

MOG-IgG-Positive Optic Neuritis in an Adolescent Female: A Case Report of Excellent Visual Recovery Following Corticosteroid Therapy

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Abstract: ***Background:** Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory demyelinating disorder of the central nervous system that can present with optic neuritis, particularly in children and young adults. MOG-IgG-associated optic neuritis may cause severe visual loss and optic disc swelling, but visual recovery after corticosteroid treatment can be substantial. **Case presentation:** A 16-year-old female presented with acute-onset visual loss in the left eye. Best-corrected visual acuity was 6/6 in the right eye and 4/60 in the left eye. A relative afferent pupillary defect and reduced colour vision were present in the affected eye, and fundus examination showed optic disc oedema. Visual-field testing demonstrated generalised depression in the left eye. Brain MRI and Orbit was reported as normal. Aquaporin-4 IgG (AQP4-IgG) was negative, while MOG-IgG was positive. The patient received intravenous methylprednisolone 1 g daily for five days, followed by oral corticosteroids at 1 mg/kg/day. Two weeks after treatment initiation, visual acuity in the left eye had improved to 6/6, with reduced disc oedema and improvement in the visual-field abnormalities. **Conclusion:** MOGAD should be considered in adolescents presenting with acute optic neuritis, particularly when severe visual loss and optic disc oedema are present. A normal brain and orbit MRI and does not exclude MOGAD. Early treatment and follow-up are important because the disease may relapse.*

Keywords: MOG-IgG; MOGAD; optic neuritis; adolescent; optic disc oedema; methylprednisolone; corticosteroids.

1. Introduction

Optic neuritis is an important cause of acute visual loss in children and adolescents. It may occur as an isolated inflammatory event or as part of a demyelinating disorder, including multiple sclerosis, aquaporin-4 IgG-positive neuromyelitis optica spectrum disorder (NMOSD), and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). Clinical assessment, neuroimaging, and appropriate antibody testing can help establish the diagnosis and guide management [1].

MOGAD is an inflammatory demyelinating disease associated with antibodies directed against myelin oligodendrocyte glycoprotein. Optic neuritis is one of its principal clinical manifestations, alongside acute disseminated encephalomyelitis and myelitis. International diagnostic criteria emphasise a compatible clinical demyelinating event, MOG-IgG positivity, and supporting clinical or radiological features where required. A validated cell-based assay is preferred for MOG-IgG testing [2].

MOG-IgG-associated optic neuritis may present with marked optic disc oedema and severe visual loss. Many patients respond well to corticosteroids; however, relapses can occur, including in association with rapid corticosteroid tapering or discontinuation [3]. We describe an adolescent with MOG-IgG-positive unilateral optic neuritis who experienced substantial visual recovery after high-dose intravenous corticosteroid treatment followed by oral therapy.

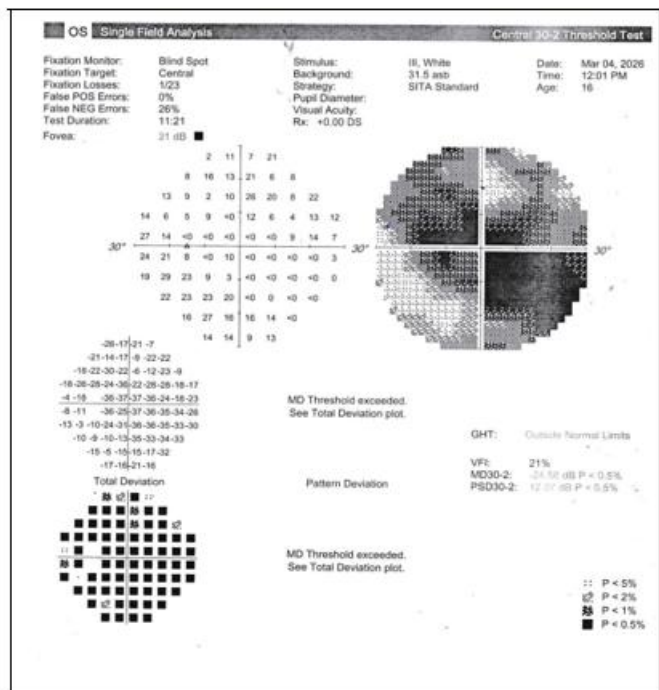
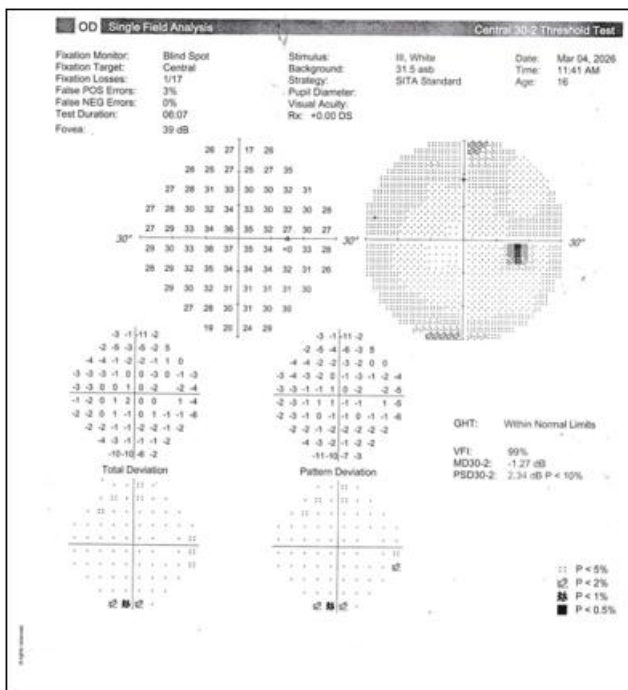
2. Case Presentation

A 16-year-old female presented to the ophthalmology department with acute-onset diminution of vision in the left eye. No previous episode of optic neuritis was documented.

At presentation, best-corrected visual acuity was 6/6 in the right eye and 4/60 in the left eye. Intraocular pressure measured with a non-contact tonometer was 17 mmHg in each eye. A relative afferent pupillary defect and reduced colour vision were noted in the left eye. Fundus examination showed optic disc oedema in the left eye.



Visual-field testing demonstrated generalised depression in the affected eye.

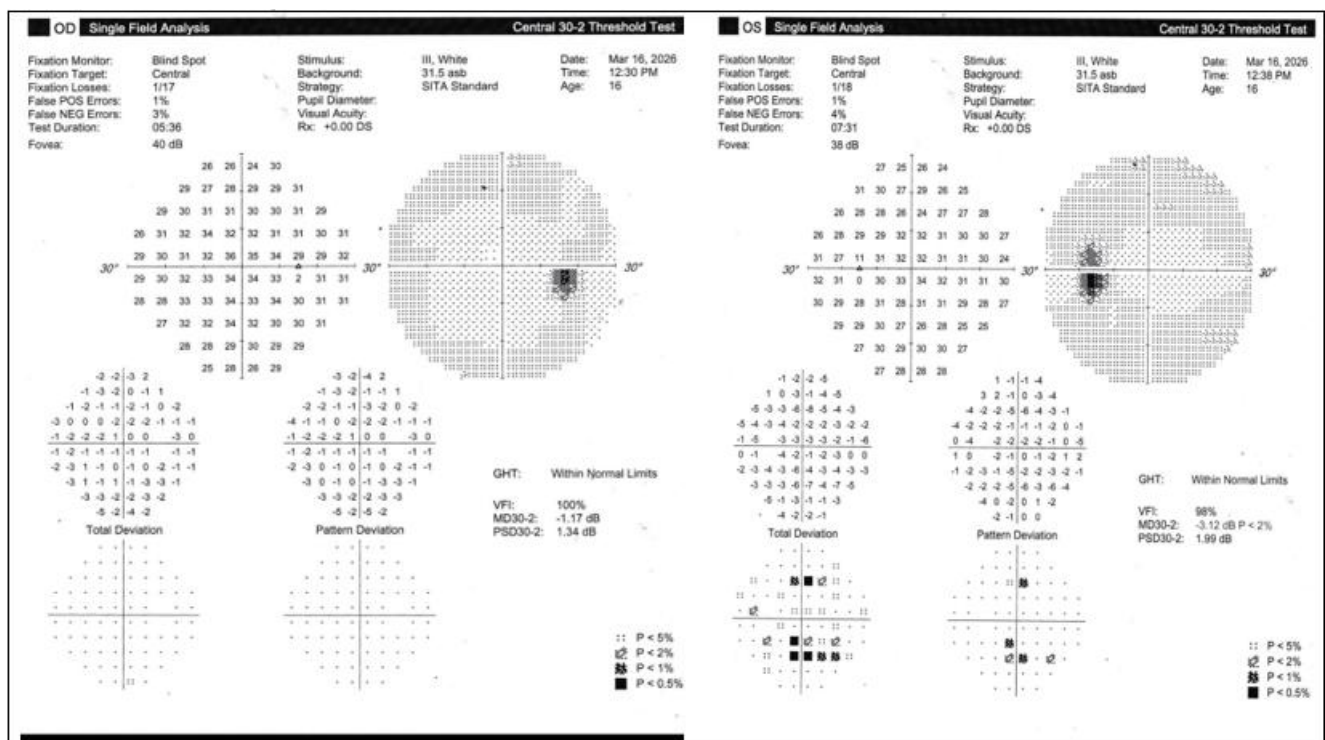


Given the acute unilateral visual loss, relative afferent pupillary defect, dyschromatopsia, disc oedema, and visual-field abnormality, inflammatory optic neuritis was suspected. MRI of the brain and orbits and serological testing for MOG-IgG and AQP4-IgG were requested, and the patient was referred for neurological assessment. The MRI brain and orbit was reported as normal. AQP4-IgG was negative and MOG-IgG was positive.

On the basis of the clinical presentation and antibody results, the patient was diagnosed with MOG-IgG-associated optic

neuritis. She received intravenous methylprednisolone 1 g daily for five consecutive days, followed by oral corticosteroids at 1 mg/kg/day. The oral corticosteroid were slowly tapered was continued over three months.

The patient improved markedly after treatment. Two weeks after treatment initiation, visual acuity in the left eye had improved from 4/60 to 6/6. Optic disc oedema had decreased, and the previously documented visual-field abnormalities had improved.



3. Discussion

MOGAD is an important cause of inflammatory optic neuritis in children and young adults. Its clinical phenotype differs from that of multiple sclerosis and AQP4-IgG-positive NMOSD; therefore, antibody testing is useful when the presentation raises suspicion for an antibody-associated demyelinating disorder. The 2023 international criteria identify MOG-IgG as a key diagnostic biomarker and require interpretation in the context of a compatible clinical syndrome [2,4].

The findings in this patient—acute, severe unilateral visual loss, a relative afferent pupillary defect, impaired colour vision, optic disc oedema, and visual-field abnormalities—were consistent with an inflammatory optic neuropathy. The positive MOG-IgG result and negative AQP4-IgG result supported MOGAD. A normal brain MRI does not exclude MOG-associated optic neuritis; brain imaging may show no

demyelinating lesions in affected patients [5]. The orbital MRI findings should be reported if they are available, as they may provide additional diagnostic information.

Optic disc oedema is a notable feature of this presentation. Although the optic disc may appear normal in some adults with optic neuritis, disc swelling is common in paediatric optic neuritis and is frequently described in MOG-IgG-associated cases [3,6]. This finding should prompt consideration of inflammatory optic neuropathy in a young patient with acute visual loss, while other causes of disc swelling are excluded.

Visual acuity improved from 4/60 to 6/6 within two weeks of initiating treatment with high-dose intravenous methylprednisolone followed by oral corticosteroids. This outcome is consistent with the often favourable acute treatment response reported in MOG-associated optic neuritis. Nevertheless, improvement after corticosteroids is

not, by itself, diagnostic of MOGAD. Treatment duration and tapering should be individualised in consultation with neurology, considering the clinical course and the risk of relapse. Published studies have reported an association between corticosteroid dose or taper duration and time to relapse, but management should follow current specialist guidance and the patient's circumstances [7,8].

MOGAD may follow either a monophasic or relapsing course. Continued ophthalmic and neurological follow-up is therefore important, including monitoring visual acuity, colour vision, pupils, optic nerve appearance, visual fields, and optical coherence tomography when appropriate. Longer-term follow-up is needed to establish whether this patient's course remains monophasic and to identify any recurrence.

4. Conclusion

MOG-IgG-associated optic neuritis should be considered in adolescents presenting with acute optic neuritis, especially when severe visual loss and optic disc oedema are present. A normal brain MRI does not exclude MOGAD, and testing for both MOG-IgG and AQP4-IgG can help distinguish important causes of inflammatory optic neuropathy. High-dose intravenous methylprednisolone followed by an appropriately planned oral corticosteroid regimen may lead to substantial visual recovery. Because MOGAD can relapse, ongoing ophthalmic and neurological follow-up is essential.

Learning Points

- MOGAD is an important cause of optic neuritis in children and adolescents.
- Severe visual loss accompanied by optic disc oedema should raise suspicion for MOG-associated optic neuritis.
- A normal brain MRI does not exclude MOGAD.
- MOG-IgG and AQP4-IgG testing can help distinguish antibody-associated demyelinating disorders.
- High-dose intravenous corticosteroids can be followed by substantial visual recovery in MOG-associated optic neuritis.
- Long-term follow-up is important because MOGAD may relapse.

Declarations

Patient consent: Written informed consent for publication, including any clinical images, should be obtained from the patient and/or parent or legal guardian in accordance with the target journal's requirements.

Conflict of interest: The authors had no any relevant conflicts of interest.

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References

- [1] de-la-Maza MS, et al. Update on pediatric optic neuritis. *Current Opinion in Neurology*. 2022;35(1):55–62.
- [2] Banwell B, Bennett JL, Marignier R, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. *The Lancet Neurology*. 2023;22(3):268–282.
- [3] Jeyakumar N, Lerch M, Dale RC, Ramanathan S. MOG antibody-associated optic neuritis. *Eye*. 2024;38(12):2289–2301.
- [4] Al-Ani A, Chen JJ, Costello F. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): current understanding and challenges. *Journal of Neurology*. 2023;270(8):4132–4150.
- [5] Wynford-Thomas R, Jacob A, Tomassini V. Neurological update: MOG antibody disease. *Journal of Neurology*. 2019;266(5):1280–1286.
- [6] Yeh EA, Graves JS, Benson LA, Wassmer E, Waldman A. Pediatric optic neuritis. *Neurology*. 2016;87(9 Suppl 2):S53–S58.
- [7] Trewin BP, Dale RC, Qiu J, et al. Oral corticosteroid dosage and taper duration at onset in myelin oligodendrocyte glycoprotein antibody-associated disease influences time to first relapse. *Journal of Neurology, Neurosurgery & Psychiatry*. 2024;95(11):1054–1063.
- [8] Chwalisz BK, Levy M. The treatment of myelin oligodendrocyte glycoprotein antibody disease: a state-of-the-art review. *Journal of Neuro-Ophthalmology*. 2022;42(3):292–296.