ReGeo ISSN: 2177-3246

PEDIATRIC OPTIC NEURITIS CLINICAL CHARACTERISTICS AND PREDICTORS OF VISUAL RECOVERY: A STRUCTURED NARRATIVE REVIEW

NEURITE ÓPTICA PEDIÁTRICA: CARACTERÍSTICAS CLÍNICAS E PREDITORES DA RECUPERAÇÃO VISUAL - UMA REVISÃO NARRATIVA ESTRUTURADA

NEURITIS ÓPTICA PEDIÁTRICA: CARACTERÍSTICAS CLÍNICAS Y PREDICTORES DE LA RECUPERACIÓN VISUAL - UNA REVISIÓN NARRATIVA ESTRUCTURADA

https://doi.org/10.56238/revgeov17n10-227

Data de submissão: 30/09/2026 **Data de publicação:** 30/10/2026

Isabela Falco Ribeiro[1], Fernanda Pires Ribas Bueno[2], Lígia Crespo de Almeida[3], Júlia Vitória Medeiro Birtche[4], Maria Clara Guerra Peixe[5], Amanda Soares do Amaral[6], Ana Luiza Borges Abreu[7]

ABSTRACT

Background: Pediatric optic neuritis often produces severe visual loss followed by substantial recovery, but its prognosis varies with the underlying inflammatory disorder. Normal high-contrast acuity does not exclude persistent contrast, field, or structural abnormalities. Objective: To examine presenting characteristics, etiological classification, and predictors of visual recovery, distinguishing recovery of an affected eye from relapse and future demyelinating disease. Methods: A targeted PubMed-based narrative review evaluated prospective pediatric cohorts, longitudinal retrospective series, antibody-associated studies, diagnostic consensus statements, and treatment recommendations available through October 24, 2026. Adult evidence was identified explicitly when used indirectly. Results: Prospective cohorts generally demonstrate substantial improvement during the first six months, with considerable variation in myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), multiple sclerosis, and aquaporin-4-associated disease frequencies across settings. Presenting acuity, age, laterality, and disc swelling have inconsistent independent prognostic value. Etiological diagnosis, recurrent attacks, early recovery trajectory, and established optic atrophy provide more clinically useful context. MOGAD can produce striking acute loss and bilateral disc edema with favorable acuity recovery despite retinal nerve fiber layer thinning. Aquaporin-4-associated optic neuritis carries greater concern for permanent visual disability. Brain MRI abnormalities and oligoclonal bands are relevant to future multiple sclerosis but are not equivalent to direct predictors of visual recovery. Conclusions: Counseling should combine etiology, repeated functional testing, and longitudinal structural assessment. Early treatment and escalation are

[1] Institution: Uninove. E-mail: isabelafalcoribeiro@gmail.com
[2] Universidade Cidade de São Paulo - UNICID. Institution: Universidade Cidade de São Paulo (UNICID). E-mail: fernandapires3@hotmail.com
[3] Institution: Faculdade de Medicina de Campos (FMC). E-mail: ligiacrespoalmeida@gmail.com
[4] São Leopoldo Mandic Campinas,SP. Institution: São Leopoldo Mandic Campinas, SP. E-mail: jbirtche@gmail.com
[5] Institution: Faculdade de Medicina de Campos. E-mail: claragpeixe@gmail.com
[6] Institution: UNIMAR. E-mail: amanda.10amaral@hotmail.com
[7] Institution: Hospital Stella Maris. E-mail: analuizaborgesabreu@hotmail.com

important in severe attacks, while precise individual prediction remains limited by small cohorts, heterogeneous outcomes, and treatment-selection bias.

Keywords: Pediatric Optic Neuritis. Visual Prognosis. MOGAD. Aquaporin-4. Optical Coherence Tomography. Demyelination.

RESUMO

Contexto: A neurite óptica pediátrica frequentemente produz perda visual grave seguida por recuperação substancial, mas seu prognóstico varia com o transtorno inflamatório subjacente. A acuidade normal de alto contraste não exclui anomalias persistentes de contraste, campo ou estruturais. Objetivo: Examinar as características de apresentação, classificação etiológica e preditores de recuperação visual, distinguindo a recuperação de um olho afetado de recidiva e doença desmielinizante futura. Métodos: Uma revisão narrativa direcionada baseada em PubMed avaliou coortes pediátricas prospectivas, séries retrospectivas longitudinais, estudos associados a anticorpos, declarações de consenso diagnóstico e recomendações de tratamento disponíveis até 24 de outubro de 2026. Evidências em adultos foram identificadas explicitamente quando usadas indiretamente. Resultados: As coortes prospectivas geralmente demonstram melhora substancial durante os primeiros seis meses, com variação considerável na doença associada ao anticorpo da glicoproteína de oligodendrócito de mielina (MOGAD), esclerose múltipla e frequências de doença associada ao aquaporina-4 em diferentes cenários. Acuidade de apresentação, idade, lateralidade e inchaço do disco têm valor prognóstico independente inconsistente. Diagnóstico etiológico, ataques recorrentes, trajetória de recuperação precoce e atrofia óptica estabelecida fornecem contexto mais clinicamente útil. MOGAD pode produzir perda aguda notável e edema bilateral do disco com recuperação de acuidade favorável apesar do afinamento da camada de fibras nervosas da retina. A neurite óptica associada ao aquaporina-4 carrega maior preocupação com incapacidade visual permanente. Anormalidades de ressonância magnética cerebral e bandas oligoclonais são relevantes para esclerose múltipla futura, mas não são equivalentes a preditores diretos de recuperação visual. Conclusões: O aconselhamento deve combinar etiologia, testes funcionais repetidos e avaliação estrutural longitudinal. O tratamento precoce e a intensificação são importantes em ataques graves, enquanto a previsão individual precisa permanece limitada por coortes pequenas, resultados heterogêneos e viés de seleção de tratamento.

Palavras-chave: Neurite Óptica Pediátrica. Prognóstico Visual. MOGAD. Aquaporina-4. Tomografia de Coerência Óptica. Desmielinização.

RESUMEN

Antecedentes: La neuritis óptica pediátrica a menudo produce pérdida visual grave seguida de recuperación sustancial, pero su pronóstico varía con el trastorno inflamatorio subyacente. La agudeza normal de alto contraste no excluye anomalías persistentes de contraste, campo o estructurales. Objetivo: Examinar las características de presentación, clasificación etiológica y predictores de recuperación visual, distinguiendo la recuperación de un ojo afectado de la recaída y la enfermedad desmielinizante futura. Métodos: Una revisión narrativa dirigida basada en PubMed evaluó cohortes pediátricas prospectivas, series retrospectivas longitudinales, estudios asociados con anticuerpos, declaraciones de consenso diagnóstico y recomendaciones de tratamiento disponibles hasta el 24 de octubre de 2026. Se identificó explícitamente evidencia en adultos cuando se utilizó indirectamente. Resultados: Las cohortes prospectivas generalmente demuestran mejora sustancial durante los primeros seis meses, con variación considerable en la enfermedad asociada con

anticuerpos de glucoproteína de oligodendrocito de mielina (MOGAD), esclerosis múltiple y frecuencias de enfermedad asociada con acuaporina-4 en diferentes contextos. La agudeza de presentación, la edad, la lateralidad y la hinchazón del disco tienen un valor pronóstico independiente inconsistente. El diagnóstico etiológico, los ataques recurrentes, la trayectoria de recuperación temprana y la atrofia óptica establecida proporcionan un contexto más clínicamente útil. MOGAD puede producir pérdida aguda llamativa y edema bilateral del disco con recuperación de agudeza favorable a pesar del adelgazamiento de la capa de fibras nerviosas de la retina. La neuritis óptica asociada con acuaporina-4 conlleva una mayor preocupación por la discapacidad visual permanente. Las anomalías de resonancia magnética cerebral y las bandas oligoclonales son relevantes para la esclerosis múltiple futura, pero no son equivalentes a predictores directos de recuperación visual. Conclusiones: El asesoramiento debe combinar etiología, pruebas funcionales repetidas y evaluación estructural longitudinal. El tratamiento temprano y la escalada son importantes en ataques graves, mientras que la predicción individual precisa sigue siendo limitada por cohortes pequeñas, resultados heterogéneos y sesgo de selección de tratamiento.

Palabras clave: Neuritis Óptica Pediátrica. Pronóstico Visual. MOGAD. Acuaporina-4. Tomografía de Coherencia Óptica. Desmielinización.

1 INTRODUCTION

Optic neuritis is an inflammatory optic neuropathy that can present in childhood as an isolated event or as part of a broader central nervous system disorder. Acute visual loss may be profound, bilateral, and frightening for the child and family, yet many children regain useful or normal high-contrast visual acuity. This favorable overall pattern should not obscure the risk of incomplete recovery, recurrent attacks, or a diagnosis requiring long-term immunotherapy.[1,2]

Antibody testing has changed the interpretation of pediatric optic neuritis. Children previously grouped under idiopathic or recurrent inflammatory disease can now be classified as having myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) or aquaporin-4-IgG-positive neuromyelitis optica spectrum disorder (AQP4-NMOSD). These conditions differ from multiple sclerosis (MS) in clinical phenotype, relapse behavior, and treatment, and should not be combined into one prognostic category.[3,4,5]

This review addresses two related questions: what clinical characteristics identify pediatric optic neuritis and its likely cause, and which findings predict visual recovery. It separates final acuity from speed of recovery, structural injury, relapse risk, and later neurological diagnosis. This distinction is necessary because a child can recover 20/20 vision while retaining substantial optic nerve damage or remaining at risk of further attacks.

2 METHODS

Targeted PubMed searches combined optic neuritis with pediatric, paediatric, children, visual outcome, recovery, prognosis, prospective, MOG, and aquaporin-4. Supplementary searches retrieved foundational cohorts and consensus diagnostic and treatment statements. Sources available through October 24, 2026 were considered, and every reference was verified in PubMed. Prospective pediatric data were prioritized for recovery trajectories and contemporary antibody-defined studies for etiological interpretation.

This is a structured narrative review rather than a systematic review or meta-analysis. Screening was selective, without a registered protocol, duplicate independent selection, or formal risk-of-bias scoring. No new pooled estimates were calculated. Studies were compared according to age, antibody testing, treatment, follow-up, and whether outcomes were measured per patient or per eye. Adult data were not treated as direct pediatric treatment evidence.

3 CLINICAL PRESENTATION AND DIFFERENTIAL DIAGNOSIS

Typical features include acute or subacute visual loss, dyschromatopsia, reduced contrast sensitivity, a visual field defect, and sometimes pain with eye movement. Young children may not describe blur or pain reliably; they may instead stop reading, move closer to objects, hesitate on stairs, or behave as though vision is poor. A normal account of symptoms from a young child therefore does not exclude substantial impairment.[1]

The examination should document age-appropriate monocular acuity, refraction when relevant, pupils, color vision, ocular motility, the optic discs, and the retina. A relative afferent pupillary defect supports asymmetric optic nerve dysfunction but may be absent in symmetric bilateral disease. Disc edema is common in some pediatric phenotypes, particularly MOG-associated disease, while a normal-appearing disc is compatible with retrobulbar neuritis.[1,6]

Bilateral involvement is common but varies considerably across cohorts. In the initial Pediatric Optic Neuritis Prospective Outcomes Study (PON1), 16/44 children had bilateral involvement; a 2026 South Korean prospective cohort reported bilateral disease in 56.8% and disc swelling in 81.8%. Differences in age, referral, and antibody-defined case mix matter. Bilaterality should prompt careful evaluation rather than be treated as a universal marker of good or poor prognosis.[2,7]

The differential includes papilledema, pseudopapilledema, retinal disease, neuroretinitis, compressive lesions, hereditary optic neuropathy, and infectious, infiltrative, or toxic causes. Bilateral disc swelling should not automatically be labeled optic neuritis when the visual pattern suggests raised intracranial pressure. Persistent progressive loss, unusual retinal findings, a mass, systemic illness, or a course inconsistent with inflammation should trigger investigation of alternatives.[1]

3 ETIOLOGICAL CLASSIFICATION

3.1 MOG ANTIBODY ASSOCIATED DISEASE

MOGAD commonly presents with optic neuritis in children and can include marked disc edema, bilateral disease, extensive optic nerve involvement, or associated acute disseminated encephalomyelitis (ADEM). Serum MOG-IgG should be assessed using an appropriate cell-based assay and interpreted with the clinical and MRI phenotype. A positive result, particularly a low-positive result in an atypical presentation, is not sufficient to diagnose MOGAD without considering the full diagnostic criteria and alternative explanations.[3]

MOG positivity does not imply uniformly poor visual recovery. A 2026 cohort of 29 children with MOGAD-associated optic neuritis included 49 affected eyes and showed substantial acuity improvement despite marked retinal nerve fiber layer loss. However, favorable recovery after one attack does not remove the possibility of recurrent disease. Diagnostic classification is therefore necessary for both immediate visual counseling and longitudinal treatment planning.[6]

3.2 AQUAPORIN 4 ASSOCIATED DISEASE

AQP4-IgG-positive NMOSD is less common in many pediatric cohorts but carries greater concern for severe residual visual disability. Optic neuritis may occur before myelitis or other characteristic manifestations. An isolated optic nerve presentation therefore does not exclude NMOSD, and delayed etiological recognition can delay preventive treatment. Diagnosis requires the appropriate clinical syndrome, antibody interpretation, and exclusion of alternatives.[4,5]

Severe bilateral disease, chiasmal or posterior pathway involvement, poor early recovery, or accompanying spinal cord or area postrema symptoms should increase attention to NMOSD, but none replaces testing and formal diagnostic assessment. In AQP4-negative cases, more stringent clinical and MRI requirements apply. MOGAD should not be folded into seronegative NMOSD solely because the first attack is severe.[3,4]

3.3 MULTIPLE SCLEROSIS AND OTHER PRESENTATIONS

MS-associated optic neuritis is more often considered in older children and adolescents with characteristic brain lesions or cerebrospinal fluid oligoclonal bands. These findings primarily inform the risk or diagnosis of disseminated disease. Their relationship to final visual acuity is more complex and varies across cohorts. ADEM-associated neuritis requires consideration of encephalopathy and the broader acute demyelinating syndrome.[5,8]

Historical series predate contemporary MOG testing and may contain etiologically mixed groups. For example, an older cohort reported a substantial subsequent MS frequency, but its case selection and diagnostic era differ from current antibody-tested populations. Such findings remain informative for long-term follow-up while providing limited estimates of today's disease-specific risk.[9]

4 EXPECTED VISUAL RECOVERY

The prospective PON1 study enrolled 44 children aged 3–15 years across 23 North American sites. Mean high-contrast acuity improved to approximately 20/25 at six months, while low-contrast acuity remained substantially worse. The study did not identify a statistically significant association between baseline factors and the amount of high-contrast acuity improvement. Recruitment fell below its original target, limiting precision for predictor analyses.[2]

At two years, only 28 of the original 44 participants completed the study visit. Mean high-contrast acuity in that follow-up group was approximately 20/25 and was broadly stable after the initial six-month improvement. Low-contrast acuity remained impaired at a group level. Attrition and changes in diagnostic classification mean that these data should not be interpreted as a guarantee of recovery or a complete natural-history estimate for every child.[10]

Other cohorts reinforce both recovery and heterogeneity. In a retrospective study of 59 children, 36 had one-year follow-up; 81% had acuity of at least 20/20 and 89% at least 20/40. In a prospective Indian cohort of 45 children and 77 affected eyes, 64/77 eyes were reported to recover visual function over a median 35 months. These rates have different denominators and outcome definitions and should not be pooled informally.[11],[12]

A 2026 South Korean multicentre prospective study enrolled 44 children and reported improvement in mean acuity from 0.99 to 0.24 logMAR at six months. MOGAD accounted for 30/44 final diagnoses, and no significant predictors of poor six-month acuity were identified. This high MOGAD proportion is a finding from that cohort, not the global prevalence among all children with optic neuritis.[7]

Table 1

Selected pediatric optic neuritis outcome cohorts

Study	Population and follow up	Visual outcome	Interpretive limitation
PON1 at six months [2]	44 children at 23 North American sites; first optic neuritis episode.	Mean high-contrast acuity improved to approximately 20/25; low-contrast acuity remained worse.	Below-target enrollment; limited power for baseline predictors. Good chart acuity does not imply full functional recovery.
PON1 at two years [10]	28/44 original participants completed two-year visit.	Mean high-contrast acuity approximately 20/25; gains largely maintained after six months.	Attrition and selected follow-up group limit generalization; distinguish recurrent from fellow-eye episodes.
Wan et al. [11]	59 children; 36 had one-year follow-up.	At one year, 81% ≥20/20 and 89% ≥20/40. Incomplete three-month recovery associated with poorer final acuity.	Retrospective cohort and incomplete follow-up; not a validated cutoff for treatment escalation.
South Korean prospective cohort [7]	44 children; 31 referral centers; six-month outcome.	Mean acuity 0.99 to 0.24 logMAR; MOGAD 30/44. No significant poor-outcome predictor identified.	Etiological distribution is setting-specific; nonsignificance does not prove absence of prognostic effects.
Pediatric MOGAD cohort [6]	29 children and 49 affected eyes; retrospective multicentre series.	Marked acuity recovery with substantial RNFL thinning; younger onset associated with recurrence.	Eye and patient denominators differ. Relapse predictors are not identical to final-acuity predictors.

logMAR, logarithm of the minimum angle of resolution; MOGAD, myelin oligodendrocyte glycoprotein antibody-associated disease; PON1, Pediatric Optic Neuritis Prospective Outcomes Study; RNFL, retinal nerve fiber layer. Visual outcome thresholds and follow-up denominators differ across studies.
Source: Prepared by the authors.

5 PREDICTORS OF VISUAL RECOVERY

5.1 PRESENTING VISUAL ACUITY AND EARLY TRAJECTORY

Severe nadir visual loss is alarming but does not consistently predict a poor final result in children. The prospective and retrospective studies show substantial recovery from profound initial deficits, especially in selected MOGAD presentations. Baseline acuity should therefore describe acute disability and guide urgency, without being used alone to make a deterministic long-term prediction.[2,6,11]

The early recovery trajectory may be more informative. In the 59-patient retrospective cohort, acuity worse than 20/20 at three months was associated with a final outcome worse than 20/40 at one year. This is an observed association in a small follow-up sample, not a validated universal cutoff. A child with little improvement should be reassessed for persistent

inflammation, an alternative diagnosis, or established injury rather than simply waiting for a predetermined endpoint.[11]

5.2 AGE AND CLINICAL SIGNS

Age has inconsistent prognostic meaning. In a 102-child retrospective multicentre cohort, age of at least ten years, optic disc pallor, and MS were associated with poorer recovery. However, prospective PON1 analyses did not establish a comparable independent predictor of improvement. Older age can also correlate with a different etiological distribution, and small cohorts cannot reliably separate these effects.[2,8]

Laterality and acute disc swelling should not be assigned a fixed prognostic direction. Bilaterality may indicate MOGAD or another syndrome with dramatic onset and substantial acuity recovery. Disc pallor, in contrast, suggests established axonal injury and should be distinguished from acute inflammatory swelling. Neither symptom pain nor the presence of swelling proves that recovery will be complete.[6,8,11]

5.3 ETIOLOGY AND RECURRENCE

Underlying diagnosis provides the most clinically useful framework for prognosis. MOGAD-associated attacks often recover high-contrast acuity well, while AQP4-associated disease raises greater concern for permanent disability and prevention of additional attacks. Nonetheless, within each disease, outcomes vary with attack severity, treatment, recurrence, and follow-up. An antibody result is an etiological marker, not an exact prediction of final acuity.[3,4,5]

Relapse prediction is a separate outcome. In the 2026 MOGAD cohort, onset younger than eight years was associated with recurrent optic neuritis. That result does not contradict an older mixed-etiology cohort associating older age with poor acuity, because the endpoints differ. Moreover, the younger-age association requires validation before it is used to justify a fixed preventive treatment rule.[6]

Repeated attacks can accumulate structural damage even if Snellen acuity returns to a reassuring level between episodes. Long-term counseling should therefore discuss both current recovery and the possibility of a relapsing disorder. MOG seropositivity at the first episode alone does not establish that a child will relapse; maintenance decisions should reflect disease course and specialist assessment.[13]

5.4 BRAIN MRI AND CEREBROSPINAL FLUID

Abnormal brain MRI and oligoclonal bands are associated with MS in pediatric optic neuritis cohorts. They help determine the broader neurological diagnosis and follow-up plan, but should not be described automatically as independent predictors of poor acuity recovery. In PON1, associated disease and white matter lesions related to worse baseline function, while baseline factors did not clearly predict the amount of acuity improvement.[2,8]

Lumbar puncture is selected according to the differential, neurological findings, MRI, and the need to investigate inflammatory or infectious disease. It is not required to assign every uncomplicated optic neuritis presentation a visual prognosis. Testing should answer a defined question and be interpreted alongside clinical and imaging evidence rather than treated as a stand-alone prognostic panel.[1,5]

5.5 OPTICAL COHERENCE TOMOGRAPHY AND FUNCTIONAL OUTCOMES

Optical coherence tomography (OCT) can document retinal nerve fiber layer and ganglion cell loss after an attack. Acute peripapillary retinal nerve fiber layer thickening can reflect edema, so a decline from a swollen baseline does not quantify axonal loss by itself. Macular ganglion cell measures and serial scans help separate the resolution of swelling from subsequent atrophy. Device consistency, segmentation quality, and age-appropriate interpretation are essential.[6,14]

Structural and functional outcomes can diverge. In the 2026 MOGAD series, average retinal nerve fiber layer thickness declined markedly over the first six months while acuity improved. A small 2024 pediatric MOGAD study similarly reported complete acuity recovery despite reduced layer thickness. These observations support monitoring beyond high-contrast acuity rather than concluding that OCT is irrelevant when the child sees 20/20.[6,14]

Evidence regarding subclinical involvement remains unsettled. A twelve-patient prospective MOGAD study reported that OCT measurements distinguished affected children from controls, including some eyes without a recorded optic neuritis history, whereas the earlier small retrospective series did not identify comparable silent changes. Differences in design and sample size preclude a universal screening cutoff or a claim that every thin layer predicts a future attack.[14,15]

Follow-up should include contrast sensitivity or low-contrast acuity when feasible, color vision, visual fields appropriate to age, and daily visual function. Reading fatigue, dim-light difficulty, reduced endurance, and school performance can remain important despite a good

chart acuity. Low-vision or educational support should be based on functional needs rather than postponed until a numerical acuity threshold is crossed.[2,10]

6 TREATMENT AND ITS PROGNOSTIC INTERPRETATION

High-dose intravenous methylprednisolone is commonly used for clinically significant pediatric attacks, often 30 mg/kg/day to a maximum of 1 g/day for three to five days. Treatment is individualized with pediatric neurology and ophthalmology, considering severity, infection, and the likely underlying disease. An urgent severe attack should not be left untreated while waiting for antibody results when the clinical assessment supports inflammatory optic neuritis.[1,13]

Evidence from the adult Optic Neuritis Treatment Trial supports faster recovery with intravenous corticosteroids in typical adult disease, but does not establish long-term pediatric benefit across MOGAD and AQP4-NMOSD. Likewise, the adult trial's oral prednisone regimen should not be generalized to all modern high-dose oral regimens or used to define pediatric care. Pediatric practice relies on observational evidence and disease-specific consensus.[1,16]

Observational MOGAD studies suggest that treatment timing may influence recovery, but confounding by severity, diagnostic delay, and referral remains. A study associating later methylprednisolone with less complete recovery included a broader MOGAD population rather than a pediatric-only randomized comparison. A 2026 pediatric retrospective series linked longer oral steroid courses with good visual outcomes, but treatment selection prevents a causal conclusion or a universal taper duration.[17,18]

In severe attacks with inadequate response, plasma exchange or intravenous immunoglobulin may be considered according to etiology and specialist judgment. Early escalation is particularly important when severe NMOSD-associated loss is suspected; waiting for a prolonged period of nonrecovery can forfeit time. Maintenance treatment is a separate decision aimed at preventing attacks rather than accelerating recovery from an already resolved episode.[4,13]

MOGAD consensus recommendations support acute intravenous corticosteroids and selected oral tapering, with maintenance therapy considered particularly after relapse. The choice and duration of treatment should be individualized, balancing recurrent disability against treatment burden. The evidence is predominantly retrospective, and no single regimen should be presented as proven optimal for every child's first attack.[13]

Table 2

Clinical interpretation of potential visual prognostic markers

Marker	What it can indicate	What it cannot establish alone	Practical use
Severe presenting acuity loss	Acute disability and need for prompt assessment.	Irreversible final loss; many children improve markedly.	Document nadir and compare serial age-appropriate testing. [2,11]
Poor early recovery	Possible persistent inflammation, structural injury, or alternative diagnosis.	A universally validated escalation threshold.	Reassess promptly rather than waiting passively for the final follow-up visit. [11]
MOG-IgG positivity	Etiological classification in a compatible phenotype.	Guaranteed complete recovery or inevitable relapse.	Apply current diagnostic criteria and monitor disease course. [3,13]
AQP4-IgG-associated disease	Greater concern for severe residual disability and additional attacks.	The exact final acuity of an individual eye.	Urgent specialist treatment and disease-specific prevention planning. [4]
Brain lesions or oligoclonal bands	Risk or diagnosis of broader demyelinating disease, particularly MS.	A direct independent prediction of final acuity in every child.	Guide neurological classification and follow-up. [2,8]
Disc pallor or OCT thinning	Established or evolving structural injury.	Current high-contrast acuity or future relapse probability by itself.	Combine with contrast, fields, acuity, and serial quality-controlled scans. [6,8]

AQP4, aquaporin-4; IgG, immunoglobulin G; MOG, myelin oligodendrocyte glycoprotein; MS, multiple sclerosis; OCT, optical coherence tomography. This is an evidence-informed interpretation framework, not a validated prognostic score.
Source: Prepared by the authors.

7 PRACTICAL FOLLOW UP AND EVIDENCE LIMITATIONS

A baseline record should identify the affected eye or eyes, age-appropriate acuity, pupils, color function, disc appearance, visual field when possible, MRI findings, and serological status. Follow-up should assess the pace of recovery and revisit etiological classification when new neurological symptoms, recurrent loss, steroid dependence, or atypical progression occurs. Testing should remain comparable across visits to avoid confusing developmental cooperation with disease change.

The principal evidence limitations are rarity, small samples, incomplete follow-up, variable antibody testing, and confounding by treatment. Eye-level analyses must account for correlation between two eyes from the same child. Studies using 20/20, 20/40, age-normal acuity, or return to baseline describe different outcomes; apparently inconsistent recovery rates can therefore coexist without contradiction.

Future prospective studies should distinguish first-attack recovery from relapse prevention, stratify by antibody-defined disease, and measure low-contrast vision, fields, OCT, and patient-reported function. They should also validate early trajectory markers and treatment-timing associations rather than rely on retrospective thresholds. For current counseling, a range of plausible outcomes is more defensible than an exact percentage derived from a small subgroup.

8 CONCLUSION

Most children with optic neuritis experience substantial acuity recovery, but prognosis depends on etiology, recovery trajectory, recurrent activity, and established structural injury. Baseline severity, age, bilaterality, and disc edema alone have limited and inconsistent predictive value. MOGAD often shows good chart acuity despite optic atrophy, while AQP4-associated disease requires particular urgency and relapse prevention. Comprehensive longitudinal assessment is therefore essential to distinguish reassuring acuity recovery from complete restoration of visual function.

REFERENCES

1. Yeh EA, Graves JS, Benson LA, Wassmer E, Waldman A. Pediatric optic neuritis. Neurology. 2016;87(9 Suppl 2):S53-8.

2. Writing Committee for the Pediatric Eye Disease Investigator Group (PEDIG), Pineles SL, Repka MX, Liu GT, Waldman AT, Borchert MS, *et al.* Assessment of Pediatric Optic Neuritis Visual Acuity Outcomes at 6 Months. JAMA Ophthalmol. 2020;138(12):1253-1261.

3. Banwell B, Bennett JL, Marignier R, Kim HJ, Brilot F, Flanagan EP, *et al.* Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. Lancet Neurol. 2023;22(3):268-282.

4. Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, *et al.* International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015;85(2):177-89.

5. Fadda G, Armangue T, Hacohen Y, Chitnis T, Banwell B. Paediatric multiple sclerosis and antibody-associated demyelination: clinical, imaging, and biological considerations for diagnosis and care. Lancet Neurol. 2021;20(2):136-149.

6. Abdul T, Nisanova A, Chopra A, Bugsch R, Virupakshaiah A, Kothapalli NP, *et al.* Clinical Characteristics and Visual Outcomes of Optic Neuritis in Pediatric Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease. J Neuroophthalmol. 2026. doi: 10.1097/WNO.0000000000002516. Online ahead of print.

7. Joo HJ, Song JR, Lim BC, Kim WJ, Choi MY, Lee HJ, *et al.* Six-Month Visual Outcomes in Pediatric Optic Neuritis: A Multicenter Study From South Korea. J Neuroophthalmol. 2026. doi: 10.1097/WNO.0000000000002474. Online ahead of print.

8. Averseng-Peaureaux D, Mizzi M, Colineaux H, Mahieu L, Pera MC, Brassat D, *et al.* Paediatric optic neuritis: factors leading to unfavourable outcome and relapses. Br J Ophthalmol. 2018;102(6):808-813.

9. Wilejto M, Shroff M, Buncic JR, Kennedy J, Goia C, Banwell B. The clinical features, MRI findings, and outcome of optic neuritis in children. Neurology. 2006;67(2):258-62.

10. Pineles SL, Henderson RJ, Repka MX, Heidary G, Liu GT, Waldman AT, *et al.* The Pediatric Optic Neuritis Prospective Outcomes Study: Two-Year Results. Ophthalmology. 2022;129(8):856-864.

11. Wan MJ, Adebona O, Benson LA, Gorman MP, Heidary G. Visual outcomes in pediatric optic neuritis. Am J Ophthalmol. 2014;158(3):503-7.e2.

12. Nair A, Sankhyan N, Sukhija J, Saini AG, Vyas S, Suthar R, *et al.* Clinical outcomes and Anti-MOG antibodies in pediatric optic neuritis: A prospective observational study. Eur J Paediatr Neurol. 2024;49:1-5.

13. Bruijstens AL, Wendel EM, Lechner C, Bartels F, Finke C, Breu M, *et al.* E.U. paediatric MOG consortium consensus: Part 5 - Treatment of paediatric myelin oligodendrocyte glycoprotein antibody-associated disorders. Eur J Paediatr Neurol. 2020;29:41-53.

14. Gericke FC, Hanson JVM, Hackenberg A, Gerth-Kahlert C. Visual outcome measures in pediatric myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD). Eur J Paediatr Neurol. 2024;48:113-120.

15. Gericke FC, Hanson JVM, Hackenberg A, Gerth-Kahlert C. Functional and structural outcomes in paediatric myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD): a prospective study. Doc Ophthalmol. 2026;152(2):171-184.

16. Beck RW, Cleary PA, Anderson MM, Keltner JL, Shults WT, Kaufman DI, *et al.* A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. The Optic Neuritis Study Group. N Engl J Med. 1992;326(9):581-8.

17. Rode J, Pique J, Maarouf A, Ayrignac X, Bourre B, Ciron J, *et al.* Time to steroids impacts visual outcome of optic neuritis in MOGAD. J Neurol Neurosurg Psychiatry. 2023;94(4):309-313.

18. Sriram M, Shivarthi T, Anand V, Muppavarapu SC, Radhakrishnan N, Rasheed R, *et al.* Long-term visual outcomes and treatment-related prognostic variables in pediatric demyelinating optic neuritis: A 10-year retrospective cohort study. Mult Scler Relat Disord. 2026;112:107293.