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Late Onset MOGAD Optic Neuritis - Case Report and Literature Review

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Abstract

In MOGAD, the age of onset exhibits a biphasic distribution, typically occurring between 5 to 10 years and 20 to 45 years, with a median age of onset ranging from 20 to 30 years. Our patient experienced onset in the sixth decade of life, which is unusual and infrequently reported, and presented with painful blurred vision in both eyes. The fundus exam revealed bilateral optic disc edema accompanied by pyramidal signs. MRI revealed diffused thickening of bilateral optic nerve up to apex with T2 hyper intensity, with focal T2 FLAIR hyper intensities in frontal and parietal white matter, CSF analysis showed marked pleocytosis (2730 cells), 58% polymorph nuclear cells, 42% mononuclear cells, nil-RBC, with increased protein. Given the patient's age and the involvement of the optic nerves and pyramidal tract, along with a cerebrospinal fluid profile that does not showcase lymphocyte predominance, there is a suspicion of NMOSD. However, since the patient tested negative for aquaporin 4, we moved forward with testing for MOG antibodies, which turned out to be positive.

Conclusion: MOG optic neuritis is infrequent in the sixth decade of life and may manifest similarly to an opticospinal disorder, affecting both the optic nerve and the brain.

Keywords: MOGAD; Bilateral Optic Neuritis; Late Onset MOGAD; Perineuritis

Abbreviations: MOGAD: Myelin Oligodentrocyte Glycoprotein Antibody -Associated Disease; NMOSD: Neuromyelitis Optica Spectrum Disorders; ADEM: Acute Disseminated Encephalomyelitis; CSF: Cerebrospinal Fluid; IVIG: Intravenous Immunoglobulin; IgG: Immunoglobulin G; ON: Optic Neuritis; AQP: Aquaporin; MRI: Magnetic Resonance Imaging; FLAIR: FLuid Attenuation Inversion Recovery; VEP: Visually Evolked Potential; RNFL: Retinal Nerve Fibre Layer; OCT: Optic Coherence Tomography

Introduction

MOG antibody associated disease (MOGAD) is a distinct CNS inflammatory disease with symptoms and imaging findings that overlap other neuro inflammatory disorders [1], with the spectrum of optic neuritis, transverse myelitis, acute disseminated encephalomyelitis (ADEM) and MOG- encephalitis. MOG Optic neuritis frequently occurs in adults, while ADEM is the most common manifestation in children. It is due to the production of IgG autoantibodies that are targeted at myelin oligodendrocyte glycoprotein (MOG), a minor transmembrane protein located on the outermost layers of oligodendrocytes within the myelin sheath of the central nervous system [2]. Approximately 50% of patients with multiple sclerosis (MS) experience typical optic neuritis, which often results in spontaneous recovery. In contrast, atypical optic neuritis associated with MOG antibody disease (MOG-AD) and neuromyelitis optica spectrum disorder (NMOSD) is less likely to resolve on its own. Optic neuritis in MOG-AD may lead to a more favourable visual outcome compared to NMOSD. Recognizing the characteristics of optic neuritis in MS, MOG-AD, and NMOSD is crucial, as the visual prognosis and treatment strategies differ [3-10].

Case Report

A 52-year-old male came in with blurred vision in his right eye that had lasted for 8 days, followed by similar issues in his left eye. He experienced pain when moving his eyes sideways, which began suddenly and was not accompanied by drooping eyelids, double vision, bulging eyes, or tearing. He has no known pre-existing co -morbidities or risk factors. Examination showed sluggishly reacting pupils, drop in the visual acuity RE-6/36 LE-6/60, dyschromatopsia for red, green with Normal extra ocular movements. In addition, he had spasticity and brisk reflexes of all four limbs. With pupils responding sluggishly, a decline in visual acuity, dyschromatopsia, and disc swelling, the patient was clinically diagnosed with bilateral optic neuritis. Further evaluation with OCT revealed swelling of the optic nerve head in both eyes along with significant RNFL thickening. Both eyes' VEP

P100 latencies were prolonged, 118.75 ms for the right eye and 111 ms for the left, with the right eye's amplitude significantly decreasing by 3.03 microvolts. Significant pleocytosis (2730), 58% polymorph nuclear cells, 42% mononuclear cells, nil-RBC, and elevated protein were all seen in the CSF study.

He was not exposed to drugs, toxins; doesn't have constitutional symptoms, no immuno compromised status with that toxic and infectious causes are ruled out. Suspected for demyelinating or paraneoplastic etiology. Patient had bilateral optic nerve disease with marked papilledema which is unlikely for multiple sclerosis and NMOSD, because multiple sclerosis will have patchy involvement of optic nerve and NMOSD involves pre chiasmatic posterior end of optic nerve [11-22]. Though his age profile, bilateral optic nerve disease with marked disc edema and associated pyramidal features were suggestive of NMOSD, MRI revealed bilateral optic nerve long segment involvement with perineuritis with bilateral fronto parietal hyperintensities, features were suggestive of MOGAD and the same was confirmed with cell-based assay. Patient diagnosed as a case of late onset MOGAD optic neuritis

Treated with injection methyl prednisolone 1g/day for 5 days tapered with oral steroids symptomatically improved and on follow up (Figure1-4).

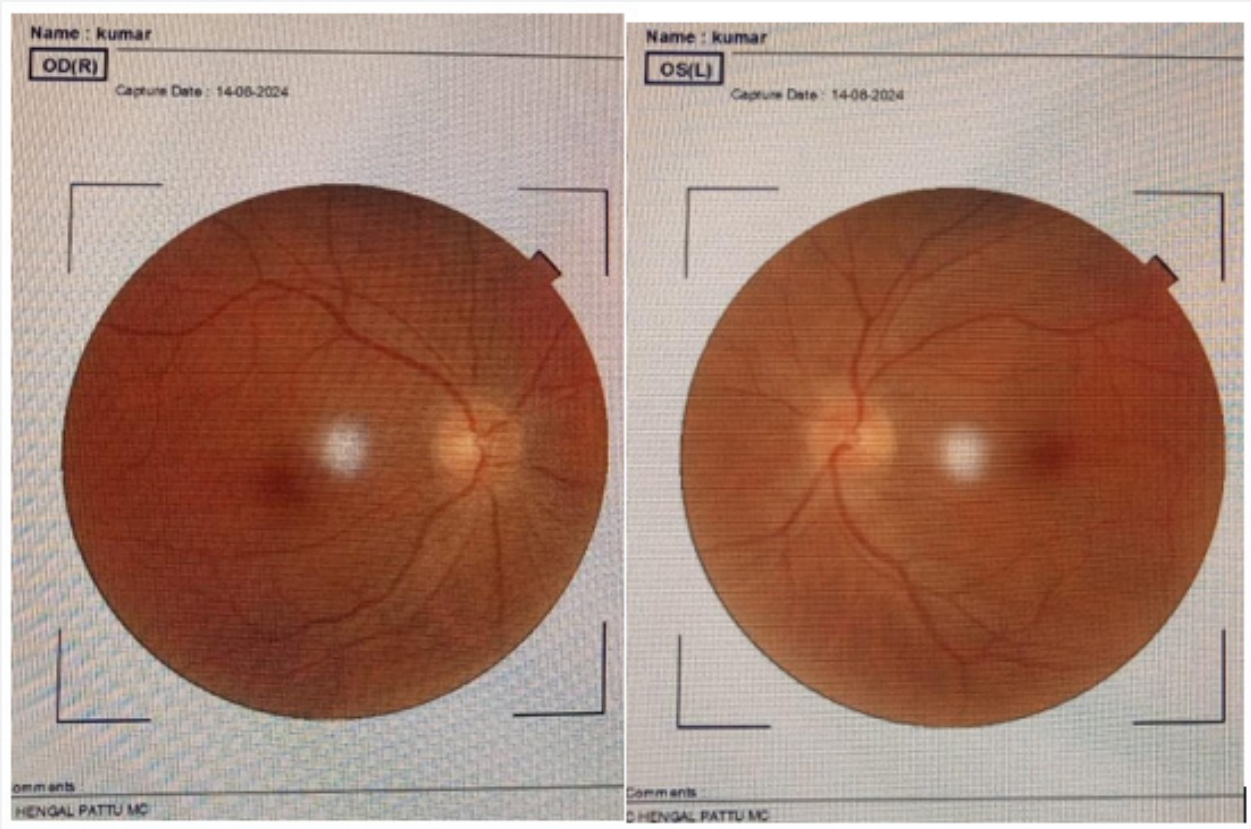

Figure 1: Fundus picture of the patient showing disc edema in both eyes.

Discussion

Patient presented in 6th decade with bilateral atypical optic neuritis with pyramidal signs, clinically suspected for NMOSD, but imaging suggestive of MOGAD-optic neuritis, serologically confirmed as MOGAD by cell-based assay. Treated with injection methyl prednisolone 15-20mg/kg/day for five days and tapered with oral steroids for eleven days. Patient visual acuity improved by two grades. MOGAD is a disease with bimodal distribution, observed in less than 45years of age, recently MOGAD optic neuritis and transverse myelitis are being reported more frequently, particularly after COVID19 on observation they also had milder and monophasic course with better outcome, as that of usual MOGAD presentation [23,24]. Features of typical and atypical optic neuritis were described in table 1 and characteristics of MOGAD, NMOSD and MS were described in table 2.

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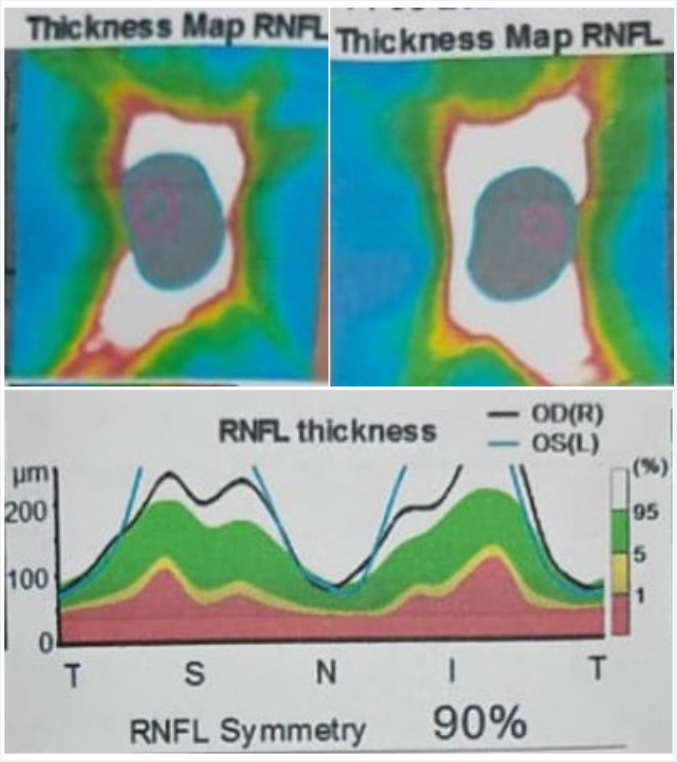

Figure 2: OCT showing bilateral retinal nerve fibre layer thickness (right<left).

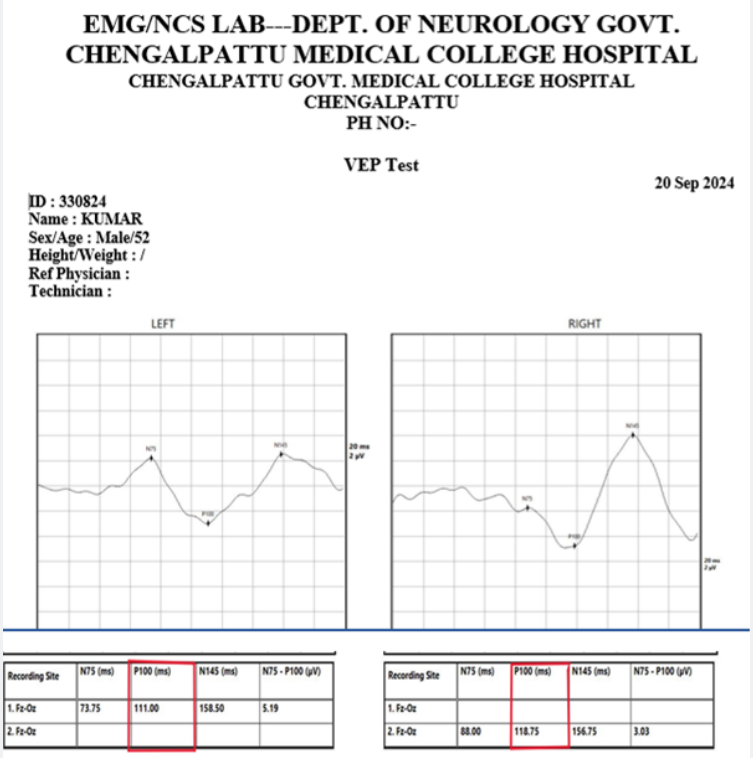

Figure 3: VEP showing prolongation of bilateral P100 latencies with mild amplitude reduction in left side.

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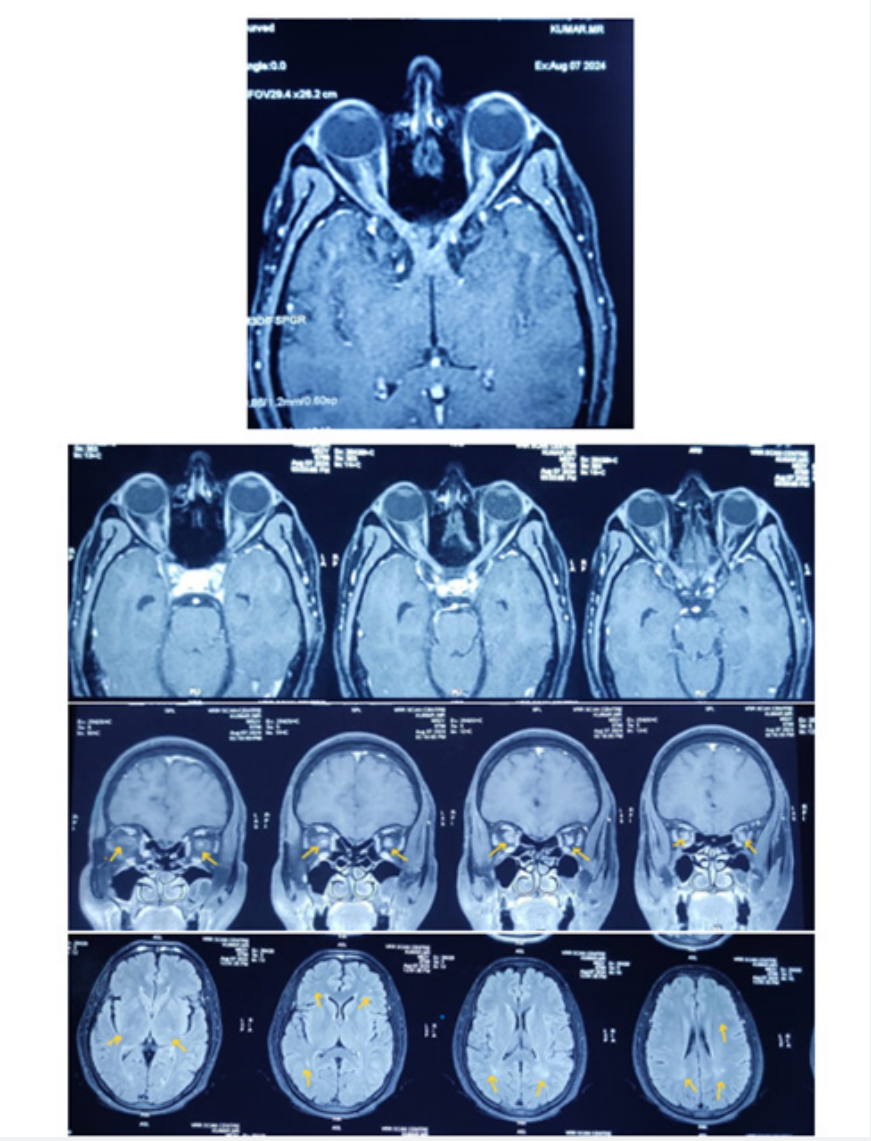

Figure 4: **MRI brain axial and coronal view – showing hyper intensities and gadolinium contrast enhancement with bilateral involvement of entire length of the optic nerve with perineural thickening and T2FLAIR hyperintensities in bilateral fronto parietal areas.**

Laboratory Diagnosis

MOG-IgG Antibody

The panel strongly recommends testing serum from patients suspected of having MOGAD using cell-based assays that use whole human MOG to detect MOG-IgG. Serum is the preferred specimen type for MOG-IgG testing. While clotting factors in plasma may influence results, studies have highlighted the low sensitivity of MOG-IgG testing in CSF alone. CSF testing for MOG IgG may be used in selected circumstances to confirm clinical and MRI findings supporting the diagnosis of MOGAD in seronegative patients with MOG-IgG [2]. ELISA is not recommended for MOG-IgG measurement owing to low sensitivity and specificity. Patients are most likely to be MOG-IgG seropositive if tested during a clinical episode. Ideally, testing should be performed before administration of corticosteroids, immunoglobulins, or apheresis, as these treatments may reduce detection of serum MOG-IgG, as observed with the AQP4 IgG assay [2].

Cerebro Spinal Fluid

CSF pleocytosis is more likely during attack than during remission and is more common in patients with ADEM or transverse myelitis than in patients with optic neuritis. CSF protein levels are elevated in 30% of patients with a first demyelinating attack and MOG-IgG, and do not distinguish MOG-IgG-associated demyelination from other neuroinflammatory disorders [2].

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Table 1.

Typical Optic Neuritis	Atypical Optic Neuritis
Most common	Rare
Unilateral or bilateral	Bilateral
Caucasian females	Males
15 to 45 years	<15years and >45 years
vision loss, dyschromatopsia, and painful eye movements	vision loss, dyschromatopsia, and painful eye movements
Normal disc	severe disc swelling, peripapillary hemorrhages, and macular exudates
Common with MS	Common with NMOSD, MOGAD
Normal or demyelination of brain parenchyma	extensive optic nerve enhancement, sheath predominant enhancement, and enhancement of the optic chiasm or optic tract
Better visual come without treatment	Poor visual outcome even with treatment

Table 2.

Characteristics	MOGAD	NMOSD	MS
Age at onset	Adult and pediatric onset	Adult onset Pediatric onset is rare	Adult onset Pediatric onset less common
F:M	1:01	7-9:1	3:01
Disease course	Monophasic or relapsing	Most often relapsing	Relapsing Secondary progressive Infrequently primary progressive
Clinically bilateral at onset	frequent	May be present	Extremely Rare
Head ache	Frequent	Rare	Rare
Visual acuity at nadir	Moderate - severely impaired	Moderate - severely impaired	Mild - moderately impaired
Fundoscopy at nadir	severe optic disc swelling is common; may have associated hemorrhages	May be present	Mild optic disc swelling may be present may have associated rarely moderate - severe
Initial recovery	Typically favorable, particularly with corticosteroids May be poor Typically favorable	May be poor	Typically, favorable
Radiological characteristics			
Radiological optic nerve head swelling	May be present	Rare	Rare
Longitudinally extensive optic nerve involvement (>½ length of optic nerve)	Frequent	Frequent	Rare typically focal optic nerve involvement
Optic nerve sheath involvement (optic perineuritis)	Frequent	Rare	Not reported
Optic chiasmal involvement	Less frequent (unless extension of contiguous longitudinally extensive optic neuritis)	May be present	Rare
Optic tract involvement	Rare	May be present	Rare
Rapid steroid responsiveness and steroid dependence	Frequent	Less frequent	Less frequent
Long term visual recovery	Favorable in absence of subsequent relapses	May be poor	Favorable

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Diagnosis of MOGAD by International MOGAD Panel Proposed Criteria [25]

Optic Neuritis

Optic neuritis is characterized by unilateral or bilateral reduced visual acuity that develops over hours to days and is often associated with retro bulbar orbital pain that is typically exacerbated with eye movement and accompanied by color vision and visual field loss. Diagnosis of optic neuritis can be supported by the presence of a T2-hyperintense signal in the optic nerve or chiasm, by enhancement of the optic nerve or chiasm with gadolinium, and by exclusion of clinical or radio graphical evidence of an alternative compressive, infiltrative, or vascular process impacting the optic nerve or retina.

MOGAD is evolving day by day, there is change in recent trend of autoimmune neuro inflammatory diseases, particularly for MOGAD after COVID pandemic. Incidence is increasing with post infectious MOGAD and even during acute infective phase of SARS COV 2 [15-17]. When comparing NMOSD and MOGAD patients in ON cases, we found that MOGAD patients had severe visual impairment during the acute phase, with some patients reaching a maximum visual functional system score (VFSS) of 6. However, during the follow-up period, most patients showed obvious improvement in VA, and no patient achieved a maximum VFSS score of 6. JARIUS's multi -center experience gave a similar result used differences in Expanded Disability Status Scale (EDSS) scores to assess recovery in MOGAD patients and showed that EDSS scores were significantly reduced in MOGAD patients compared with AQP-4 antibody-positive patients [3].

Treatment with IVIG in addition to oral corticosteroids significantly reduced relapse rates with few side effects. Rituximab, mycophenolate mofetil, and azathioprine have also shown good efficacy and tolerability and can be used as suitable alternatives. Disease-modifying therapies should be avoided, but intravenous immunoglobulin and oral corticosteroids may be appropriate first-line treatment for patients with MOGAD [18], visual outcomes at hospital discharge and at 3-month follow-up were significantly better in those patients treated with plasmapheresis [19].

Red Flags Against a Diagnosis of Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease [2]

- Progressive neurological impairment in the absence of attacks
- Rapid worsening of clinical deficits from onset to nadir within minutes to hours
- No improvement following treatment with high-dose corticosteroids for an acute attack
- MRI findings of well circumscribed T2-hyperintense lesions in a pattern meeting dissemination in space criteria for multiple sclerosis, especially when accompanied by CSF oligoclonal bands and by the accrual over time of new silent T2-hyperintense focal lesions and retention of most previous T2-hyperintense lesions
- Lesion contrast enhancement that persists for 6 months or more.

Conclusion

Although a small proportion of MOGAD patients with extensive optic nerve damage experienced severe and irreversible visual impairment, the long-term visual outcomes of MOGAD patients after 5 years of ON were generally comparable to those of MS patients and significantly better than those of AQP4-positive NMOSD patients [2,20-22]. MODAD optic neuritis is a severe neuro inflammatory disease with good visual outcome with prompt immunosuppressive therapy, atypical neuritis in atypical presentations to be suspected, diagnosed and to be treated early for the best outcome.

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