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Optic neuritis: a comprehensive review of current therapies and emerging treatment strategies

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Background: Optic neuritis (ON) is an inflammatory condition of the optic nerve that can lead to significant visual impairment. It is often associated with multiple sclerosis (MS) but can also occur in other demyelinating diseases, such as neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody disease (MOGAD). Understanding the current therapeutic approaches and emerging treatment strategies is critical for optimizing patient outcomes.

Objective: This review provides a focused overview of current therapies for demyelinating optic neuritis associated with MS, NMOSD, and MOGAD. Less common autoimmune optic neuropathies, non-autoimmune causes (e.g., infections) and pediatric optic neuritis are not covered.

Methods: A review of the literature was conducted, including clinical trials, observational studies, and expert recommendations on the treatment and management of demyelinating ON. The efficacy, safety, and limitations of various therapeutic modalities were assessed.

Results: High-dose intravenous corticosteroids remain the mainstay of acute demyelinating ON treatment, accelerating visual recovery but not altering long-term visual outcomes. Immunomodulatory therapies, such as disease-modifying treatments for MS, play a crucial role in preventing recurrent episodes in demyelinating diseases. Emerging therapies, including re-myelination agents, neuroprotective strategies, and novel immunotherapies, show promise in improving visual prognosis and reducing long-term disability.

Conclusion: While corticosteroids remain the primary treatment for acute demyelinating ON, ongoing research into neuroprotective and re-myelinating therapies offers hope for better visual recovery and long-term management. Future studies should focus on optimizing treatment strategies and exploring novel therapeutics to enhance patient outcomes.

KEYWORDS

optic neuritis, multiple sclerosis, corticosteroids, treatment in optic neuritis, emerging therapies

1 Introduction

Optic neuritis (ON) is a demyelinating disorder of the optic nerve that can lead to sudden vision loss in one or both eyes. Optic neuritis can be caused by both infectious and non-infectious factors. The most common non-infectious cause of optic neuritis worldwide is multiple sclerosis (MS) (1), but it can also

occur in association with other demyelinating diseases, such as neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), or without an identifiable cause. The relative incidence of different causes varies according to geographic location.

The standard treatment for optic neuritis is intravenous methylprednisolone (IVMP) (2), which has been shown to hasten the recovery of vision but not impact long-term visual outcome of MS and idiopathic forms. In recent years, there has been a growing interest in the use of other treatments for optic neuritis, such as oral steroids, plasma exchange, and other immunomodulatory drugs.

This review focuses on current treatment strategies for demyelinating ON in the context of MS, NMOSD, and MOGAD. Rare autoimmune [e.g., Chronic Relapsing Inflammatory Optic Neuritis (CRION), Autoimmune glial fibrillary acid protein (GFAP), autoimmune collapsin response-mediator protein-5 (CRMP5)], non-autoimmune causes (e.g., infectious ON) and pediatric optic neuritis are beyond the scope of this article. Furthermore, we will discuss the potential future directions of research in the field of optic neuritis treatment.

2 Overview of autoimmune optic neuritis

Autoimmune ON is primarily categorized into two types: typical ON (idiopathic or MS-associated) and atypical ON. The common atypical ON cases, characterized by biomarkers such as aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG) antibodies, represents distinct CNS demyelinating diseases. Less common autoimmune optic neuropathies such as CRION, GFAP-associated astrocytopathy, and paraneoplastic CRMP5 optic neuropathy, are differentiated by clinical presentation and testing. Differentiating between typical and atypical ON is essential for understanding prognosis and treatment variations. While the Optic Neuritis Treatment Trial (ONTT) (3) remains foundational in guiding the acute treatment of typical optic neuritis, several limitations must be acknowledged in light of evolving understanding. Notably, the study was conducted prior to the identification of biomarkers for NMOSD and MOGAD. Furthermore, patients with bilateral, recurrent or severe ON phenotypes were excluded. As a result, its findings are not generalizable to NMOSD or MOGAD-associated optic neuritis. In addition, the ONTT was predominantly composed of young white females from North America, limiting its applicability to more diverse populations. The emergence of serological testing and advanced MRI biomarkers has significantly refined ON diagnostic and prognostic stratification, necessitating an updated framework for interpreting ONTT results in today's clinical practice (3).

2.1 Typical optic neuritis (MS-associated, idiopathic)

Typical ON usually presents with unilateral vision loss in young adults and tends to recover spontaneously. While most patients regain good visual acuity, MRI and CSF analysis are important for

assessing MS risk. Recent diagnostic criteria updates now allow earlier MS diagnosis in ON patients, which may influence early therapeutic decisions (4–6).

2.2 Atypical optic neuritis (NMO, MOG)

Atypical ON is often bilateral, more severe, and recurrent, with poorer visual prognosis. NMOSD-associated ON is linked with AQP4-IgG and often involves the optic chiasm (7). MOGAD-associated ON is typically seen in younger patients and presents with marked disc edema and good recovery, though relapses are not common (8, 9). Accurate diagnosis of both conditions via antibody testing and/or clinical criteria is essential, as treatment differs from typical ON (10, 11).

As our understanding of optic neuritis expands, especially with the identification of atypical forms such as NMOSD and MOGAD, accurate classification has become increasingly important for guiding management. These subtypes differ markedly in clinical presentation, diagnostic pathways, and therapeutic approaches. Due to overlapping features and evolving diagnostic criteria, clinicians may face challenges in early differentiation. To support clinical decision-making, Table 1 provides a visual summary that contrasts typical and atypical ON, highlighting key diagnostic clues and outlining distinct treatment strategies for demyelinating ON. Management of optic neuritis can be challenging due to decisions for treatment being required prior to availability of supporting evidence (MRI, NMO serology, MOG serology) to triage as to the underlying cause. In these situations clinical features can assist in preliminary triage as MS/idiopathic, NMO-SD (severe vision loss, bilateral involvement) and MOG (severe disc edema). It is particularly important to be alert for features of NMO-SD so that therapy can be escalated accordingly.

3 Treatment approaches for optic neuritis and review of clinical studies

3.1 Acute treatment

High-dose corticosteroids, either oral or intravenous (IV), are the primary treatments for acute autoimmune ON. The largest trial, ONTT, found that high dose intravenous corticosteroids (1 g/day for 3 consecutive days) expedite initial visual recovery within the first 2 weeks, by about 1–2 lines of Snellen visual acuity. Interestingly, low-dose oral prednisone (1 mg/kg) was associated with increased risk of ON relapse within the initial 2 years, discouraging its use. Recent studies have shown that high-dose oral corticosteroids may be non-inferior to high-dose intravenous steroid treatments for treating MS relapses (12). Additionally, high-dose oral methylprednisolone may be a cost-effective alternative with comparable patient satisfaction to traditional intravenous administration (13).

Notably, ONTT results may not be broadly generalizable, especially for atypical ON variants like NMOSD-ON. Acute flares of NMOSD-ON, including optic neuritis, should be treated with IVMP (1 g/day for 3–5 consecutive days with or without a PO prednisone taper). Early treatment has been shown to

TABLE 1 Simplified clinical and therapeutic approach to typical vs. atypical optic neuritis.

Category	Typical (MS-associated/idiopathic)	Atypical (NMO-SD)	Atypical (MOGAD)
Clinical features	- Young adult - Usually Unilateral - Mild-moderate vision loss - Usually no optic nerve head swelling - Short optic nerve enhancement on MRI - Good spontaneous recovery	- Adult - Unilateral or bilateral - Severe vision loss - Mild or no optic nerve head swelling - Long posterior optic nerve enhancement on MRI - Poor spontaneous recovery	- Pediatric and adult - Unilateral or bilateral - Mild-severe vision loss - Optic nerve head usually swelling - Long anterior optic nerve with perineural enhancement on MRI - Variable spontaneous recovery
Acute treatment	High-dose steroids with short taper	High-dose steroids with long taper + PLEX	High-dose steroids with long taper Add PLEX if severe or refractory
Disease diagnosis	Clinical criteria + MRI +/- CSF oligoclonal band	Serum AQP4 IgG with clinical criteria	Serum MOG IgG with clinical criteria
Long term maintenance	MS DMTs	Early immunotherapy avoid MS DMTs	Immunotherapy in patients with severe or relapsing disease MS DMTs is not effective

MS, multiple sclerosis; NMOSD, neuromyelitis optica spectrum disorder; MOGAD, myelin oligodendrocyte glycoprotein associated disease; MRI, magnetic resonance imaging; CSF, cerebrospinal fluid; IVMP, intravenous methylprednisolone; PLEX, plasma exchange; AQP4, aquaporin 4; IgG, immunoglobulin G; DMTs, disease modifying therapies.

correlate with preservation of peripapillary retinal nerve fiber layer. Retrospective studies suggest that IV corticosteroids alone might be suboptimal for visual recovery in NMOSD-ON, and that concurrent or sequential plasma exchange (PLEX) may improve outcomes. PLEX can be initiated in IVMP-refractory disease, with one series reporting average final visual acuity of 20/50 in NMOSD-ON patients receiving sequential IVMP and PLEX compared to 20/400 in those receiving IVMP alone. In two non-randomized studies of acute NMOSD (including some ON cases), 40%–50% of attacks treated with PLEX within 2 days of symptom onset experienced complete recovery, and 0%–5% recovering fully with PLEX initiation after 20 days (14, 15). Although high-quality randomized controlled trial data specifically addressing the impact of PLEX in NMOSD are currently lacking, retrospective studies suggest potential benefits from early PLEX therapy when combined with high-dose corticosteroids.

In cases of MOGAD, much like with other ON, the standard approach to acute treatment typically involves IVMP, which tends to yield rapid responses in most patients (16, 17). One retrospective study encompassing both AQP4-IgG+NMOSD and MOGAD cases found a potential benefit from initiating treatment at an earlier stage (18). For individuals experiencing severe attacks with significant disability at the peak of the attack, it is advisable to consider the early implementation of a combined therapy involving intravenous corticosteroids and plasma exchange (PLEX) (19).

Several studies have evaluated non-steroid treatments for acute optic neuritis, but none have shown a clear benefit (Table 2). Some of the failure to demonstrate benefit might relate to trial design. Future trials should prioritize earlier administration post-injury, focus on well-characterized patient subgroups with features likely to benefit from the intervention (e.g., active demyelination but limited neurodegeneration in the case of re-myelinating agents), and adopt more sensitive and specific outcome measures, such as functional imaging and electrophysiological assessments targeting affected white matter tracts. Due to varying recovery patterns in different ON subtypes (NMOSD, MOG, etc.), expert-opinion-based treatment approaches are being proposed.

3.2 Long term management

Prevention of recurrence and preservation of long-term visual function are the primary objectives of long-term treatment following acute optic neuritis. The prognosis following the initial attack, the likelihood of relapses and the appropriate treatment are contingent upon the underlying inflammatory condition (i.e., MS, NMO or MOG). When a diagnosis of MS, NMO or MOG cannot be established it is important to reassess the patient on a regular basis to determine if diagnostic criteria have been met so that appropriate treatment can be initiated.

Optic neuritis associated with NMOSD typically exhibits the poorest visual prognosis from the outset, and subsequent relapses can worsen this. In NMOSD, extended courses of oral prednisone are typically given following acute therapy [intravenous methylprednisolone (IVMP) with or without PLEX], with consideration for early initiation of immunosuppressive disease modifying therapy to reduce risk of future episodes of optic neuritis.

Similarly, MOGAD presents a less favorable visual prognosis compared to multiple sclerosis (MS) optic neuritis. After the administration of IVMP, patients are often started on an oral prednisone tapering regimen, which typically extends beyond the two-week duration recommended by the ONTT, and consideration of PLEX for severe cases with poor recovery. Disease modifying therapy initiation in MOGAD is based on relapses and persistence of serum MOG antibodies.

Multiple sclerosis (MS) treatment encompasses a comprehensive array of strategies aimed at managing the disease's complex facets. Many FDA-approved medications have demonstrated efficacy in managing MS over the long term. Disease-modifying therapy (DMT) forms the cornerstone of management of MS by reducing relapse frequency and neurological disability. These therapies have evolved over time, with newer options replacing earlier, less targeted immunosuppressive treatments. Treatment strategies vary, with escalation from low efficacy to high efficacy medications. However, a newer approach involves

TABLE 2 Research on non-steroid treatments for acute optic neuritis.

Citation	Intervention	Sample size	Study design	Result
Roed et al. (20) Denmark	IVIG	30 treatment, 34 placebo	Single-Center, randomized controlled trial	There was no effect of IVIG on long-term visual function, nor an effect in reducing latency on VEP.
Tsakiri et al. (21) Denmark	Simvastatin 80 mg	32 treatment, 32 placebo	Single-Center, randomized controlled trial	Simvastatin had a beneficial effect on VEP in both latency and amplitude, a borderline effect on the Arden score and Visual analog scale, and no effect on brain MRI or on relapse rate between the groups.
Suhs et al. (22) Germany	33,000 IU recombinant human erythropoietin intravenously daily for 3 days—add-on treatment with IVMP	20 treatment, 17 placebo	Single-center, phase 2, randomized controlled trial	Testing of visual functions at 16 weeks follow up revealed trends toward an improved outcome after erythropoietin treatment.
Raftopoulos et al. (23) UK	Phenytoin 4–6 mg/kg/day 3 months—after treating with IVMP	39 treatment, 42 placebo	Multi-center, phase 2, randomized controlled trial	At 6 months follow up, 30% reduction in the extent of RNFL loss with phenytoin compared with placebo.
Cadavid et al. (24) Australia	Opicinumab 100 mg/kg intravenous once weekly for 6 dose—after treating with IVMP	33 treatment, 36 placebo	Multi-center, phase 2, randomized controlled trial	Remyelination did not differ significantly between the opicinumab and placebo groups at week 24.
McKee et al. (25) UK	Amiloride 10 mg/day 5 months—after treating with IVMP	22 treatment, 26 placebo	Multi-center, phase 2, randomized controlled trial	Amiloride has not demonstrated any neuroprotective benefit within this trial.
Falardeau et al. (26) USA	Oral lipoic acid 1,200 mg/day 6 weeks—after treating with IVMP	15 treatment, 16 placebo	Single center, randomized controlled trial	No conclusion that lipoic acid treatment was neuroprotective in acute optic neuritis
Albert et al. (27) Germany	0.5 mg oral fingolimod daily VS subcutaneous IFN-β 1b 250 μg every other day. For 6 months - after treating with IVMP	6 Fingolimod 7 IFN-β 1b	Multi-Center, randomized controlled trial	Not enough data to conclude.

IVIG, intravenous immunoglobulin; VEP, visual evoked potential; MRI, magnetic resonance imaging; IVMP, intravenous methylprednisolone; RNFL, retinal nerve fiber layer; IFN-β 1b, interferon beta 1 B.

initiating higher-efficacy treatments at diagnosis to achieve better relapse control.

3.2.1 Multiple sclerosis DMT

MS disease modifying therapies act by modulating the immune system through diverse mechanisms, including sequestration of lymphocytes, alteration of cytokine secretion patterns, and immune cell depletion and vary by efficacy, adverse effects and route of administration (Table 3). Newly developed DMTs, including monoclonal antibodies exhibit heightened efficacy compared to traditional oral and injectable options. While infusion reactions and autoimmune side effects remain potential concerns, their overall benefits are significant.

Selection of specific MS DMT are tailored to individual patient’s needs. Although DMT treatment is typically long-term, people with stable disease while on certain DMTs may choose to de-escalate therapy. The reader is referred to the excellent review by McGinley et al. for more information (28).

3.2.2 NMOSD

Certain drugs that are approved for multiple sclerosis (MS) treatment should not be used in patients with neuromyelitis optica

TABLE 3 The table categorizes the FDA-approved disease-modifying therapies (DMTs) for multiple sclerosis (MS) by their method of administration (29).

Category	Medications
Self-injectable	Glatiramer acetate (Copaxone®, Glatopa®), Interferon beta-1a (Avonex®, Betaseron®, Extavia®, Rebif®), Peginterferon beta-1a (Plegridy®), Ofatumumab (Kesimpta®)
Oral	Cladribine (Mavenclad®), Dimethyl fumarate (Tecfidera®), Diroximel fumarate (Vumerity®), Fingolimod (Gilenya®), Tascenso ODT®), Monomethyl fumarate (Bafiertam™), Ozanimod (Zeposia®), Ponesimod (Ponvory™), Siponimod (Mayzent®), Teriflunomide (Aubagio®)
Infused	Alemtuzumab (Lemtrada®), Mitoxantrone (Novantrone®), Natalizumab (Tysabri®) Natalizumab-sztn (Tyruko®), Ocrelizumab (Ocrevus®), Ublituximab-xiiv (Briumvi™)

spectrum disorder (NMOSD) (30, 31), especially in those who are AQP4-IgG-positive. Some of these drugs, such as glatiramer acetate, are simply ineffective in preventing NMOSD attacks (32). Others, such as interferon beta (31), natalizumab (33), fingolimod, alemtuzumab (34), and dimethyl fumarate (35), have been reported to trigger severe NMOSD attacks.

Mitoxantrone may have some effect on reducing the frequency of NMOSD attacks, but it should no longer be used due to its unfavorable safety profile and the limited duration of treatment (36). Cyclophosphamide is another drug that has been tried in NMOSD, but the results have been conflicting. It is not recommended for use in NMOSD due to the limited total dose allowance and potentially severe side effects (37).

Many studies have been conducted in the last few years to develop medications to treat NMOSD. These new drugs have shown significant promise in clinical trials, reducing the frequency of relapses and improving neurological function in NMOSD patients. As a result, four new drugs have been approved in the U.S. for the treatment of NMOSD: eculizumab, inebilizumab, satralizumab, and ravulizumab-cwvz.

- Eculizumab is a monoclonal antibody that blocks the complement protein C5. It is the only drug approved for both AQP4-IgG seropositive and AQP4-IgG seronegative NMOSD (38).
- Inebilizumab is a monoclonal antibody that targets CD19-positive B cells. It is approved for the treatment of AQP4-IgG seropositive NMOSD (39).
- Satralizumab is a monoclonal antibody that targets the interleukin-6 receptor. It is approved for the treatment of AQP4-IgG seropositive NMOSD as an adjunct therapy to other immunosuppressants or as monotherapy (40, 41).
- Ravulizumab-cwvz is a long-acting C5 complement inhibitor. It is approved for the treatment of adult patients with AQP4-IgG seropositive NMOSD (42).

In addition to these new drugs, other immunosuppressants, such as azathioprine, mycophenolate mofetil, and rituximab, are also commonly used to treat NMOSD based on historical experience. However, these drugs are not FDA-approved for NMOSD. The choice of treatment for NMOSD should be individualized based on the patient's clinical presentation, serologic status, and response to previous treatments. Additional information can be found in the excellent review by Gospe et al. (43).

3.2.3 MOGAD

Long-term management of myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) focuses on preventing relapses and limiting cumulative neurological damage. The risk of relapse is influenced by several factors, including persistent seropositivity for MOG-IgG, especially when present at high titers, and a history of multiple clinical events. Conversely, pediatric patients (44), male patients presenting with spinal cord involvement and those who receive corticosteroid therapy for at least 1 month during the first attack are more likely to experience a monophasic disease course and a longer time to first relapse (45). Additionally, seroconversion to negative MOG-IgG has been associated with a reduced risk of recurrence (45). About half of people with MOGAD only have one relapse, and those who do have relapses often make a good recovery after the first one (46). Some experts advise that patients with

MOGAD who make a full recovery after the first attack do not need to take long-term immunosuppressive drugs until they are proven to have relapsing disease by having another relapse (47).

Currently, there are no FDA approved treatments for MOGAD. Disease-modifying MS medications, such as interferon- β and glatiramer acetate have been shown to be ineffective in MOGAD, while data on natalizumab are ambiguous (25). In contrast, immunosuppressants such as mycophenolate mofetil (MMF), azathioprine, intravenous immunoglobulin (IVIG), and rituximab have all been associated with reductions in the annualized relapse rate (ARR) (48, 49). A recent multi-center retrospective study found that IVIG was associated with the lowest relapse rate among a variety of immunosuppressants (50). Furthermore, a recent meta-analysis of 41 primarily retrospective observational studies demonstrated that azathioprine, MMF, rituximab, IVIG, and tocilizumab are effective in reducing relapse risk in both pediatric and adult patients with MOGAD (51). Despite these promising findings, it is important to note that, to date, no randomized controlled trials have been conducted for MOGAD, underscoring the urgent need for high-quality prospective studies to guide evidence-based treatment strategies.

4 Future directions in optic neuritis treatment

Current treatments for optic neuritis primarily aim to reduce acute inflammation and prevent long-term relapses, but they are not always effective. There is an urgent need for new and more effective treatments for optic neuritis. Some of the most promising future directions in optic neuritis treatment include:

- Neuroprotective treatments that aim to prevent damage to the optic nerve during inflammatory attacks. Promising agents include memantine, erythropoietin, interferon-beta, phenytoin, and clemastine. However, recent literature reviews have not yet demonstrated significant clinical differences, and further studies are needed in the future (52).
- Remyelination therapies that promote the repair of damaged myelin in the optic nerve. Notable remyelinating agents under clinical investigation for treating optic neuritis in multiple sclerosis (MS) include Ibudilast and Mesenchymal Stem Cells (MSC) (53).

Among remyelination-promoting therapies, opicinumab, a monoclonal antibody targeting LINGO1, a CNS protein that inhibits remyelination, was developed based on preclinical studies showing promotion of oligodendrocyte survival, axonal regeneration, and remyelination. Phase I trials confirmed its safety, leading to three Phase II studies: RENEW (optic neuritis) (24), SYNERGY (MS) (54), and AFFINITY (MS). While RENEW showed delayed improvement in visual evoked potentials, none of the trials met their primary endpoints, and development was discontinued following the failure of AFFINITY (55). Despite promising results in animal models, the human clinical translation of opicinumab has been limited by challenges such as blood-brain barrier penetration,

identification of reliable biomarkers, and determining the optimal treatment window (56). Early intervention, ideally within 25 days of symptom onset, and careful patient selection appear critical, with better responses seen in older patients with optic neuritis and younger MS patients with preserved CNS structure (24, 54, 55).

- Gene therapy is a rapidly advancing field with the potential to develop regenerative treatments for optic neuritis and other optic neuropathy. One promising approach involves delivering neuroprotective gene-encoding proteins directly into the optic nerve. This strategy is currently under preclinical investigation and may also hold therapeutic potential for other neurological disorders in the future (51). Another emerging direction is gene therapy aimed at enabling or enhancing retinal ganglion cell (RGC) regeneration and functional integration (57). While this remains in the experimental stage, such therapies could ultimately transform care for patients with irreversible vision loss. Large-scale collaborative efforts, such as the RGC Repopulation, Stem Cell Transplantation, and Optic Nerve Regeneration (RReSTORE) Consortium, are essential to overcoming current challenges and translating these advances into clinical practice (58). For more information, the author suggests an excellent review by Esposito et al. on this topic (59).

Further research is needed to elucidate the underlying pathophysiological mechanisms of MS-, NMOSD- and MOGAD-associated optic neuritis. A better understanding of these mechanisms, may facilitate the development of newer, more precise and targeted therapies aimed at preserving vision and improving quality of life for the affected individuals.

5 Conclusion

In conclusion, optic neuritis is a complex condition with various underlying causes. It presents with sudden and often painful vision loss and can be associated with demyelinating diseases like multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and myelin oligodendrocyte glycoprotein antibody disease (MOGAD). While intravenous methylprednisolone (IVMP) remains the standard treatment for acute optic neuritis of these varieties, its impact on long-term visual outcomes is limited. Therefore, exploring alternative treatments has become an area of growing interest.

Typical optic neuritis, often associated with MS, tends to have a favorable prognosis, with most patients experiencing significant visual recovery. However, monitoring and early diagnosis are crucial for identifying those at risk of developing MS. Treatment strategies for MS involve disease-modifying therapies (DMTs), which aim to reduce relapse frequency and disability.

Atypical optic neuritis, especially in the context of NMOSD and MOGAD, presents unique challenges. Differentiating between typical and atypical forms is essential, as their prognoses and

treatment approaches differ significantly. NMOSD, characterized by the presence of AQP4-IgG antibodies, benefits from PLEX in the acute setting and immunosuppressive therapies like eculizumab, inebilizumab, and satralizumab in the long term. MOGAD ON can have excellent recovery and minimal risk of relapse, but some cases have poor recovery and relapsing disease. In these more severe cases PLEX and chronic treatments like mycophenolate mofetil (MMF), azathioprine, intravenous immunoglobulin (IVIG), and rituximab show promise in reducing relapse rates.

Ongoing research in neuroprotective therapies, remyelination strategies, gene therapy and disease-specific pathophysiology holds promise for the development of targeted and effective treatments for optic neuritis.

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