Original Research Paper

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Opthalomology

A CLINICAL STUDY ON OPTIC NERVE INVOLVEMENT IN NEUROMYELITIS OPTICA SPECTRUM DISORDER (NMOSD)

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ABSTRACT **Purpose-** To study optic nerve involvement in NMOSD and visual outcome after treatment with IVMP. **Methods-** A prospective evaluatory study was conducted on 34 eyes (17 patients) diagnosed with NMOSD. VA, complete eye examination was done. OCT, visual field and VEP was done wherever feasible. Patients were treated with IV and oral steroids and followed up. **Results-** Out of 17 patients 15 female and 2 male. 5 (29.4%) patients had unilateral, 9 (52.9%) had bilateral and 3 (17.6%) had no eye involvement. 3 (13%) eyes had complete vision loss and 20 (86.9%) had partial vision loss. 12 (52.1%) eyes had optic neuritis, 6 (26.08%) had disc edema and 5 (21.7%) had optic atrophy. Significant visual improvement after treatment was seen in 14 (60.8%) eyes and minimal in 9 (39.1%) eyes. **Discussion-** NMOSD may cause recurrent attacks of ON, optic atrophy and paralysis. IVMP therapy can improve visual outcome in affected eyes. Importance of adequate treatment and regular follow up is emphasized.

KEYWORDS : Neuromyelitis optica, Optic Neuritis, Optic Atrophy, Antibody AQP-4 and MOG, Corticosteroids.

INTRODUCTION

Neuromyelitis optica spectrum disorder (NMOSD), also known as Devic disease, is a chronic disorder of the brain and spinal cord dominated by inflammation of the optic nerve (optic neuritis) and inflammation of the spinal cord (myelitis)[1]. Neuromyelitis optica (NMO) is an autoimmune demyelinating disease associated with recurrent episodes of optic neuritis and transverse myelitis, often resulting in permanent blindness and/or paralysis.

The prevalence of NMOSD is approximately 10 per 100,000 individuals. Most commonly seen in 30-40 years of age group, affecting 10 times more females than males[2].

Pathophysiology is expected to be via immune mediated mechanism, having NMOSD specific antibody AQP-4 (Aquaporin-4)[3]. Other antibody seen is anti- MOG (Myelin Oligodendrocyte Glycoprotein) acting on oligodendrocytes. The inflammatory processes in NMOSD primarily target astrocytes, leading to immune-mediated inflammation and secondary demyelination[4].

Hallmark features of NMOSD include acute attacks of bilateral or, rapidly sequential optic neuritis (leading to severe visual loss) or transverse myelitis (often causing limb weakness, sensory loss, and bladder dysfunction) with a typically relapsing course. Relapsing form of disease is 4-5 times more common in females. Other suggestive symptoms include episodes of intractable nausea, vomiting, hiccups.

Optic neuritis can be caused by any inflammatory condition or may be idiopathic. Sellner et al.[5] suggested that most optic neuritis attacks in NMOSD are unilateral, but sequential optic neuritis or bilateral simultaneous optic neuritis is highly suggestive of NMOSD. Recurrent episodes of ON leads to Optic atrophy and complete loss of vision.

For acute attacks, the standard treatment is high-dose IV corticosteroids, typically methylprednisolone. Plasma exchange may be effective in patients who experience acute severe attacks that do not response to intravenous corticosteroids.[6]

For long-term suppression of the disease, a variety of immunosuppressive drugs are available such as Corticosteroids, azathioprine, mycophenolate mofetil and rituximab.[6]

In 2019, Soliris (eculizumab) was approved by the U.S. FDA for the treatment of NMOSD in adult patients who are anti-AQP4 antibody positive. In 2020, Uplizna (inebilizumab) and Enspryng (satralizumab) were approved for the treatment of NMOSD in adult patients with the same AQP4 antibody.[1]

PATIENTS AND METHODS

A prospective evaluatory study was conducted on 34 eyes (17 patients) diagnosed with NMOSD visiting neurology department from January to March 2025.

Demographic data and proper history was elicited.

Visual acuity, colour vision, anterior segment and posterior segment examination was done. OCT, visual field testing and VEP was done wherever feasible. MRI findings were noted.

Patients with active disease were treated with IV methylprednisolone 1gm/day for 3 days and then oral prednisolone was continued, which was gradually tapered and followed up after 1, 3 and 6 months.

RESULTS

In our study, out of 17 patients, 2 were males and 15 females.

5 patients came with complaints of decreased vision. Other patients came for regular check up or with other complaints and they were taken up for eye examination also. 14 patients prediagnosed with NMOSD and 3 were newly diagnosed.

Among these, 14 patients had eye involvement i.e., 2 males and 12 females.

2 (14%) patients had associated painful eye movements.

5 patients had unilateral involvement and 9 patients had bilateral involvement, among these 2 males had bilateral involvement, thus total of 23 eyes were affected either due to fundus changes or color vison defects.

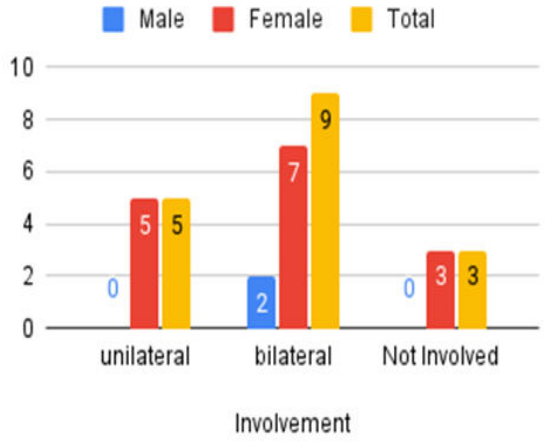

Fig-1 Laterality of eye involvement

10 patients initially had unilateral involvement and gradually some of them progressed to bilateral involvement. Now total 9 patients had bilateral involvement.

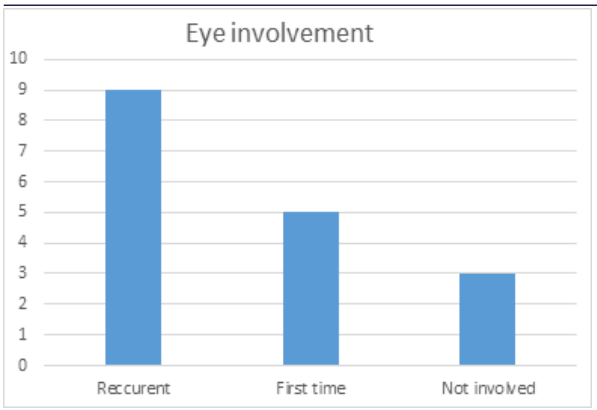

Fig-2 First time v/s recurrent eye involvement

In our study, 9 patient presented with recurrent eye involvement whereas 5 patients complaint of eye involvement for the first time.

12 eyes had Relative Afferent Pupillary Defect (RAPD), 4 eyes had colour vision defect.

17 eyes had fundus changes i.e. 12 had optic neuritis and 5 had developed optic atrophy.

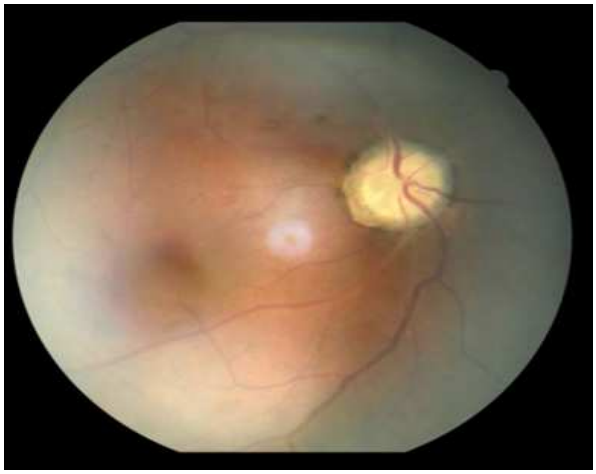

Fig 3- Fundus image showing optic atrophy

VEP was done in 4 patients, 1 patient showed absent P wave and 3 showed increased P-100 latency.

OCT was done in 4 patients, 3 patients showed decreased RNFL thickness and 1 showed normal RNFL thickness.

Visual field testing was done in 4 patients, 2 showed central scotoma and another 2 patients showed centrocecal scotoma.

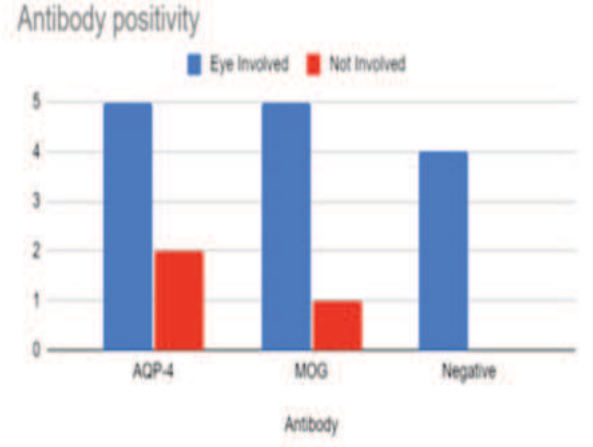

Fig 4- AQP-4 and MOG antibody status

AQP-4 antibody was positive in 7 patients out of which 5 had eye involvement. MOG antibody was positive in 6 patients out of which 5 had eye involvement.

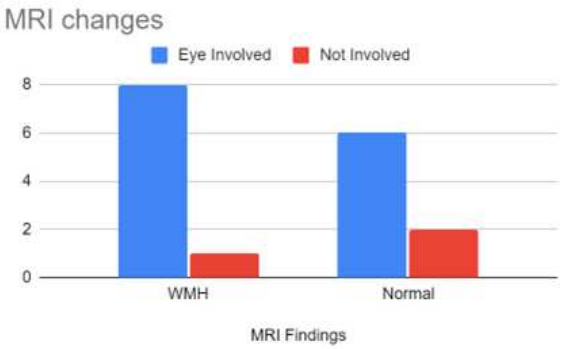

Fig 5- Optic Nerve involvement on MRI

9 patients showed White Matter hyperintensities in Optic Nerve, out of which 8 patients had eye complaints.

Vision improvement post corticosteroid treatment was significant in 14 eyes whereas it was minimal in 9 eyes. Among signifcantly improved vision 8 eyes had Anti- MOG positivity.

CONCLUSION

NMOSD is an idiopathic, chronic, inflammatory disease, having wide spectrum of presentations, mainly affecting optic nerve and spinal cord.

Female preponderence is commonly seen, more common in 30-40 years of age.

Patients who had eye involvement in our study presented with decreased vision, color vision defect, abnormal pupil reactions and fundus changes.

Patients shows good response to IVMP and high dose corticosteroids and shows significant vision improvement. Patients positive for anti-MOG antibody showed more vision improvement.

Patients presenting to ophthalmologists with optic neuritis should be treated suspiciously for these kind of disorders and should be referred to neurologist for evaluation.

Timely diagnosis, adequate treatment and regular follow up can help prevent blindness in these patients.

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