Neuroophthalmologische Präsentation der Retrobulbärneuritis im Rahmen der Myelin-Oligodendrozyten-Glykoprotein-Antikörper-assoziierten Erkrankung [Neuro-ophthalmological presentation of optic neuritis in myelin oligodendrocyte glycoprotein antibody-associated disease]

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Neuro-ophthalmological presentation of optic neuritis in myelin oligodendrocyte glycoprotein antibody-associated disease

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Abstract

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a rare demyelinating autoimmune disorder of the central nervous system. MOGAD frequently manifests with severe, bilateral, and recurrent optic neuritis (ON) episodes and is an important differential diagnosis to multiple sclerosis and aquaporin-4-IgG seropositive neuromyelitis optica spectrum disorders. The clinical manifestations of MOGAD commonly include, besides ON, transverse myelitis, acute disseminated encephalomyelitis, or brainstem encephalitis.

In this article, we summarize the current knowledge of the neuro-ophthalmological presentation of MOGAD-ON. We describe epidemiological aspects, including the association with COVID-19 and other infections or vaccinations, clinical presentation, and imaging findings of MOGAD-ON in the acute stage and during remission. Furthermore, we report findings regarding prognosis, treatment response, and changes in ON-unaffected eyes. Specifically, we touch upon findings on visual acuity, visual fields, visual evoked potentials, as well as structural changes assessed with optical coherence tomography. Moreover, we elaborate on how to differentiate MOGAD from its differential diagnoses, including other neuroinflammatory disorders (multiple sclerosis and neuromyelitis optica spectrum disorders), but also idiopathic intracranial hypertension.

1. Introduction

Cases of severe, often bilateral simultaneous and recurrent optic neuritis (ON) have often been diagnosed as chronic relapsing inflammatory optic neuropathy (CRION) [1]. In the last decade, however, many of these cases have been linked to immunoglobulin G (IgG) autoantibodies against myelin oligodendrocyte glycoprotein (MOG) [1]. Besides ON, MOG-IgG are associated with further demyelinating inflammatory autoimmune syndromes of the central nervous system (CNS), i.e. transverse myelitis (long or short), acute disseminated encephalomyelitis (ADEM), brainstem and cerebellar pathology and cortical disease with seizures [2–6]. These syndromes have recently been referred to as MOG-antibody-associated disease (MOGAD) [7].

MOGAD can have a monophasic (17-56%) or relapsing (44-83%) disease course [8]. In adults, ON is the most common clinical presentation at disease onset (55-64%) and at subsequent relapse [2], occurring bilaterally in about 50% of the cases [2,9,10]. Even though MOGAD shares similar clinical manifestations with aquaporin-4-IgG positive (AQP4-IgG$^+$) neuromyelitis optica spectrum disorders (NMOSD), it is now recognized as a distinct disease entity [11–13]. AQP4-IgG$^+$ NMOSD is classified as an autoimmune astrocytopathy, whereas MOGAD is an autoimmune oligodendrocytopathy [14]. Furthermore, MOGAD should also be considered as differential diagnosis of multiple sclerosis (MS).

Given that there is a phenotypic overlap of MOGAD with NMOSD and MS, it is helpful to be aware of details differentiating the three disease entities. Optical coherence tomography (OCT) allows for detailed measurement of the retinal neuroaxonal loss throughout the disease course in several neuroinflammatory diseases featuring retinal and optic nerve alterations [15–18]. The integrity of the retinal ganglion cell axons is measured with peripapillary retinal nerve fiber layer thickness (pRNFL) [19,20], whereas the cell bodies and dendrites of the ganglion cell are assessed with ganglion cell and inner plexiform layer thickness (GCIPL) measurement [21]. Adjacent to the GCIPL, inner nuclear layer (INL) may also be relevant to neuroaxonal damages as a marker of inflammation [22,23]. These OCT metrics are valuable imaging biomarkers to measure the extent of neuroaxonal damage in the afferent visual system.

In this review, we will describe the neuroophthalmological presentation of MOGAD-ON and summarize the current understanding of the clinical repercussions, including functional, electrophysiological and structural changes of the afferent visual system. A separate publication in this issue is dedicated to serum and cerebrospinal fluid (CSF) analysis and magnetic resonance imaging (MRI) findings in patients with MOGAD [Mewes et al.].

2. Demographic, epidemiological and clinical aspects of MOGAD-ON

MOGAD can arise in all decades of life with one peak during childhood, most frequently presenting with monophasic ADEM [24], and a second peak in adults at a median age of onset in the thirties [8]. While younger adults aged between 20 and 45 years at disease onset most often present with unilateral ON (36%), patients above 45 years of age at onset most often present with bilateral ON (39%) [2]. In all MOGAD presentation forms, a slight female predominance with females having a slightly higher risk for a relapsing course has been shown [8]. However, MOGAD with an only ON presentation show no predilection regarding sex [25].

Bilateral ON is an important clinical hallmark for MOGAD associated ON, being less frequently observed in the two most important differential diagnoses MS and AQP4-IgG^{+} NMOSD (< 5% and 8-30% respectively) [2,10]. Among ON presentations, 4 to 9% are associated with MOG-IgG in the Western world [25] and around 20% in Asian populations [26]. The overall age- and sex-specific incidence of MOG-ON is 0.2 (0 – 0.4) per 100,000 people in the Western world [25]. The clinical characteristics of MOG-ON are summarized in Table 1.

3. Acute MOGAD-ON presentation

3.1 Symptoms

A patient with typical MOGAD-ON presents during adulthood, with a simultaneous, bilateral, extensive and painful vision loss [27–29]. Retrobulbar pain presenting in up to 90% of patients with MOGAD-ON is mostly associated with eye movement [30]. Intense migraine-like headaches can precede visual loss by a few days and may be associated with an inflammatory edema that may spread to the meningeal optic nerve sheath containing nociceptive fibers of trigeminal origin [28,31].

3.2 Findings

Acute ON in MOGAD often involves the anterior optic nerve, leading to retrobulbar optic nerve swelling [32,33]. Therefore, fundus examination of patients with acute MOGAD-ON frequently reveal optic disc edema, and sometimes with hemorrhages. During the acute presentation of ON in MOGAD, visual impairment is typically severe, with high contrast Snellen visual acuity scores of 20/40 or worse [3]. The visual functional findings in MOGAD-ON are summarized in Table 2.

3.3 Differential diagnosis to idiopathic intracranial hypertension

The finding of bilateral optic disc edema in a patient complaining of headaches and blurred vision, will often lead to prompt neuroimaging to rule out a cerebral mass. With normal neuroimaging, and especially in a young, obese female, a diagnosis of idiopathic intracranial hypertension (IIH) often comes to mind [34,35]. Therefore, when treating patients presenting with either a migraine-like headache or symptoms of

raised intracranial pressure as well as visual symptoms compatible with a diagnosis of ON, MOGAD should be taken into consideration as a differential diagnosis [31,34,35]. On OCT, optic disc swelling during the acute phase of ON in MOGAD can be evidenced by thickening of the pRNFL. A recent study has investigated the potential of pRNFL, not only for diagnosing acute ON in MOGAD, but also for differentiating MOGAD from MS [36]. The study showed that during acute ON, pRNFL measurements in MOGAD are significantly higher than that in MS (164 µm vs. 103 µm). Furthermore, with a cutoff of 118 µm, a sensitivity of 74% and a specificity of 82% can be reached to distinguish acute MOGAD-ON from acute MS-ON [36].

3.4 Magnetic resonance imaging and serology

Specific radiological signs include extensive inflammation of the anterior optic nerve with perineural enhancement. The use of a cell-based assay to investigate MOG-IgG seropositivity is strongly recommended [37]. AQP4-IgG is typically negative (double-positive results are extremely rare, and should lead to critical review of the diagnosis or question the validity of the serological results). Typical CSF features comprise pleocytosis (occurring in more than 70%), elevated protein concentration in about 50% (>1g/l in 10%) and absence of evidence of oligoclonal bands [38,39]. The presence of oligoclonal bands should lead to a diagnostic review, as they are only found in less than 10% of MOGAD cases [38–41]. For details of MOGAD MRI manifestations and serology, please refer to the publication by Mewes et al. in the same issue.

4. MOGAD-ON and infections

In up to 20% of MOGAD patients, associations were found between a possible trigger and a first MOGAD event [3,8,42–45]. Temporal associations have been reported with N-methyl-d-aspartate receptor encephalitis, infections, including herpes simplex virus, Borrelia, Epstein–Barr as well as, more recently, severe acute respiratory syndrome coronavirus type 2 (SARS-sCoV-2) [8,42–47] and, albeit less frequently, with vaccinations (mostly with SARS-CoV-2 vaccination but also with diphtheria, tetanus, pertussis, polio, and influenza vaccination) [3,8,42–45]. Recent research has shown that post-vaccination ON in the presence of MOG-IgG is particularly severe, with around 50% of affected patients experiencing severe and debilitating vision loss [45]. Of note, current data suggest a favorable safety and tolerability profile of the SARS-CoV-2 vaccines among persons already diagnosed with MOGAD [48].

5. Remission and prognosis after MOGAD-ON

5.1 Structural damage

OCT-derived measures, particularly pRNFL and GCIPL, have proven to be useful imaging biomarkers to evaluate the extent of optic nerve damage in patients with MOGAD (Table 2). When looking at the temporal

dynamic changes of the retinal neurodegeneration, the retinal ganglion cell loss following an ON episode can be observed by inner retinal layer thinning, specifically pRNFL and GCIPL. The neuroaxonal damage can accumulate after each ON episode, leading to a profound thinning of both pRNFL and GCIPL [49–51]. Real world evidence from recent publications investigating pRNFL measures in patients with MOGAD are summarized in Table 3. Patients with a higher frequency of ON episode often leads to a more extensive neuroaxonal damage (Figure 1). Nevertheless, the pRNFL thinning can be obscured by the initial axonal swelling, making it difficult to properly quantify the pRNFL thinning in the first few months after ON attack. Additionally, in comparison with other etiologies of ON, MOGAD-ON might take longer time (12 months vs. 6 months) to resolve from its relatively more extensive optic disc swelling or edema [52].

Compared to AQP4-IgG$^+$ NMOSD, each ON event in MOGAD cause less damage to the retina. However, the higher ON recurrence rate in MOGAD will lead to comparable retinal neuroaxonal loss as in AQP4-IgG$^+$ NMOSD [53]. Two recent review studies have systemically summarized the OCT metrics comparison between MOGAD-ON eyes and AQP4-IgG$^+$ NMOSD ON eyes [26,54]. Both studies showed no significant difference between the two disease entities in terms of pRNFL and GCIPL thinning. Additionally, when compared to MS-associated ON eyes, both MOGAD-ON and AQP4-IgG$^+$ NMOSD ON eyes had lower pRNFL and GCIPL. Last but not least, the frequency of macular microcysts in INL, which are assumed to be an inflammatory reaction to severe neuroaxonal damage, is comparable between MOGAD-ON and AQP4-IgG$^+$ NMOSD ON eyes (both around 20% of ON eyes, in comparison to around 5% of MS-ON [53,55,56]).

5.2 Functional damage

Several studies have shown an association of neuroaxonal damage, i.e. pRNFL and GCIPL layer thinning, with visual impairment [57–59]. Moreover, retrospective studies have shown that although there is visual function recovery after an episode of ON, this recovery is not complete. Studies show that almost 50% of MOGAD patients had an incomplete visual recovery after an episode of ON [3,57,60,61]. Up to 92.3% of MOGAD patients had reduced high contrast visual acuity after an episode of ON. This has been further supported by retrospective and observational studies showing severe visual impairment in MOGAD patients after a case of ON [60]. Furthermore, a retrospective study of 32 MOGAD patients showed that ON relapses were significantly associated with poor visual outcomes [57]. Although other studies have shown different proportions [6], this can probably be explained with differences in the study design.

A systematic review and meta-analysis has shown that eyes of AQP4-IgG$^+$ NMOSD patients with history of ON have worse visual acuity outcome when compared with those of MOGAD and MS patients [26], with other studies showing similar results [62–66]. In eyes with comparable pRNFL and GCIPL thinning,

the degree of visual impairment in MOGAD patients is worse than that of MS patients but better than that of AQP4-IgG$^+$ NMOSD patients [64]. Visual acuity in MOGAD patients with a history of ON (n eyes = 11) was worse at nadir, but their recovery was better when compared with MS ON (n eyes = 22), though still worse than in healthy controls (n eyes = 33) [59].

Additionally, the visual acuity evolution after ON differs in several aspects between pediatric and adult patients. Pediatric patients show regularly a complete recovery at the last follow-up after ON, while adult patients show a high rate of visual recovery but usually a certain degree of residual visual impairment [61,67,68].

Studies have shown that visual fields are also affected in MOGAD patients with a history of ON. These patients usually presented with central scotoma [3,63], or complete visual field loss in some studies [63]. Moreover, the degree of visual field defects seems to differ between neuroimmunological diseases. For instance, MOGAD patients show a smaller mean visual field defect when compared with AQP4-IgG$^+$ NMOSD patients [69] and MS patients [59]. However, and in line with results characterizing the visual acuity after MOGAD-ON, studies showed a complete recovery of visual field defects in more than 50% of affected patients [57,60].

Only few observational studies have used visual evoked potentials (VEP) to evaluate the functional performance of the anterior visual system. VEP records the electrical impulses that are generated in response to light stimulation. The afferent visual pathway damage can be presented in forms of prolonged latency or reduced amplitude, indicating demyelination or axonal damage, respectively. A study found that VEP latencies are moderately prolonged in both pediatric and adult MOGAD patients [68]. Apart from prolonged VEP latency, also amplitude reduction in MOGAD patients has been reported [68,70].

6. Response to treatment in MOGAD-ON

Although MOGAD patients presenting with ON as the first symptom are at a higher risk for subsequent relapses, the overall long-term outcome tends to be more favorable than in patients first presenting with isolated transverse myelitis or an ADEM-like phenotype [2]. Despite severe vision loss in the acute stage of ON, MOGAD patients show good response to intravenous methylprednisolone treatment (1 g/day for 3–5 days, first line) as well as to immunoadsorption, plasma exchange, and intravenous immunoglobulins (IVIG) (second line), and patients show a favorable long-term recovery of their visual function [2,38,71]. With treatment, unilateral MOGAD-ON has a remission rate of 66% compared to 44% in patients presenting with simultaneous bilateral ON [2,38]. Also in comparison to ON in AQP4-IgG$^+$ NMOSD, MOGAD-ON has a far better recovery rate [2], similar to that of an MS associated ON [72]. While only 6–

14% of patients with MOGAD-ON expect a visual outcome of 20/200 or worse, this will be the case for over 30% of patients with AQP4-IgG$^+$ NMOSD related ON. Data on the visual recovery without acute attack treatment of MOGAD-ON are scarce and the natural history of visual outcome in untreated MOGAD-ON patients is not well-defined [9]. Of note, long steroid taper (6 months) is associated with a lower risk for relapses [72–74].

Long-term treatment is recommended for patients at risk for relapse and current therapies comprise the off-label use of prednisolone, steroid-sparing immunosuppression with azathioprine, methotrexate, mycophenolat mofetil, rituximab and IVIG [75–79]. Maintenance treatment is given either as monotherapy or as combination therapy [9,38,42]. Current data do not show any indication for a relapse-independent disease progression, but the course of symptoms including visual quality of life over time from the patients' perspective need further investigation.

7. MOGAD in absence of ON

Damage to retinal neuroaxonal integrity in eyes independent of ON in MOGAD (MOGAD-NON) are also of great clinical interest. If MOGAD-NON eyes do not feature retinal neurodegeneration in terms of structural damage, prevention of future ON attacks might be sufficient to maintain visual function. On the other hand, even if retinal neuroaxonal loss occurs in absence of ON, the clinical relevance of non-ON eyes may still be different from ON eyes.

While multiple studies have investigated ON-independent OCT-assessed retinal neuroaxonal damage, the results are controversial. Three studies have performed exploratory investigation of retinal neurodegeneration in MOGAD-NON eyes. On a cross-sectional level compared to controls eyes, MOGAD-NON eyes consistently showed inner retinal layer thinning in the macular region, while the results in pRNFL were mixed [80–82]. While two studies found pRNFL loss [80,81], particularly in the temporal quadrant, the third study revealed that the pRNFL drop in MOGAD-NON eyes was minimal when compared to disease-free controls [82]. When looking at longitudinal evidence, the latter study observed a reduction of pRNFL during follow-up, but not of GCIPL [82]. However, as the latter study included contralateral non-ON eyes of patients with unilateral ON, cross-over effects of chiasm-involving ON could not be ruled out. This could explain the fact that thinner pRNFL could not be identified at baseline as in other two studies. The pRNFL reduction during follow-up could also be attributed to the reduction of the initial swelling. Based on the above-mentioned evidence, the importance of ON prevention could be of great clinical interest if the absence of progressive retinal ganglion cell loss in eyes independent of ON in MOGAD patients can be further validated.

A multi-national and multi-center retinal imaging study recently reported longitudinal OCT results from 80 MOGAD patients [52]. No progressive GCIPL thinning was observed in MOGAD (in absence of ON during follow-up) compared to controls. Further studies investigating the longitudinal change differences between ON and non-ON eyes are warranted to better understand the clinical course of visual system damage in MOGAD.

8. Conclusion

Afferent visual pathway damage is one of the key clinical hallmarks in MOGAD. MOG-IgG testing should be considered in patients with bilateral ON, extensive vision loss, and optic disc edema [83,84], who previously might have been diagnosed with CRION [1]. Understanding the clinical presentation, temporal course, and functional and structural changes of the visual system are important in clinical practice. Various quantifiable neuro-ophthalmological modalities, including OCT and VEP, can help visualize and quantify microstructural changes of the visual system in patients with MOGAD. Given the fact that the current evidence of multimodal visual assessments in MOGAD are still quite limited and sometimes controversial, likely as a result of limited sample size, consistent conclusions from large, multicenter studies are warranted to define these neuro-ophthalmological measures as reliable biomarkers. Consolidating the utilities of these imaging biomarkers, the clinicians and researchers can gradually disentangle the mechanisms of underlying pathophysiology, monitor the disease course, improve clinical decisions, and eventually enhance the clinical outcome in patients with MOGAD.

While long-term treatment options for MOGAD are currently rare, two randomized, double-blind, placebo-controlled, multicenter phase 3 trials have recently commenced: The cosMOG study (NCT05063162) and the Meteoroid study (NCT05271409), both investigating monoclonal antibody-based treatments. Assessments of visual function and structural changes with OCT are part of the protocol of both studies, recognizing the high relevance of the visual system in MOGAD.

9. Tables

Table 1. Clinical presentation of MOGAD associated ON

Predisposing factors (in 20%)	• Infections
	  • Borrelia burgdorferi
	  • HSV
	  • SARS-CoV-2
	• NMDA-receptor encephalitis
	• Vaccinations
	  • Diphtheria, tetanus, pertussis, polio, and influenza
	  • SARS-CoV-2

Onset Age	• Around 30 years of age • Pediatric onset (mainly ADEM)
Sex	• Slight female predominance for MOGAD, but no association for MOG-IgG ON
Clinical features	• Prodromal headache • Extensive painful vision loss • +/- Bilateral • RAPD (when unilateral or bilateral and asymmetric) • Optic disc swelling
Acute treatment	• Time is vision: early treatment • Prevention of rebound ON • 1. Line: IVMP (1 g/day for 3–5 days) • 2. Line: IA; PLEX, IVIG

Long-term treatment	• For patients at risk for relapse
	• Off-label use of IST
	  • Azathioprine
	  • Methotrexate
	  • Mycophenolate mofetil
	  • Rituximab
	  • Prednisolone
	  • IVIG

Abbreviations: ADEM: acute disseminated encephalomyelitis; HSV: herpes simplex virus; IA: immunoadsorption; IST: immunosuppressive therapy; IVIG: intravenous immunoglobulins; IVMP: intravenous methylprednisolone; NMDA: N-methyl-d-aspartate; ON: optic neuritis; PLEX: plasma exchange; RAPD: relative afferent pupillary defect; SARS-CoV-2: severe acute respiratory syndrome coronavirus type 2.

Table 2. Visual functional findings in MOGAD-ON

Visual acuity	• VA impairment is common during acute ON (severe, debilitating blindness to VA ≤ 20/40). • Visual function recovery after MOGAD-ON is usually incomplete • After ON, the degree of visual impairment in MOGAD is worse than in MS but better than in AQP4-IgG$^+$ NMOSD. • Pediatric patients regularly show a complete recovery after ON, while adult patients usually have a certain degree of residual visual impairment
Visual fields	• Central scotoma is a common presentation after ON. • After ON, MOGAD patients usually have a lesser degree of visual field defect than NMOSD patients and a better recovery than MS patients. • A high proportion of MOGAD patients with visual field defects after ON will show a complete recovery.
VEP	• Both pediatric and adult cohorts commonly show a delayed latency after ON, which could stay as a residual alteration. • MOGAD patients show a significant amplitude reduction.
OCT	• In acute phase of ON, more profound optic disc edema and pRNFL thickening can be observed in MOGAD compared to MS and AQP4-IgG$^+$ NMOSD. • In MOGAD, the initial pRNFL thickening due to optic disc edema might take longer time to resolve than other etiologies of ON. • ON in MOGAD and AQP4-IgG$^+$ NMOSD will lead to comparable pRNFL and GCIPL thinning, which are more severe than ON in MS. • Non-ON eyes in MOGAD also have slightly thinner pRNFL and GCIPL compared to eyes in healthy subjects, probably due to cross-over effects of chiasm-involving ON lesions.

Abbreviations: AQP4-IgG: aquaporin-4 immunoglobulin G; GCIPL: ganglion cell and inner plexiform layer; MOGAD: myelin oligodendrocyte glycoprotein antibody associated disorders; MS: multiple sclerosis; NMOSD: neuromyelitis optica spectrum disorder; ON: optic neuritis; pRNFL: peripapillary retinal nerve fiber layer; VA: visual acuity; VEP: visual evoked potential.

Table 3. Absolute measures of pRNFL thickness in MOG-IgG seropositive patients with a history of ON.

Study	**Age (years)**	**Sample Size (Eyes with ON)**	**Bilateral ON**	**Time from onset (years)**	**pRNFL thickness (µm)**
Akaishi et al. (2016) [85]	33 (12 – 70)[a]	12 (17)	n.s.	1 (1 – 5)	- Global: 94 (73 – 147) - Superior Quadrant: 108 (78 – 152) - Inferior Quadrant: 116 (90 – 164) - Temporal Quadrant: 64 (47 – 90) - Nasal Quadrant: 71 (53 – 128)
Martinez-Lapiscina et al. (2016) [86]	54.4 53.4–58.1	4 (6)	3/4 (75%)	8.3 1.8 – 15.5[b]	- Global: 68 [48–78]
Pache et al. (2016) [53]	44.0 ± 15.2	14 (23)	12/14 (86%)	6.9 ± 6.5 1.4 (0.3 – 10.4)[b]	- Global: 59 ± 23 - Temporal Quadrant: 44 ± 21 - Nasal Quadrant: 44 ± 16
Stiebel-Kalish et al. (2017) [69]	42.5 (29.5 – 52)	6 (9)	3/6 (50%)	1.5 (1.3 – 2.4)	- Global: 75.3 ± 14.7

Havla et al. (2017) [80]	41.4 ± 14.0	13 (13)	3/13 (23%)	8.1 ± 6.7 5.0 ± 6.3[b]	- Global: 59.0 ± 20.1 - Temporal Quadrant: 41.2 ± 17.5 - Nasal Quadrant: 46.5 ± 16.2
Akaishi et al. (2017) [87]	34.1 ± 16.8	16 (16)	0/16 (0%)[c]	5.1 ± 3.5	- Global: 101.6 ± 24.8
Zhao et al. (2017) [88]	31.3 ± 15.3	49 (52)	15/49 (31%)	2.3 ± 1.6	- Global: 58.0 ± 8.7 - Superior Quadrant: 79.7 ± 8.5 - Inferior Quadrant: 81.2 ± 18.4 - Temporal Quadrant: 46.4 ± 12.2 - Nasal Quadrant: 47.2 ± 9.1
Deschamps et al. (2018) [89]	35 (16 – 57)[a]	25 (41)	10/25 (40%)	1.4 (0.3 – 15)	- Global: 58 (30 – 106)
Mekhasingharak et al. (2018) [90]	38.3 ± 14.9	6 (8)	3/6 (50%)	4.4 ± 2.7	- Global: 57 ± 13 - Superior Quadrant: 65 ± 19 - Inferior Quadrant: 64 ± 25 - Temporal Quadrant: 46 ± 10 - Nasal Quadrant: 55 ± 6
Oertel et al. (2019) [82]	40.4 ± 13.5	24 (20	n.s.	2.2 (0.4 – 14.9)	- Global: 58.3 ± 22.6
Song et al. (2019) [91]	9.7 (3 – 17)[a]	25 (24)	13/25 (52%)	1.4 ± 0.4	- Global: 76.8 ± 9.5 - Superior Quadrant: 103.3 ± 16.9 - Inferior Quadrant: 97.5 ± 17.1 - Temporal Quadrant: 46.4 ± 11.2 - Nasal Quadrant: 60.1 ± 9.5
Song et al. (2019) [92]	20.3 (3 – 61)[a]	44 (49)	13/44 (30%)	3.1 ± 3.2	- Global: 68.1 ± 13.8
Sotirchos et al. (2020) [64]	43.8 ± 13.3	16 (27)	11/16 (69%)	5.9 [2.1 – 10.4]	- Global: 60.9 ± 11.2 - Superior Quadrant: 72.8 ± 16.3 - Inferior Quadrant: 74.3 ± 18.6 - Temporal Quadrant: 39.8 ± 10.6 - Nasal Quadrant: 56.9 ± 7.4
Vicini et al. (2021) [59]	26.3 ± 11.8	6 (11)	5/6 (83%)	3.3 ± 2.8	- Global: 59.5 ± 19.6 - Temporal Quadrant: 37.5 ± 13.6 - Nasal Quadrant: 43.6 ± 17.7
Gao et al. (2021) [62]	41.1 ± 12.9	11 (16)	5/11 (45%)	3.2 ± 3.7	- Global: 73.1 ± 16.9
Oertel et al. (2022) [52]	38 ± 14[d]	43 (69)	26/43 (60%)	3 [1 – 8] 2.1 [0.9 – 7.0][b]	- Global: 64.3 ± 21.3

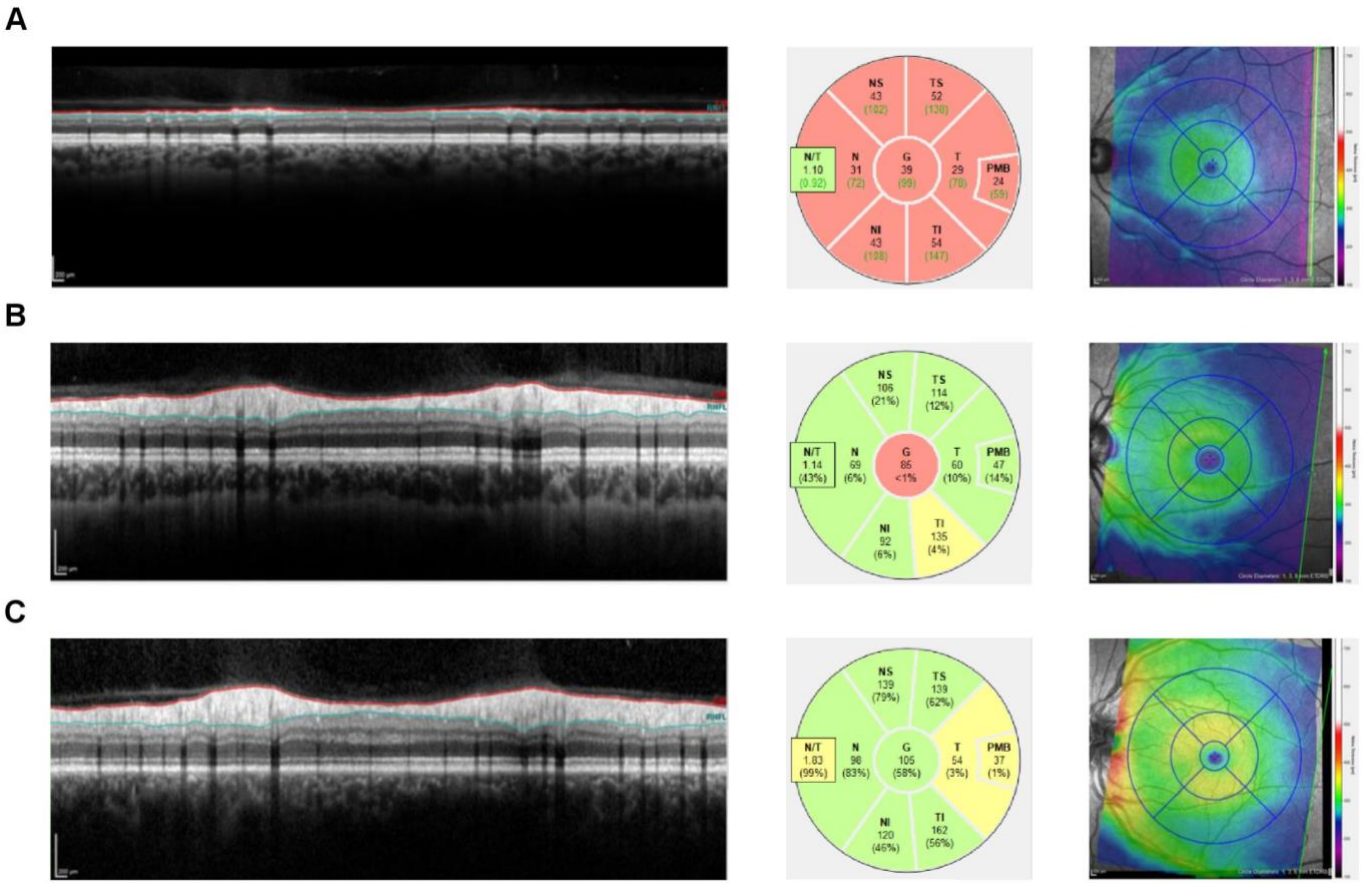
The results were presented as mean ± SD, median [IQR], or median (range).

a) age at onset, b) time since last ON attack, c) bilateral ON excluded in study design, d) includes patients without ON in both eyes.

Abbreviations: MOG: myelin oligodendrocyte glycoprotein; ON: optic neuritis; n.s.: not specified; pRNFL: peripapillary retinal nerve fiber layer.

10. Figures

Figure 1. OCT peripapillary ring scan measuring pRNFL thickness (left, middle) and macular scan measuring total macular volume around the fovea (right) in eyes of MOGAD patients with (A) multiple ON episodes, (B) single ON episode and (C) no ON episode.



Color-coded image of the pRNFL thickness (middle) compared to healthy controls from the device's normative database: green: not reduced compared to healthy cohort (>5th percentile), yellow: borderline thinned compared to healthy cohort (<5th percentile), red: severely reduced compared to healthy cohort (<1st percentile).

Abbreviations: MOGAD: myelin oligodendrocyte glycoprotein antibody associated disorders; OCT: optical coherence tomography; ON: optic neuritis; pRNFL: peripapillary retinal nerve fiber layer; G: global averaged; T: temporal; N: nasal; TS: temporal superior; NS: nasal superior; TI: temporal inferior; NI: nasal inferior; PMB: papillomacular bundle; N/T: nasal-to-temporal ratio.

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