



OPTIC NERVE INVOLVEMENT AND VISUAL OUTCOMES FOLLOWING CORTICOSTEROID THERAPY IN NEUROMYELITIS OPTICA SPECTRUM DISORDER: A PROSPECTIVE OBSERVATIONAL STUDY

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ABSTRACT

Background: Neuromyelitis optica spectrum disorder (NMOSD) is an autoimmune inflammatory disorder of the central nervous system with prominent involvement of the optic nerves and spinal cord. Optic neuritis in NMOSD can result in severe visual impairment, and recurrent attacks may lead to optic atrophy and permanent visual loss. This study evaluated optic nerve involvement and visual outcomes following corticosteroid therapy in patients with NMOSD.

Methods: A prospective evaluatory study was conducted among 17 patients (34 eyes) diagnosed with NMOSD who attended the neurology department. Demographic and clinical history were recorded. Visual acuity, colour vision, pupillary reactions, anterior and posterior segment examinations were performed. OCT, visual field testing and VEP were performed wherever feasible. MRI findings and AQP4/MOG antibody status were documented. Patients with active disease received intravenous methylprednisolone 1 g/day for 3 days followed by oral prednisolone with gradual tapering and were followed up at 1, 3 and 6 months.

Results: Of 17 patients, 15 (88.2%) were female and 2 (11.8%) were male. Eye involvement was present in 14 patients. Nine patients (52.9%) had bilateral involvement, five (29.4%) had unilateral involvement and three (17.6%) had no eye involvement. Among affected eyes, 12 (52.1%) showed optic neuritis, 6 (26.1%) had disc edema and 5 (21.7%) had optic atrophy. Three eyes (13.0%) had complete visual loss, while 20 (86.9%) had partial visual loss. Significant visual improvement after treatment was reported in 14 affected eyes (60.8%), while 9 (39.1%) showed minimal improvement. Among the significantly improved eyes, 8 were associated with anti-MOG positivity.

Conclusion: Optic nerve involvement is a major ocular manifestation of NMOSD and may progress from unilateral to bilateral disease with recurrent attacks. High-dose intravenous methylprednisolone followed by oral corticosteroid therapy was associated with visual improvement in a substantial proportion of affected eyes. The observed better recovery among anti-MOG-positive eyes is an exploratory finding that requires confirmation in larger studies.

KEYWORDS: Neuromyelitis Optica Spectrum Disorder, Optic Neuritis, Optic Atrophy, anti-AQP4, Anti-MOG, Corticosteroids, Visual Outcome.

Introduction

Neuromyelitis optica spectrum disorder (NMOSD), historically referred to as Devic disease, is a chronic autoimmune inflammatory disorder of the central nervous system characterized predominantly by optic neuritis and myelitis. It is now recognized as a distinct neuroimmunological disorder rather than a variant of multiple sclerosis, with a characteristic clinical spectrum, serological profile and treatment approach[1,2]. Recurrent attacks may result in substantial and sometimes permanent neurological disability, including severe visual loss and paralysis[1,5].

NMOSD predominantly affects women and usually begins in adulthood. Epidemiological studies have demonstrated considerable geographic variation, with the highest prevalence generally reported among females and a peak in the fourth decade in several populations[7]. The disease is frequently associated with antibodies against aquaporin-4 (AQP4-IgG), a highly specific biomarker that supports diagnosis and reflects an astrocytopathic disease mechanism involving complement-mediated injury[3,8]. Myelin oligodendrocyte glycoprotein (MOG-IgG) is identified in a subset of patients with an overlapping optic neuritis phenotype, although MOG antibody-associated disease is now considered a distinct disorder rather than simply a seronegative form of NMOSD[5].

Optic neuritis is one of the characteristic manifestations of NMOSD. Attacks may be unilateral, sequentially bilateral or simultaneously bilateral, and recurrent optic neuritis can result in optic atrophy and profound visual loss[1,5]. In the 2015 international diagnostic criteria, severe optic neuritis, bilateral or rapidly sequential optic neuritis, and optic chiasm involvement are among the clinical features that should raise suspicion for NMOSD[1]. Other characteristic manifestations include longitudinally extensive transverse myelitis and area postrema symptoms such as persistent nausea, vomiting and hiccups[1,5]. Optical coherence tomography and visual evoked potentials can provide objective evidence of optic nerve injury and may be useful in longitudinal monitoring[5].

Acute NMOSD attacks require prompt treatment because delayed therapy is associated with poorer neurological and visual recovery. High-dose intravenous methylprednisolone is the standard initial treatment, while plasma exchange or immunoadsorption should be considered early when recovery after corticosteroids is inadequate or when the attack is severe[6]. Long-term relapse prevention has

evolved considerably and includes immunosuppressive agents such as azathioprine, mycophenolate mofetil and rituximab, as well as disease-specific therapies including eculizumab, inebilizumab and satralizumab for appropriate patients[6,10,11].

The present study evaluated the pattern of optic nerve involvement in patients with NMOSD and assessed visual outcomes following corticosteroid treatment.

Aims and Objectives

1. To evaluate the pattern and frequency of optic nerve involvement in patients with NMOSD.
2. To document clinical, fundus, OCT, visual field and VEP findings wherever feasible.
3. To assess visual outcomes following intravenous methylprednisolone and subsequent oral corticosteroid therapy.
4. To explore the relationship between antibody status and visual recovery.

Patients and Methods

A prospective evaluatory study was conducted among 17 patients (34 eyes) diagnosed with NMOSD who attended the neurology department. Demographic details and relevant clinical history were recorded. Visual acuity, colour vision, pupillary reactions, anterior segment and posterior segment examinations were performed. OCT, visual field testing and VEP were performed wherever feasible, and MRI findings were documented.

Patients with active disease received intravenous methylprednisolone 1 g/day for 3 days, followed by oral prednisolone with gradual tapering. Follow-up assessments were performed at 1, 3 and 6 months.

Results

Seventeen patients were included; 15 were female and 2 were male. Five patients presented with decreased vision, while the remaining patients were evaluated during routine follow-up or for other complaints. Fourteen patients had a pre-existing diagnosis of NMOSD and three were newly diagnosed.

Eye involvement was present in 14 patients, including 2 males and 12 females. Five patients had unilateral involvement and nine had bilateral involvement. The original study also recorded that 10 patients initially had unilateral involvement and that some subsequently progressed to bilateral disease. Nine patients had recurrent ocular involvement, whereas five reported ocular involvement for the first time.

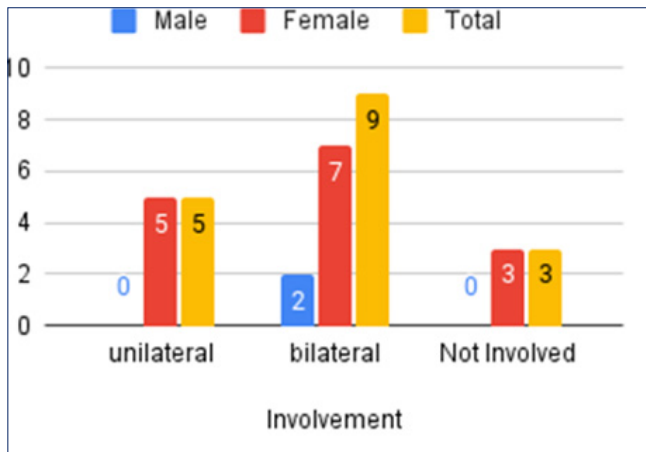


Figure 1. Laterality of eye involvement

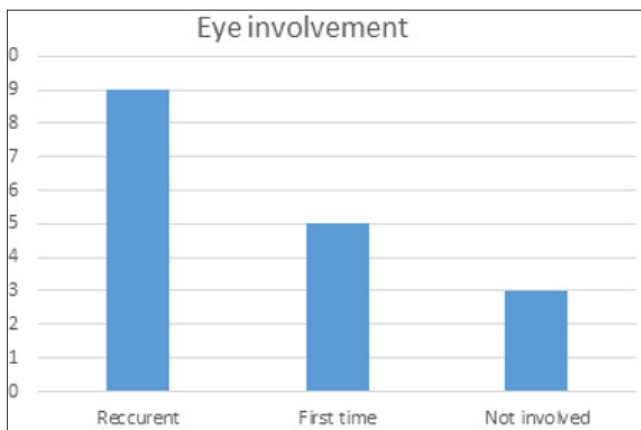


Figure 2. First-time versus recurrent eye involvement

Twelve affected eyes had a relative afferent pupillary defect and four eyes demonstrated colour vision defects. Seventeen eyes showed fundus changes: 12 had optic neuritis and 5 had optic atrophy. Two patients with ocular involvement had painful eye movements.

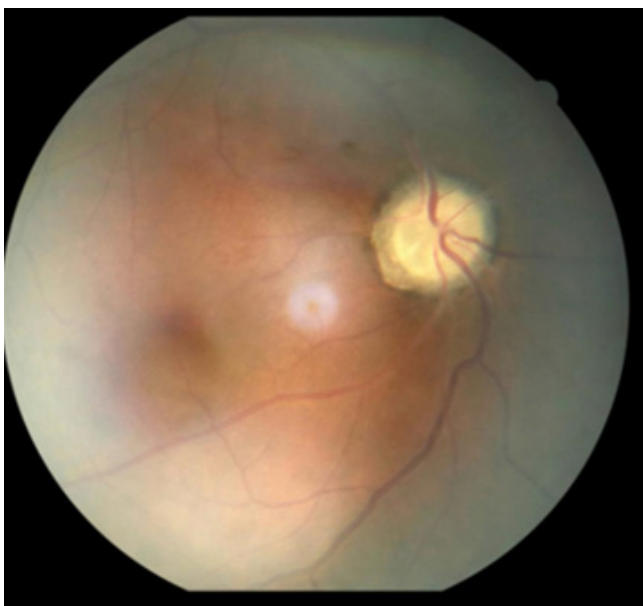


Figure 3. Fundus image showing optic atrophy

VEP was performed in four patients; one showed an absent P-wave response and three showed increased P100 latency. OCT was performed in four patients; three showed decreased RNFL thickness and one showed normal RNFL thickness. Visual field testing was performed in four patients; two showed central scotoma and two showed centrocecal scotoma.

AQP4 antibody was positive in seven patients, of whom five had eye involvement. MOG antibody was positive in six patients, of whom five had eye involvement.

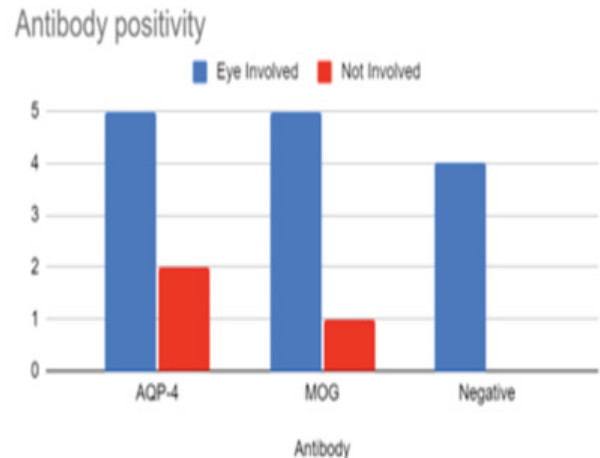


Figure 4. AQP4 and MOG antibody status

MRI demonstrated white-matter hyperintensities involving the optic nerve in 9 patients, of whom eight had eye complaints. MRI was normal for remaining 8 patients, of whom 6 showed eye involvement.

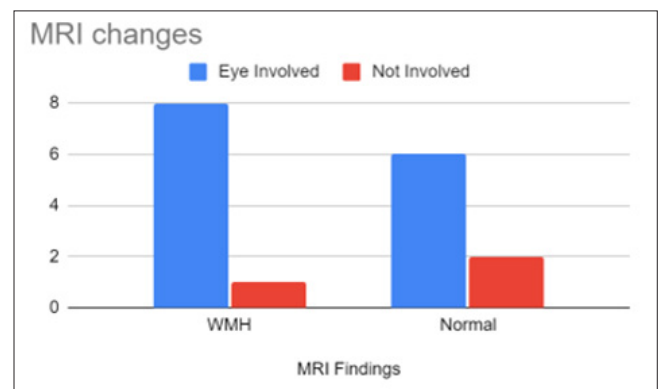


Figure 5. Optic nerve MRI findings

After corticosteroid treatment on 3 and 6 month follow up, 14 affected eyes showed significant visual improvement, whereas 9 showed minimal improvement. Among the eyes with significant improvement, 8 were associated with anti-MOG positivity.

Discussion

In this prospective series, ocular involvement was common among patients with NMOSD and was characterized predominantly by optic neuritis, disc edema and optic atrophy.

The marked female predominance observed in the study is consistent with the established epidemiological pattern of NMOSD, in which women are affected substantially more often than men[5,7]. The age distribution and clinical presentation are also broadly consistent with the adult-onset pattern described in contemporary cohorts[5].

The study demonstrated that optic nerve involvement may begin unilaterally and subsequently become bilateral. This observation is clinically relevant because optic neuritis may be the first manifestation of NMOSD, and recurrent or bilateral optic neuritis should prompt appropriate serological and neuroimaging evaluation[1,5]. Recurrent optic neuritis was common in our cohort, and the presence of optic atrophy emphasizes the potential for cumulative optic nerve damage and permanent visual disability. The 2015 international diagnostic criteria specifically recognize severe or bilateral/rapidly sequential optic neuritis as presentations that should increase suspicion for NMOSD[1].

VEP, OCT and visual field findings provided objective evidence of optic nerve dysfunction in the subset of patients in whom these investigations were feasible. This is consistent with current recommendations that visual acuity, fundus examination, OCT and, where appropriate, electrophysiological testing can complement clinical assessment in NMOSD[5]. OCT studies have demonstrated retinal nerve fibre layer and ganglion-cell loss after optic neuritis in both AQP4- and MOG-antibody-associated disease, reflecting structural injury even when visual function subsequently improves[9]. The apparent association between anti-MOG positivity and better visual recovery in our cohort is noteworthy but should be interpreted cautiously because of the small sample size and limited availability of ancillary investigations. Published comparative studies and meta-analyses similarly report, on average, better visual recovery after MOG-associated optic neuritis than after AQP4-IgG-positive NMOSD optic neuritis[9].

The visual improvement observed after high-dose intravenous methylprednisolone followed by oral corticosteroid therapy supports the importance of prompt treatment during active attacks. Contemporary recommendations emphasize early initiation of high-dose intravenous glucocorticoids, with early escalation to plasma exchange or immunoadsorption in patients with severe attacks or insufficient response to corticosteroids[6]. The visual recovery observed in our cohort should therefore be interpreted in the context of early anti-inflammatory treatment rather than as evidence of corticosteroid efficacy from a controlled comparison. For relapse prevention, modern NMOSD management includes targeted therapies directed against complement, B cells or the interleukin-6 pathway in appropriate patients; randomized trials have demonstrated significant reductions in relapse risk with agents such as eculizumab and satralizumab[10,11].

Conclusion

Optic nerve involvement is an important manifestation of

NMOSD and may present with unilateral or bilateral optic neuritis, disc edema, colour vision abnormalities, RAPD and optic atrophy. Recurrent attacks may lead to progressive bilateral involvement and permanent visual impairment. In this study, corticosteroid therapy was associated with significant visual improvement in a substantial proportion of affected eyes, with greater improvement observed among anti-MOG-positive eyes. Early recognition, timely neurological evaluation, appropriate treatment and regular ophthalmic follow-up are important to reduce the risk of irreversible visual disability.

Limitations

The study included a small sample of 17 patients and was conducted over a short period. OCT, visual field testing and VEP were available only in a subset of patients. These factors limit the ability to draw statistically robust conclusions regarding associations between antibody status and visual recovery. Larger prospective studies with standardized imaging and functional testing would help validate these observations.

Conflict of Interest

The authors do not have any conflicts of interest.

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