Case Report

From Darkness to Sight: Rapid Recovery in MOG- Associated Optic Neuritis with High-dose Steroid Therapy

Muhammad Ihtisham

ABSTRACT

Myelin Oligodendrocyte Glycoprotein Optic Neuritis (MOG-ON) is a severe inflammatory condition that can cause significant vision loss if untreated. High-dose corticosteroids are known to aid recovery, though the response varies. This case highlights the potential for near-complete visual restoration with early intervention.

A 30-year-old male of Pakistani origin presented with a five-day history of blurred vision and mild left eye pain. His initial visual acuity was 6/12, improving to 6/9 with pinhole correction. Within five days, vision deteriorated to hand movements despite normal routine blood tests. Following neuro-ophthalmology consultation, intravenous methylprednisolone (1g daily for five days) was initiated, followed by an oral taper. Vision improvement began by the third dose, reaching 6/6 with pinhole correction by day five. Color vision, initially 0/17 on Ishihara testing, fully recovered. OCT imaging and visual field testing confirmed significant optic nerve recovery. The patient tested positive for MOG antibodies and was referred to neurology for further care.

This case demonstrates the efficacy of early, high-dose corticosteroid therapy in rapidly restoring vision in severe MOG-related optic neuritis. Prompt intervention, guided by an interdisciplinary approach, can prevent lasting impairment. MOG antibody disease should be considered in optic neuritis cases to ensure timely diagnosis and optimal treatment.

INTRODUCTION

Myelin Oligodendrocyte Glycoprotein-associated Optic Neuritis (MOG-ON) is an antibody-mediated central nervous system demyelinating disease. It is distinct from Multiple Sclerosis (MS) and Neuromyelitis Optica Spectrum Disorder (NMOSD). Typical retrobulbar Optic Neuritis (ON) presents with acute, unilateral visual acuity loss, retrobulbar pain, reduced color vision, and RAPD with a normal fundus.[1] However, MOG-ON often manifests as bilateral or unilateral optic disc edema, marked steroid responsiveness, and frequent relapses.

MOG-ON is associated with certain HLA haplotypes and affects individuals of any age, ethnicity, or gender.[2] Pediatric patients with MOG antibodies frequently present with Acute Disseminated Encephalo Myelitis (ADEM), while adults aged 20 to 45 commonly experience unilateral ON. Early diagnosis and aggressive treatment are essential to prevent permanent visual disability and relapse.

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Correspondence:
Muhammad Ihtisham
muhammad.ihtisham@stockport.nhs.uk
Stockport Eye Centre
Stepping Hill Hospital Manchester, UK

Author(s) Affiliation: Stockport Eye CentreStepping Hill Hospital Manchester, UK

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CASE REPORT

Patient Presentation:

A male in early thirties, from Pakistan, presented with a five-day history of blurred vision in the left eye, accompanied by mild pain on eye movement. His vision deteriorated to hand movement over five days, while the right eye remained unaffected. There was no significant medical, family, or ocular history.

Clinical-Examination

Initial and follow-up findings:

- **Visual acuity (VA)**: Left eye - 6/9 initially, deteriorating to hand movement over five days; right eye 6/6.
- **Intraocular pressure (IOP)**: 15 mmHg bilaterally.

- **Extraocular movements (EOM)**: Full but associated with mild to moderate pain in the left eye.
- **Red saturation**: Normal initially but reduced over time.
- **Relative afferent pupillary defect (RAPD)**: Absent initially but became prominent later.
- **Fundus examination**: Mild optic disc swelling initially, progressing to severe optic disc edema with vessel tortuosity and engorgement over five days.

Investigations .

Comprehensive autoimmune, inflammatory, and infectious workup yielded normal results. MOG-IgG testing was positive, confirming the diagnosis of MOG-ON. MRI of the head and orbit confirmed isolated left optic neuritis, consistent with the clinical presentation.

Treatment-Plan .

The patient underwent:

1.Acute Treatment: Neuro-ophthalmology consultation followed by 1 g of intravenous methylprednisolone daily for five days. Significant improvement was observed after the fifth dose, including recovery of visual acuity (6/6), resolution of pain, restoration of color vision, and disappearance of RAPD.

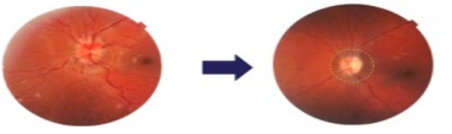
prednisolone (60 mg/day), tapered by 10 mg weekly. Weekly follow-ups showed consistent improvement. However, a relapse occurred when reduced to 5 mg/day from 10 mg/day. Oral prednisolone was increased again to 60 mg/day with slow tapering of 10 mg every 2 weeks this time. The patient is awaiting a neurology appointment for long-term management.

DISCUSSION

MOG-ON is characterized by acute unilateral or bilateral optic neuritis with marked responsiveness to corticosteroids.[3] In this case, the patient presented with unilateral optic disc edema and rapid deterioration of vision. MOG-ON often distinguishes itself from MS and NMOSD by its clinical and imaging features, such as severe optic disc swelling and the absence of demyelinating brain lesions typically seen in MS.

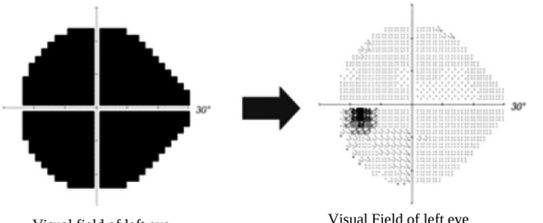
Corticosteroid therapy remains the mainstay of treatment for acute attacks. Evidence suggests that methylprednisolone can enhance recovery by 10-20%.[4] For patients unresponsive to steroids, intravenous immunoglobulin (IVIG) or plasma exchange has shown partial recovery in 40% of cases. Prolonged high-dose steroid therapy significantly reduces relapse rates, although early tapering or discontinuation increases recurrence risks. Immunosuppressants like rituximab are promising options for long-term relapse prevention in recurrent cases.

Prognosis for MOG-ON is favorable, with up to 90% of patients achieving partial or complete visual recovery. However, persistent MOG antibodies and younger age at onset are associated with higher relapse rates and worse visual outcomes. This case underscores the importance of early recognition, aggressive treatment, and vigilant follow-up in managing MOG-ON effectively.



Fundus Photo of Left eye | Fundus Photo of Left eye after 4 weeks

Figure 1(a): Fundus photographs of left eye



Visual field of left eye at Presentation | Visual Field of left eye after 4 weeks

Figure: 1(b): Visual field of left eye

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PATIENT CONSENT

Informed consent was obtained from the patient for publication of this case report and accompanying imag