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Inaugural Optic Neuritis in Morocco: A 15-Year Retrospective Study of Aetiology and Visual Prognosis

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Abstract

Introduction: Optic neuritis (ON) is a frequent cause of acute visual loss in young adults and often the first manifestation of demyelinating diseases such as multiple sclerosis (MS) or neuromyelitis optica spectrum disorder (NMOSD). Data from North Africa are limited. This study aimed to characterise inaugural ON in Morocco, with a focus on aetiology and visual prognosis.

Methods: We conducted a retrospective, descriptive study at the University Hospital of Rabat over 15 years (2008–2023). All patients presenting with acute-onset visual loss and diagnosed with ON were included. Clinical history, ophthalmological and neurological examinations, brain and spinal MRI, cerebrospinal fluid analysis, and serum antibody testing (AQP4 and MOG, when indicated) were reviewed. Patients were categorised by aetiology. Visual outcomes and disability scores were assessed over a follow-up period of up to 5 years.

Results: Seventy patients (mean age 34.4 years; 71% female) were analysed. MS was the leading cause (61.4%, 43/70), followed by NMOSD (15.7%, 11/70), clinically isolated syndrome (7.1%, 5/70), and idiopathic ON (7.1%, 5/70). Rare causes included MOGAD, Leber's neuropathy, Behçet's disease, Horton's arteritis, and infectious or toxic ON (1.4% each). Bilateral involvement occurred in 34.3% (24/70), and painful eye movements in 52.8% (37/70). Visual recovery was complete in 62.8% (44/70), though permanent blindness occurred in 45.5% (5/11) of NMOSD cases.

Discussion: Inaugural ON most often revealed MS, which generally had a favourable outcome. Atypical ON, particularly NMOSD, carried a higher risk of severe visual loss. Early aetiological diagnosis remains key to optimising outcomes. The study reflects the need for better diagnostic access and interdisciplinary collaboration in resource-limited settings.

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Introduction

ON is an inflammatory demyelinating process of the optic nerve, leading to acute visual acuity loss. It is a common cause of optic neuropathy in young adults and may present either in isolation or in association with an inflammatory demyelinating disorder of the central nervous system, such MS or NMOSD. ON is a frequent reason for consultation in neuro-ophthalmology, with a global annual incidence ranging between 0.94 and 2.18 per 100,000 inhabitants, depending on the study[1,2].

Diagnosis is purely clinical, based on the rapid progression of visual acuity decline over a few days, with or without pain upon ocular movement. Clinical examination typically reveals a relative afferent pupillary defect (RAPD). Visual field deficits (central or cecocentral scotoma, arcuate or altitudinal defects) and colour vision disturbances may also be present. Given that the inflammation in ON often affects the posterior portion of the optic nerve, it is classified as retrobulbar ON, and fundoscopy is usually normal. Less frequently, involvement of the anterior portion of the nerve manifests as papillitis[3].

ON can be classified into two forms: typical and atypical. The typical form is characterized by moderate, painful, unilateral visual impairment with a normal fundoscopy and complete or almost complete visual recovery within 10 to 15 days. In contrast, atypical ON is suggested by bilateral visual involvement, profound visual loss with no light perception in the absence of pain, initial optic atrophy and severe papilledema[4,5].

The aetiologies of ON are diverse, with central nervous system inflammatory disorders, such a MS and NMOSD, being the most common causes. Other systemic inflammatory diseases, including sarcoidosis, connective tissue disorders, and Behçet's disease, as well as infectious conditions such as syphilis and Lyme disease have also been implicated. Identifying the underlying cause of ON is crucial for initiating appropriate treatment and optimizing visual prognosis[3,4].

Optic neuritis (ON) is a common inflammatory optic neuropathy that may signal underlying demyelinating diseases such as multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD). While several studies from Europe, North America, and Asia have explored the etiological and prognostic aspects of ON, there is a lack of published data from North Africa. A 2013 abstract from Casablanca represents the only prior report from Morocco, with limited detail and no data on visual outcomes or newly recognized entities such as MOGAD. The primary aim of this study is to describe the clinical presentation and etiologies of inaugural ON and to evaluate their relationship with long-term visual prognosis in a Moroccan population. Secondary objectives include the analysis of epidemiological and paraclinical findings and the distribution of specific underlying causes, including NMOSD and multiple sclerosis, in comparison with existing literature.

Methods

Study Design

We conducted a descriptive, retrospective, monocentric study with inaugural ON, collected from the Department of Neurology A and Neuropsychology at the University Hospital of Specialties, Avicenne, Rabat, Morocco. The study was carried out over a 15-year period, from January 2008 to January 2023. The choice of a retrospective design was guided by the organisation of ON care in Morocco, where many cases, especially of infectious or toxic origin, are managed by ophthalmologists and not systematically referred to neurologists unless a neurological disease such as MS or NMOSD is suspected. This approach enabled the inclusion of a larger and more representative cohort. This study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines for reporting observational research. A completed STROBE checklist is provided as supplementary material in the Appendix.

Inclusion Criteria

Inaugural optic neuritis was defined as the first-ever clinical episode of optic neuritis in a patient (that is, the initial presentation of acute inflammation of the optic nerve leading to visual symptoms) without any prior diagnosis of an underlying neurological condition such as multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD). Using a registry of hospitalised patients, we idenfitied eligble patients. We included all patients, regardless of age, presenting with acute-onset visual acuity impairment. A thorough clinical history was taken to identify criteria supporting an ON diagnosis, with a specific focus on the patient's medical history, including previous episodes of visual or neurological impairment, a prior infectious episode, and recent vaccinations.

Clinical and Ophthalmological Evaluation

All patients underwent a comprehensive ophthalmologic examination, including visual acuity measurement, intraocular pressure assessment, fundoscopy, and visual field testing. Additionally, a complete neurological examination was performed including: assessment for other cranial nerve palsies, assessment of the patient’s tone, power, reflexes and sensation in both upper and lower limbs, and presence of other syndromes indicative of NMOSD such as: transverse myelitis, area postrema syndrome, acute diencephalic syndrome, cerebral and brainstem syndromes.

Upon confirmation of inaugural ON, an extensive etiological workup was initiated. All patients underwent brain and spinal MRI. All MRI images were interpreted by radiologists as part of routine clinical care. There was no centralised neuroradiology review. Lesion classification (e.g. typical MS or NMOSD lesions) was based on the original radiology reports, with typical NMOSD lesions defined according to established criteria such as longitudinally extensive transverse myelitis (LETM) and area postrema involvement when documented.

Lumbar puncture was performed for cytobacteriological and biochemical analysis of cerebrospinal fluid (CSF), as well as isoelectric focusing. Blood tests included complete blood count, C-reactive protein (CRP), erythrocyte sedimentation rate, antinuclear antibodies (ANA), and anti-DNA antibodies. Testing for anti-aquaporin-4 (AQP4) and anti-myelin oligodendrocyte glycoprotein (MOG) antibodies was performed using immunofluorescence techniques when clinically indicated.

Classification Criteria

Based on clinical and paraclinical findings, patients were classified into distinct ON aetiological groups: ON associated with MS, ON associated with NMOSD, ON secondary to systemic disease, infectious ON, toxic ON, and idiopathic ON. The diagnosis of MS was made according to the 2010 McDonald criteria for patients hospitalised before 2018, and according to the 2017 revised criteria for those hospitalised thereafter. NMOSD was diagnosed following the 2015 international consensus criteria, applicable to both seropositive and seronegative presentations.

Treatment and Follow-Up

All patients received appropriate treatment, including initial treatment for the acute phase, followed by maintenance therapy if needed. Follow-up was conducted over a 5-year period. In patients with MS or NMOSD, disease progression was monitored using visual acuity measurements and the Expanded Disability Status Scale (EDSS), assessed by the treating neurologist at 6 months (M6), 12 months (M12), and 18 months (M18), and compared with the baseline EDSS score at treatment initiation. For patients with ON of non-inflammatory origin, follow-up was conducted through ophthalmologic assessments, including visual acuity measurement and fundoscopy, after treatment cessation.

Data Collection and Statistical Analysis

Study data were gathered through a structured data collection form that included demographic, clinical, paraclinical, therapeutic, and follow-up information. Data analysis was performed using JAMOVI software (version 2.1.16). Quantitative variables were expressed as means or medians, depending on data distribution, while qualitative variables were presented as frequencies and percentages.

Ethical Considerations

This retrospective study was based exclusively on anonymised patient records retrieved from the archives of the Neurology Department. No direct patient contact, clinical intervention, or use of identifiable information (such as photographs or names) was involved at any stage of the study. In accordance with national regulations and institutional policy regarding non-interventional retrospective research, formal ethical approval was not required. Additionally, prior to the initiation of the study, written authorisation was obtained from the Medical Director of the hospital, allowing access to and use of the medical records for research purposes. The study was conducted in full compliance with the ethical standards outlined in the Declaration of Helsinki, particularly regarding the use of clinical data for research in the absence of patient-identifiable information.

Results

Epidemiological characteristics and etiological profile

We reviewed 78 medical records, with 8 cases excluded due to missing data. The final analysis included 70 cases of optic neuritis (ON), with a mean patient age of 34.4 years (range: 8 to 58 years). ON associated with MS was more frequent in younger individuals, with an average age of 30 years, while NMOSD presented later, with a mean age of 45 years. A female predominance was observed across all etiologies, with a female-to-male ratio of 2.5:1.

Regarding etiological distribution, MS accounted for 61.4% of cases (43 patients), including 33 relapsing forms (21 non-active and 12 active) and 10 progressive forms (3 active and 7 non-active). Clinically isolated syndrome (CIS) represented 7% of cases (5 patients), and NMOSD accounted for 15.7% (11 patients), of whom 5 were seropositive (45.45%) and 6 seronegative (54.54%, Figure 1). Other identified causes included one case each of MOG-associated disorder (1.4%), idiopathic ON (7%), toxic ON (1.4%), viral ON (1.4%), Horton's disease (giant cell arteritis, 1.4%), Behçet's disease (1.4%), and Leber's hereditary optic neuropathy (1.4%, Figure 1).

Clinical characteristics

The mean delay between symptom onset and hospital admission was 5 days, varying from 2 days for NMOSD-related ON to 12 days for Behçet-associated ON. Bilateral ocular involvement was present in 34% of patients and 52.8% reported painful eye movements. Visual acuity was above 5/10 in 32.8% of patients, between 5/10 and 1/10 in 30%, severely impaired in another 30%, and complete blindness (no light perception) was observed in 7%. Fundoscopic examination was normal in 67.6% of patients. Papilledema was found in 8.5%, and optic atrophy in 24.2%. RAPD was detected in 5 of the 24 tested patients (20.8%). When expressed as a proportion of the total cohort (n=50), this corresponds to 10% (n=5), explaining the apparent difference from Table 2, where percentages are calculated using the total sample as denominator. Neurological examination revealed no abnormalities in 45% of patients, while 27% showed signs of pyramidal tract dysfunction, such as para- or tetraparesis with spasticity, indicating possible spinal cord involvement (Table 1, 2).

Paraclinical characteristics

No missing data were reported for key variables, except for visual field testing 22.8% of cases. Optic nerve imaging was normal in 81% of the cases. Brain MRI was performed in all patients and revealed typical MS-like demyelinating lesions in 57% of cases. Contrast enhancement was noted in 10%, while MRI was normal in 32.9%. Spinal MRI was performed in 92% of patients and showed longitudinally extensive transverse myelitis in 14% of cases, spinal cord lesions under three vertebral segments in 40%, and no abnormalities in 60%. Lumbar puncture results were normal in 82.8% of patients. Lymphocytic pleocytosis was detected in 15.7% of the samples analysed (Table 3).

Treatment and outcomes

During the acute phase, nearly all patients (97.9%) received intravenous methylprednisolone at a dose of 1 gram per day for 3 to 7 days, depending on clinical severity. Long-term immunosuppressive therapy was prescribed for patients diagnosed with MS, NMOSD, or MOG-associated disorder. Among MS cases, Azathioprine was the most commonly used drug (30%), followed by Rituximab (18.9%). For NMOSD, treatments included Azathioprine and Rituximab in equal proportions (27% each), as well as Cyclophosphamide (33%). The single patient with MOGAD was treated with Rituximab.

Visual outcomes varied significantly depending on the underlying condition. Complete recovery was observed in 76% of MS cases and in all patients with CIS. In contrast, NMOSD had a poorer prognosis, with only 18% achieving full recovery and 45% experiencing permanent blindness. Among MS patients with ON, the mean Expanded Disability Status Scale (EDSS) score at 3 months was 1.75 ± 1.26, whereas in MS patients whose disease onset was unrelated to ON, the EDSS score was higher, averaging 2.82 ± 1.97. (Table 4)

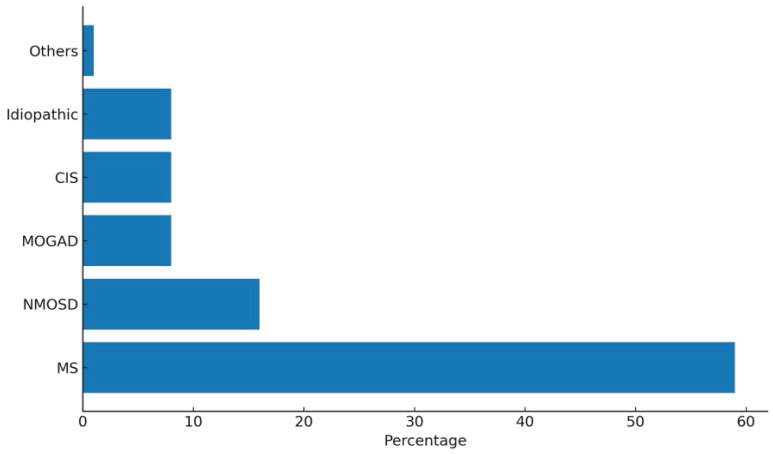

Figure 1: Distribution of Optic Neuritis aetiologies. Multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD), clinically isolated syndrome (CIS).

Table 1: Clinical features

Feature	MS (n=43)	NMOSD (n=11)	CIS (n=5)	MOGAD (n=1)	Idiopathic (n=5)	Others (n=5)	Total (n=70)
Clinical Presentation							
Mean time to consultation (days)	3.4	2.6	3	2	8.2	12	5.2
Bilateral ocular involvement	8 (18.6%)	7 (63.6%)	1 (20%)	0 (0%)	3 (60%)	5 (100%)	24 (34.3%)
Painful eye movement	27 (62.8%)	2 (18.2%)	4 (80%)	1 (100%)	2 (40%)	1 (20%)	37 (52.9%)
Neurological Examination							
Normal	18 (41.9%)	1 (9.1%)	5 (100%)	0 (0%)	5 (100%)	3 (60%)	32 (45.7%)
Spinal cord syndrome	10 (23.3%)	8 (72.7%)	0 (0%)	1 (100%)	0 (0%)	0 (0%)	19 (27.1%)
Cortical symptoms	4 (9.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (20%)	5 (7.1%)
Cerebellar syndrome	4 (9.3%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	4 (5.7%)
Area postrema syndrome	0 (0%)	1 (9.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.4%)
Cranial nerve involvement	8 (18.6%)	1 (9.1%)	0 (0%)	0 (0%)	0 (0%)	1 (20%)	10 (14.3%)

Multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD), clinically isolated syndrome (CIS).

Table 2: Ophthalmic features

Feature	MS (n=43)	NMOSD (n=11)	CIS (n=5)	MOGAD (n=1)	Idiopathic (n=5)	Others (n=5)	Total (n=70)
Visual Acuity (impairment)							
>5/10 (Good vision)	18 (41.9%)	1 (9.1%)	2 (40%)	0 (0%)	2 (40%)	0 (0%)	23 (32.9%)
1/10 to 5/10 (Moderate)	15 (34.9%)	0 (0%)	2 (40%)	1 (100%)	0 (0%)	3 (60%)	21 (30%)
<1/10 (Severe)	9 (20.9%)	7 (63.6%)	1 (20%)	0 (0%)	2 (40%)	2 (40%)	21 (30%)
Blindness	1 (2.3%)	5 (45.5%)	0 (0%)	0 (0%)	1 (20%)	0 (0%)	5 (7.1%)
Fundoscopy							
Normal	36 (83.7%)	3 (27.3%)	3 (60%)	1 (100%)	3 (60%)	1 (20%)	47 (67.1%)
Papilledema	1 (2.3%)	1 (9.1%)	2 (40%)	0 (0%)	1 (20%)	1 (20%)	6 (8.6%)
Optic atrophy	6 (14.0%)	7 (63.6%)	0 (0%)	0 (0%)	1 (20%)	3 (60%)	17 (24.3%)
Relative Afferent Pupillary Defect (RAPD)							
Present	5 (11.6%)	1 (9.1%)	1 (20%)	0 (0%)	0 (0%)	0 (0%)	7 (10%)
Absent	38 (88.4%)	10 (90.9%)	4 (80%)	1 (100%)	5 (100%)	5 (100%)	63 (90%)

Multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD), clinically isolated syndrome (CIS).

Visual field testing was not available for all patients due to limited access and lack of reimbursement and was excluded from analysis when missing. Although the study was conducted at a single tertiary centre in Rabat, which is a national referral hospital for neuroinflammatory diseases, caution is warranted regarding the generalisability of findings. Future multicentre collaborations involving neurology and ophthalmology departments across Morocco are needed to enhance data representativeness.

Discussion

The main finding of this study was that multiple sclerosis was the leading cause of inaugural optic neuritis in the Moroccan cohort, accounting for 61.4% of cases. MS-associated ON typically presented in younger adults and was associated with favourable functional outcomes, with 76% of patients achieving full visual recovery and most exhibiting an EDSS score below 2 at 3 and 6 months. In contrast, atypical forms of ON, particularly neuromyelitis optica spectrum disorder, were associated with markedly worse visual prognoses. Among NMOSD patients, only 18% recovered full visual acuity, while nearly half remained permanently blind. These findings underscore the clinical importance of prompt etiological differentiation, especially in atypical or severe cases, to enable early and targeted immunosuppressive treatment[5].

Secondary findings of interest included a pronounced female predominance (71%) and a mean age of 34.4 years, consistent with previous reports on ON epidemiology[6-9]. Bilateral involvement and painful eye movements were observed in roughly one-third and half of the cases, respectively, with these features more common in NMOSD and MOGAD. Though rare in our cohort, MOGAD presented in a 14-year-old with bilateral, painful ON, a pattern that aligns with prior descriptions of MOG-related disease in paediatric populations[13,14]. Notably, the majority of patients experienced delayed access to advanced diagnostic tools such as MOG or AQP4 antibody testing, reflecting the systemic barriers in resource-limited settings. Idiopathic ON was suspected in five patients only after a minimum three-year follow-up without recurrence or conversion to another diagnosis. Visual outcomes varied widely across aetiologies, further highlighting the necessity of robust diagnostic evaluation and follow-up protocols.

Our findings are supported by established literature. Soelberg et al. reported a similar prevalence of MS among adult ON patients, with longitudinal data showing conversion rates from clinically isolated syndrome to MS reaching 54% over 30 years[2]. In agreement with Ally and colleagues, we found that patients with MS presenting initially with ON often had milder disease and lower disability scores during follow-up[10]. ON in NMOSD, on the other hand, is widely recognized as severe and often bilateral, with poor recovery due to irreversible axonal damage[11,12]. MOGAD, although rare in our study, has been increasingly reported in children and young adults as a cause of recurrent or steroid-dependent ON with generally good recovery but frequent relapses[15-17]. Imaging features were also consistent with established disease patterns: MS lesions were short and periventricular, while NMOSD was associated with longitudinally extensive spinal lesions and posterior optic nerve involvement[18]. Paediatric ON, although uncommon in our cohort, is known to differ significantly in aetiology, being more frequently post-infectious or associated with acute disseminated encephalomyelitis [19, 20].

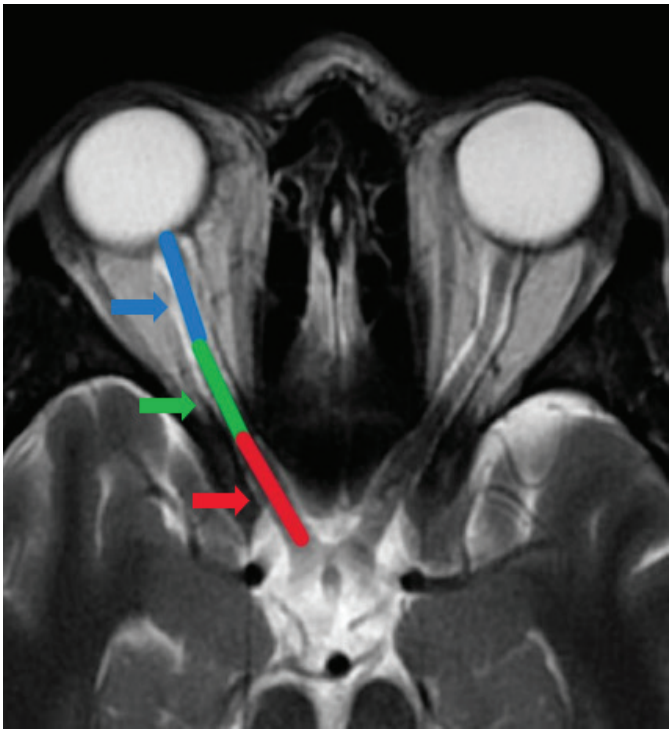

Figure 2: MRI of the optic nerve showing typical locations of lesions in: MOG-associated disorder: anterior third (blue arrow), Multiple sclerosis: middle third (Green arrow), Neuromyelitis Optica Spectrum Disorders: posterior third (Red arrow).

Table 3: Paraclinical features

Feature	MS (n=43)	NMOSD (n=11)	CIS (n=5)	MOGAD (n=1)	Idiopathic (n=5)	Others (n=5)	Total (n=70)
Brain MRI Findings							
Normal	0 (0%)	10 (90.9%)	3 (60%)	1 (100%)	4 (80%)	5 (100%)	23 (32.9%)
Typical MS lesions	40 (93%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	40 (57.1%)
Typical NMOSD lesions	0 (0%)	1 (9.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	1 (1.4%)
Non-specific lesions	1 (2.3%)	0 (0%)	2 (40%)	0 (0%)	1 (20%)	0 (0%)	4 (5.7%)
MRI enhancement	8 (18.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	8 (11.4%)
Spinal MRI Findings							
Normal	22 (51.2%)	0 (0%)	5 (100%)	1 (100%)	5 (100%)	5 (100%)	38 (54.3%)
Hypersignal <3 segments	20 (46.5%)	1 (9.1%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	21 (30%)
Hypersignal ≥3 segments	0 (0%)	10 (90.9%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	10 (14.3%)
Cervical involvement	20 (46.5%)	8 (72.7%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	26 (37.1%)
Thoracolumbar involvement	9 (20.9%)	7 (63.6%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	15 (21.4%)
MRI enhancement	3 (7%)	4 (36.4%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	6 (8.6%)
Optic Nerve Imaging							
Normal	39 (90.7%)	9 (81.8%)	5 (100%)	1 (100%)	3 (60%)	No data	57 (81.4%)
Anterior hypersignal	4 (9.3%)	0 (0%)	0 (0%)	0 (0%)	2 (40%)	No data	6 (8.6%)
Posterior hypersignal	0 (0%)	2 (18.2%)	0 (0%)	0 (0%)	0 (0%)	No data	2 (2.9%)
MRI enhancement	0 (0%)	0 (0%)	0 (0%)	0 (0%)	2 (40%)	No data	2 (2.9%)
CSF Findings							
Normal	37 (86.0%)	7 (63.6%)	3 (75%)	1 (100%)	5 (100%)	5 (100%)	58 (82.9%)
Pleocytosis	6 (14.0%)	4 (36.4%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	11 (15.7%)
Type 2 oligoclonal bands	13 (30.2%)	0 (0%)	1 (25%)	0 (0%)	0 (0%)	0 (0%)	14 (20.0%)

Multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), or MOG antibody-associated disease (MOGAD), clinically isolated syndrome (CIS).

Table 4: Treatment and outcome

Treatment and Outcome	n (%)
Steroid Treatment	
IV methylprednisolone 1 g/day for 3-5 days	68 (97.9%)
Oral corticosteroids only	4 (5.7%)
No corticosteroids	6 (8.6%)
Plasma Exchange	4
Immunosuppressants Initiated After ON	24
Disease-modifying therapy for MS	17 (24.3%)
Rituximab (for NMOSD)	5 (7.1%)
Azathioprine	2 (2.9%)
Visual Recovery	
Full recovery	41 (58.6%)
Partial improvement	18 (25.7%)
No improvement / blindness	6 (8.6%)
Not documented	5 (7.1%)

This study has several strengths. It is the first large-scale retrospective study of inaugural ON in Morocco spanning 15 years, with systematic data collection and long-term follow-up. Detailed clinical, paraclinical, and radiological data allowed for robust etiological classification and outcome analysis. However, limitations must be acknowledged. The retrospective design is vulnerable to information bias and incomplete documentation, especially for earlier cases. The single-centre setting may limit the generalizability of findings, as our hospital serves as a national referral centre and may not reflect community-level presentations. The relatively small number of paediatric patients reflects our adult neurology focus and limits conclusions about childhood ON. Furthermore, follow-up duration, though up to five years, may still be insufficient to fully exclude future diagnoses in patients currently classified as idiopathic.

Future research should focus on prospective, multicentre studies involving both neurology and ophthalmology departments across Morocco to enhance representativeness. Broader access to advanced serological testing for AQP4 and MOG antibodies is urgently needed, particularly in public-sector hospitals. Development of standardized diagnostic and treatment pathways tailored to local realities would also be beneficial. Lastly, given the significant variation in outcomes based on aetiology, interdisciplinary management and early referral protocols should be reinforced to optimize patient care and reduce the risk of irreversible vision loss.

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