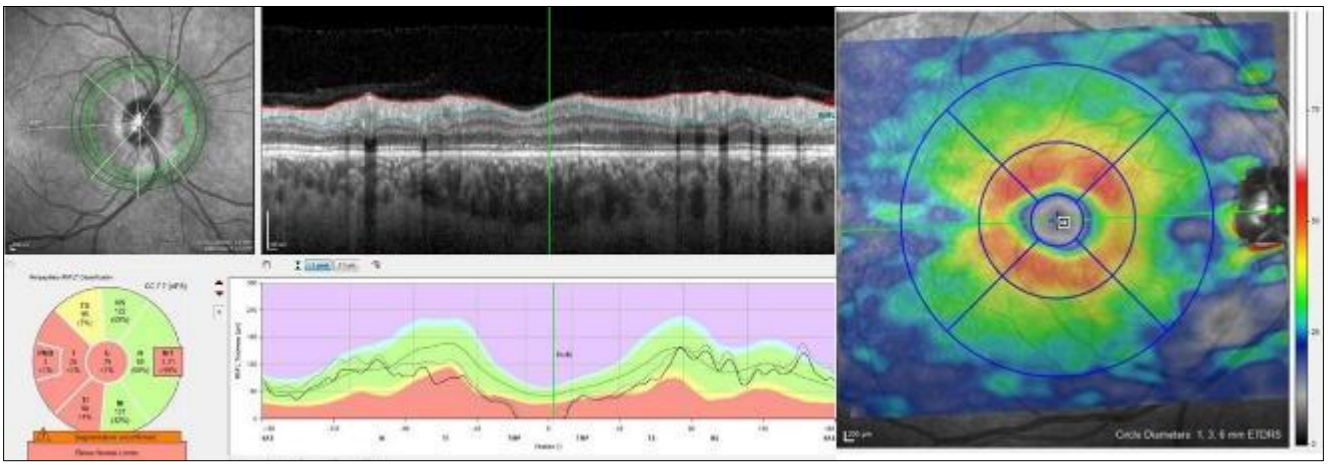

Figure 6 OCT of the right eye revealing multiple optic fiber deficits in the temporal and nasal regions, as well as damage to the ganglion cell complex

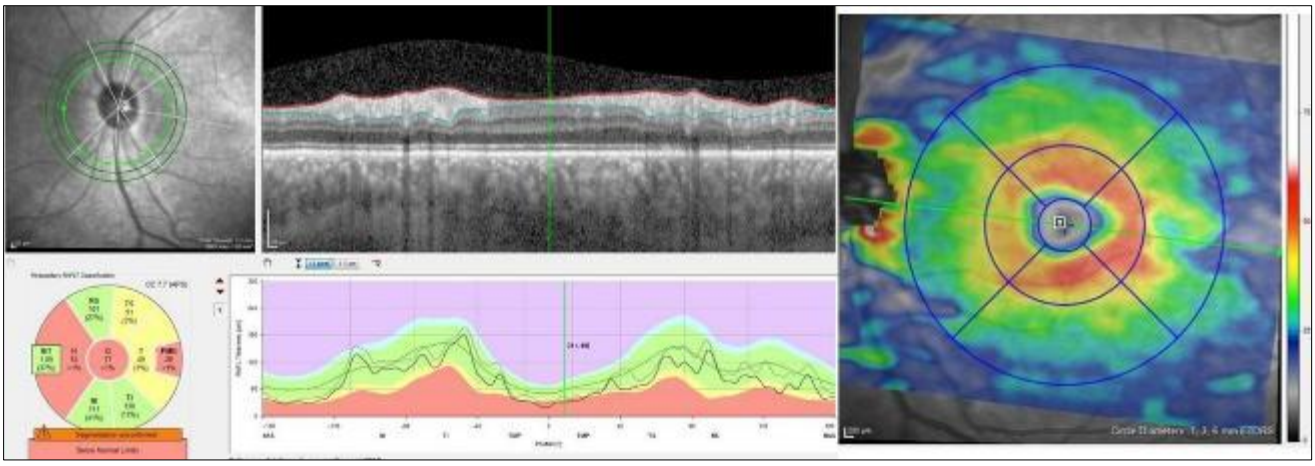

Figure 7 OCT of the right eye revealing multiple optic fiber deficits in the temporal and nasal regions, as well as damage to the ganglion cell complex

3. Discussion

MOGAD is a spectrum of diseases that include conditions like optic neuritis, transverse myelitis, and acute disseminated encephalomyelitis. MOGAD is driven by the activation of T cells and MOG antibodies crossing the blood-brain barrier, leading to inflammation and demyelination. Unlike multiple sclerosis (MS), where the immune system attacks a protein called myelin basic protein, MOGAD targets MOG, resulting in different clinical features and disease behavior[2]. In multiple sclerosis (MS), the pediatric form is more severe than the adult-onset form, whereas in MOGAD, severity is similar in both age groups[3].

They differ also by the way the immune system attacks the central nervous system. MS often affects areas like the periventricular white matter, while MOGAD can affect optic nerves, spinal cord, and brainstem more predominantly[2].

While MOGAD shares some similarities with NMOSD (Neuromyelitis Optica Spectrum Disorder), particularly optic neuritis and transverse myelitis, it differs in the antibodies involved. NMOSD is linked to antibodies targeting aquaporin-4, while MOGAD targets MOG[2].

MOGAD can present in various ways, often leading to episodes of neurological dysfunction. MOGAD in adults often presents with optic neuritis, transverse myelitis, and acute disseminated encephalomyelitis (ADEM)[4]. However, it can also manifest as other neurological disorders, including brainstem and cerebellar syndromes. Damage to the optic nerves is by far the primary mode of onset of MOGAD ; 61% of patients in the French MOGADOR cohort began their disease with isolated optic neuritis (ON), and almost 8% with a combination of ON and myelitis[5].

ON associated with anti-MOG antibodies has its own, albeit non-specific, characteristics, such as bilateral involvement (29 to 45% of cases), severe initial involvement (visual acuity (VA) ≤ 1/10 in 59 to 74% of patients) and, during the first episode, optic disc swelling, sometimes major (66 to 86% of cases)[6]. MRI examination of the optic pathways reveals extensive involvement, predominantly in the anterior region, affecting the optic disc. In contrast, lesions associated with anti-AQP4 antibodies are more posterior, involving the optic chiasm and retrochiasmatic regions[1].

MOGAD diagnostic criteria were proposed by a panel of international experts and published in 2023. Based on their proposed criteria, MOGAD is most commonly associated with acute disseminated encephalomyelitis, optic neuritis, or transverse myelitis, and less frequently with cerebral cortical encephalitis, brainstem, or cerebellar presentations. MOGAD may present as either a monophasic or relapsing condition with the latter being more common, and MOG-IgG cell-based assays play a vital role in accurate diagnosis. While diagnoses such as multiple sclerosis should be ruled out, not all multiple sclerosis patients need to be screened for MOG-IgG. Although these diagnostic criteria require further validation, they hold promise for enhancing the identification of MOGAD cases, which is crucial for determining long-term clinical outcomes, refining clinical trial inclusion criteria, and identifying predictors of relapsing versus monophasic disease progression[4,7].

The main differential diagnosis of ON in MOGAD is MS, by far the most common cause of ON. Cerebral MRI is helpful in making this diagnosis, showing suggestive white matter lesions[6].

The presence of bilateral papilledema in a painful context can sometimes suggest intracranial hypertension (ICHT), as in our patient's case: however, a profound and rapid drop in visual acuity should call into question the diagnosis of ICHT and suggest MOGAD[6].

The management of patients with MOGAD remains the subject of much debate. An international survey gathered data from clinicians worldwide regarding their treatment practices for MOGAD. Findings revealed that steroid therapy is commonly used as the first line of treatment, with some patients also receiving plasma exchange or immunosuppressive drugs like azathioprine or rituximab, especially in cases of relapsing disease. The study emphasizes the need for standardized treatment protocols and further research to optimize long-term outcomes for patients with MOGAD[8].

Disease-modifying treatments could reduce the risk of a new relapse of MOGAD, but they expose patients to long-term side effects, especially in those who may not necessarily experience a relapse. Currently, there are no specific recommendations regarding the initiation of disease-modifying treatments. It seems reasonable to offer preventive treatment for relapses in a patient who has severe functional sequelae from a first episode of MOGAD or in a patient who has experienced multiple relapses of MOGAD[6].

We monitor patients through visual acuity assessments, along with regular OCT exams and visual field tests at least every 6 months. Patients with anti-MOG antibody-positive optic neuritis tend to have good visual outcomes but may experience residual visual field defects and a high risk of recurrence[9].

Structural damage occurs several months after acute damage. The ganglion cell layer thins in less than a month, and optic fiber atrophy appears in 2 to 3 months. In MOGAD-type optic neuritis, optic fiber atrophy is most often global[6].

The MOGADOR study found 44.8% relapses at 2 years and 61.8% at 5 years[5]. The flare-ups are severe, causing significant decreases in visual acuity, but the long-term prognosis remains relatively good in most cases. A study evaluating the long-term visual outcomes of optic neuritis (ON) in patients with MOGAD over a five-year period concluded that their outcomes were generally as good as those in patients with MS and significantly better than those in patients with anti-AQP4-positive NMOSD[10].

4. Conclusion

MOG-related optic neuritis are likely still underdiagnosed. While flare-ups are often severe, the long-term prognosis remains relatively favorable. However, recurrences are very frequent, even with maintenance therapy. The optimal approach for both acute and long-term treatment has yet to be clearly established.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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