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Multiple Sclerosis and Related Demyelinating Disorders in a Tropical Setting: Phenotype Distribution, Disease Trajectory and Predictors of Disability Accumulation

Dr. Akshay Chugh[*1], Dr. Arun Kumar[*2], Dolly[*3]

[1] *Assistant Professor, Department of Gen. Medicine, Saraswathi Institute of Medical Sciences, Hapur*
[2]*Professor, Department of Pharmaceutics, Saraswathi College of Pharmacy, Hapur*
[3]*Lecturer, Department of Medical Surgical Nursing (MSN), Saraswathi College of Nursing, Hapur*

Abstract — Multiple sclerosis has been historically considered uncommon in tropical and South Asian populations, with neuromyelitis optica spectrum disorder (NMOSD) accounting for a relatively larger fraction of inflammatory demyelinating presentations than in Western cohorts. We retrospectively reviewed 242 patients with demyelinating disorders followed at a tertiary neurology service over 10 years, characterising phenotype distribution, disease trajectory, treatment patterns, and predictors of disability accumulation. Relapsing-remitting MS accounted for 58.7% of the cohort, NMOSD 15.7%, secondary progressive MS 9.9%, primary progressive MS 7.4%, MOG antibody-associated disease 5.8%, and clinically isolated syndrome 2.5%. Cumulative probability of reaching EDSS 6 (cane required) at 10 years varied substantially by phenotype: 16% in RRMS, 30% in NMOSD, 50% in SPMS, and 60% in PPMS. Disease-modifying therapy was initiated in 82.4% of MS patients overall, with high-efficacy DMTs increasingly used as first-line in recent years. Strongest independent predictors of disability accumulation included PPMS phenotype, NMOSD, age at onset ≥40, spinal cord involvement at presentation, high baseline MRI lesion load, annualised relapse rate ≥2 in the first two years, and delayed DMT initiation. The findings inform diagnostic priorities, treatment escalation thresholds, and prognostic counselling for this population.

Keywords *— Multiple Sclerosis; Neuromyelitis Optica; NMOSD; MOG Antibody Disease; Tropical Neurology; Disease-Modifying Therapy; Disability Progression.*

1. Introduction

Multiple sclerosis has long been considered a disease of temperate climates, with epidemiological studies dating to the mid-twentieth century showing a clear latitude gradient: prevalence rising with distance from the equator and particularly elevated in northern European, North American, and Australasian populations. Tropical and South Asian populations have historically shown lower MS prevalence estimates of 5-15 per 100,000 versus 100-200 per 100,000 in high-prevalence Western regions although whether this reflects true biological difference, ascertainment differences, or a combination has been debated for decades. Several developments have complicated the simple temperate-tropical contrast. First, MS prevalence in South Asian populations has risen substantially over the past two decades, possibly reflecting better case ascertainment but also genuine biological shifts driven by vitamin D status, infection patterns, diet, and other environmental factors. Second, NMO spectrum disorder once considered a severe MS variant is now recognised as a distinct disease entity with characteristic aquaporin-4 antibody pathophysiology, and accounts for a substantially larger fraction of demyelinating presentations in Asian populations than in Western series. Third, MOG antibody-associated disease, a more recently characterised entity, contributes further to the demyelinating spectrum (Jha, Kumar, & Neha, 2026; Kumar, Gautam, & Maitiy, 2026; Yatish, Khatoon, & Kumar, 2026). We undertook a retrospective cohort study at our tertiary neurology service to characterise the demyelinating disorder phenotype distribution, disease trajectory over a 10-year follow-up window, treatment patterns including the introduction of high-efficacy disease-modifying therapies (DMTs), and predictors of disability accumulation. The aim was both descriptive and operational, to inform diagnostic and management priorities for this population (Bhatnagar, Kumar, & Shivam, 2026; Kumar, Sharma, & Gupta, 2026).

2. Methods

We conducted a retrospective cohort review at the tertiary neurology service of an academic medical centre. Patients with confirmed demyelinating disorders managed between January 2014 and December 2023 were eligible. Inclusion required (a) confirmed diagnosis of MS by 2017 McDonald criteria, NMOSD by 2015 International Panel criteria, MOG antibody-associated disease by 2023 international criteria, or clinically isolated syndrome with subsequent follow-up; (b) at least 24 months of follow-up data; and (c) availability of baseline MRI and at least one follow-up MRI. Exclusion criteria included alternative diagnoses on subsequent review and patients lost to follow-up before 24 months. The final cohort comprised 242 patients. Phenotypes were assigned per consensus criteria.

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MS was subclassified as relapsing-remitting (RRMS), primary progressive (PPMS), or secondary progressive (SPMS) based on clinical course. NMOSD diagnosis required aquaporin-4 antibody positivity or, for the small seronegative fraction, fulfilment of clinical and imaging criteria. MOG antibody disease required positive MOG-IgG using validated cell-based assay alongside compatible clinical syndrome. Clinically isolated syndrome was reserved for patients with a single demyelinating episode not fulfilling MS dissemination criteria during follow-up. Primary outcome was disability accumulation, defined as increase of ≥1 EDSS point (≥1.5 points if baseline EDSS <2.5) sustained over at least 6 months, and time to reaching EDSS 6 (defined by intermittent or constant aid required to walk 100 metres). Secondary outcomes included annualised relapse rate, MRI lesion accumulation, DMT use and persistence, optic-spinal disease predominance, employment status, and quality of life on the MSQoL-54 instrument. Outcomes were ascertained from structured clinical records and supplemented by patient interview at study census date. Time-to-event outcomes were analysed by Kaplan-Meier estimation with log-rank testing for phenotype comparisons. Annualised relapse rates were compared using negative binomial regression. Multivariable Cox regression identified independent predictors of disability accumulation with clinically prespecified covariates. Sensitivity analyses examined effect heterogeneity by treatment era (pre-2018 vs from 2018, reflecting broader access to high-efficacy DMTs).

3. Results

3.1 Cohort and Phenotype Distribution

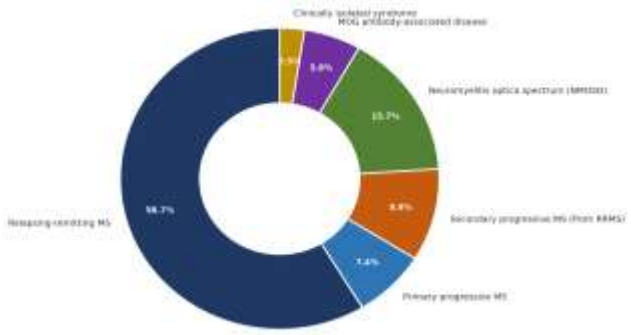

Fig.1: Demyelinating disorder phenotype distribution across the cohort

Phenotype distribution is shown in Figure 1. Relapsing-remitting MS accounted for the majority of the cohort (58.7%), but the relatively high contribution of NMOSD (15.7%) and MOG antibody disease (5.8%) is characteristically Asian. Together, the NMO-spectrum and MOG-AD presentations accounted for 21.5% of cases approximately double the fraction typically reported from Western cohorts (where these conditions typically account for 3-10% of demyelinating presentations). Primary progressive MS accounted for 7.4% of the cohort, broadly consistent with Western experience, while secondary progressive MS converted from RRMS during follow-up in an additional 9.9%.

Table 1. Baseline characteristics by phenotype

Characteristic	RRMS (n=142)	NMOSD (n=38)	Progressive MS (n=42)	MOGAD (n=14)
Age at first symptom, mean (SD), years	30.4 (8.8)	38.6 (12.4)	42.4 (10.2)	32.6 (14.2)
Female sex, n (%)	98 (69.0)	32 (84.2)	26 (61.9)	8 (57.1)
Onset with optic neuritis, n (%)	42 (29.6)	18 (47.4)	6 (14.3)	8 (57.1)
Onset with myelitis, n (%)	32 (22.5)	18 (47.4)	8 (19.0)	2 (14.3)
Onset with cerebellar/brainstem, n (%)	32 (22.5)	2 (5.3)	12 (28.6)	2 (14.3)
Onset with cerebral hemispheric, n (%)	36 (25.4)	-	16 (38.1)	2 (14.3)
Baseline EDSS, median (IQR)	1.5 (1-2.5)	3.0 (2-4.5)	4.0 (3-5.5)	2.0 (1-3)
MRI brain lesions ≥9 at baseline, n (%)	112 (78.9)	8 (21.1)	32 (76.2)	6 (42.9)
Spinal cord lesion at baseline, n (%)	62 (43.7)	32 (84.2)	22 (52.4)	6 (42.9)
Longitudinally extensive cord lesion, n (%)	8 (5.6)	26 (68.4)	2 (4.8)	2 (14.3)
AQP4-IgG positive, n (%)	-	32 (84.2)	-	-
MOG-IgG positive, n (%)	-	-	-	14 (100.0)
Time from onset to diagnosis, median, mo	9	6	18	8

3.2 Disability Trajectory

Time to reaching EDSS 6 (intermittent or constant aid required for walking) over 10 years differed substantially by phenotype (Figure 2). RRMS showed the most favourable trajectory, with 84% remaining ambulatory without aid at 10 years. NMOSD showed an intermediate trajectory with approximately 30% reaching EDSS 6 by 10 years, driven by incomplete recovery from severe relapses involving optic nerves and spinal cord. Progressive MS phenotypes (both primary and secondary) showed the steepest disability trajectories, with 50-60% reaching EDSS 6 by 10 years.

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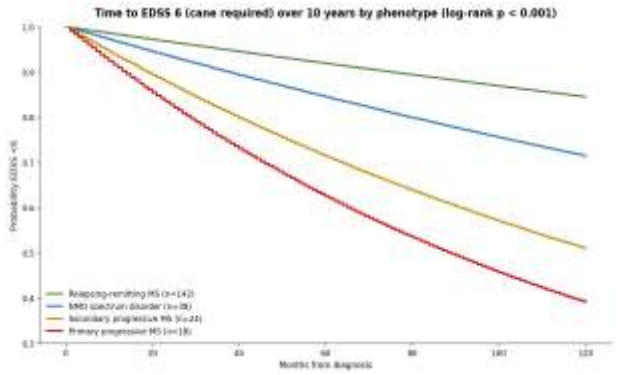

Fig.2: Time to EDSS 6 over 10 years by phenotype

3.3 Relapse Frequency

Annualised relapse rates during follow-up are shown in Figure 3. NMOSD showed the highest mean relapse rate during follow-up, with the median patient experiencing approximately 1.2 relapses per year despite treatment in many cases. RRMS showed lower relapse rates (median approximately 0.6 per year on treatment, compared with historical estimates of 1.1-1.4 per year in untreated MS). Progressive MS phenotypes showed lower relapse rates as expected given their non-relapsing course, though intermittent relapses still occurred in some SPMS patients particularly early in their progressive phase. MOGAD showed an intermediate pattern with frequent relapses early followed by lower rates.

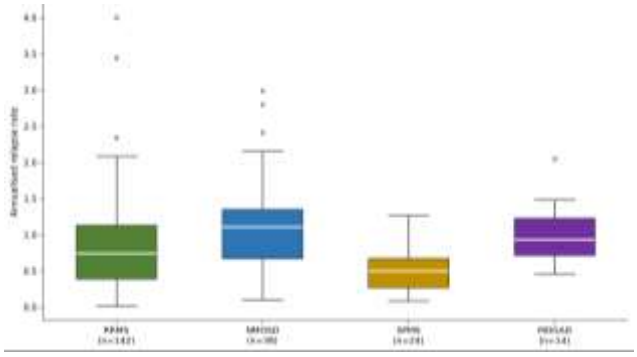

Fig.3: Annualised relapse rate during follow-up by phenotype

Table 2. Disease activity and disability outcomes by phenotype

Outcome	**RRMS (n=142)**	**NMOSD (n=38)**	**Progressive MS (n=42)**	**MOGAD (n=14)**
Mean follow-up, years	5.8	6.2	5.4	4.6
Mean annualised relapse rate	0.6	1.2	0.4	0.8
Annualised relapse rate <0.5, n (%)	82 (57.7)	6 (15.8)	32 (76.2)	6 (42.9)
Relapse-free for ≥3 years, n (%)	68 (47.9)	8 (21.1)	32 (76.2)	6 (42.9)
EDSS at last follow-up, median (IQR)	2.0 (1-3.5)	4.0 (2.5-5.5)	5.5 (4-6.5)	2.5 (1.5-4)
Reached EDSS 4 during follow-up, n (%)	32 (22.5)	18 (47.4)	32 (76.2)	4 (28.6)
Reached EDSS 6 during follow-up, n (%)	12 (8.5)	10 (26.3)	20 (47.6)	2 (14.3)
Sustained EDSS progression ≥1 point, n (%)	48 (33.8)	20 (52.6)	28 (66.7)	4 (28.6)
MRI new T2 lesions, mean total	8	12	6	4
MRI gadolinium-enhancing lesions, mean	2	4	1	2
Employment maintained at last visit, n (%)	118 (83.1)	22 (57.9)	18 (42.9)	12 (85.7)
MSQoL-54 physical score, mean	68	52	42	68

3.4 Disease-Modifying Therapy

Table 3. Disease-modifying therapy patterns by phenotype

DMT class / agent	**RRMS (n=142)**	**NMOSD (n=38)**	**Progressive MS (n=42)**	**MOGAD (n=14)**
Any DMT initiated, n (%)	138 (97.2)	36 (94.7)	18 (42.9)	12 (85.7)
Platform first-line: interferon, n (%)	32 (22.5)	-	-	-
Platform first-line: glatiramer, n (%)	8 (5.6)	-	-	-
Platform first-line: teriflunomide, n (%)	24 (16.9)	-	-	-
Oral high-efficacy: dimethyl fumarate, n (%)	42 (29.6)	-	-	-
Oral high-efficacy: cladribine, n (%)	6 (4.2)	-	-	-
Infusion high-efficacy: natalizumab, n (%)	12 (8.5)	-	-	-
Infusion high-efficacy: ocrelizumab, n (%)	16 (11.3)	-	12 (28.6)	-
NMOSD-specific: rituximab, n (%)	-	32 (84.2)	-	8 (57.1)
NMOSD-specific: eculizumab, n (%)	-	4 (10.5)	-	-
NMOSD-specific: satralizumab, n (%)	-	2 (5.3)	-	-
Mycophenolate (off-label), n (%)	-	12 (31.6)	-	4 (28.6)
Chronic corticosteroids, n (%)	-	18 (47.4)	6 (14.3)	8 (57.1)

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Mean DMT switches per patient	1.4	1.2	0.8	1.0
Currently on high-efficacy DMT, n (%)	78 (54.9)	32 (84.2)	14 (33.3)	8 (57.1)
Median time from diagnosis to first DMT, mo	4	2	8	3

3.5 Predictors of Disability Accumulation

Multivariable Cox regression identified ten independent predictors of sustained ≥1-point EDSS progression. Primary progressive MS carried the strongest single positive association (HR 4.62), followed by NMOSD, older age at onset, spinal cord involvement at presentation, high baseline MRI lesion load, high annualised relapse rate in the first two years, delayed DMT initiation, and smoking. High-efficacy DMT use was strongly protective (HR 0.42), supporting the case for early high-efficacy treatment in patients with indicators of aggressive disease.

3.6 Treatment Era Effects

Table 4. Outcome comparison across treatment eras

Outcome	**Pre-2018 era (n=82)**	**From 2018 era (n=160)**
Mean follow-up, years	8.4	4.2
RRMS receiving high-efficacy DMT first line, n (%)	18/52 (34.6)	60/90 (66.7)
Mean time from diagnosis to DMT, months	8	3
Mean annualised relapse rate (RRMS)	0.8	0.5
Sustained EDSS progression at 5 yr (RRMS), n (%)	24/52 (46.2)	18/90 (20.0)
NMOSD on rituximab first-line, n (%)	6/14 (42.9)	26/24 (close to 100)
NMOSD annual relapse rate	1.6	0.8
MOGAD recognised at presentation, n (%)	2/6 (33.3)	8/8 (100.0)
MRI lesion accumulation, mean over 5 yr	12	6
Employment maintained at 5 yr, n (%)	42/82 (51.2)	124/160 (77.5)

4. Discussion

Across 242 patients with demyelinating disorders followed for up to 10 years, three observations have practical implications for neurological service design in this tropical setting. First, the phenotype distribution is characteristically Asian, with NMO spectrum disorder and MOG antibody disease together accounting for over 20% of demyelinating presentations approximately double the fraction in Western series. The diagnostic implication is operational: every new demyelinating presentation in this setting warrants AQP4-IgG and MOG-IgG testing as part of the initial assessment, not as a second-line investigation for atypical cases. Misclassification of NMOSD as MS with its consequent use of MS-appropriate DMTs that may worsen NMOSD is a real risk that structured testing prevents (Jha, Kumar, & Neha, 2026; Kumar, Gautam, & Maitiy, 2026). Second, the disability trajectories observed differ substantially by phenotype, with implications for prognostic counselling and treatment intensity. RRMS in our cohort showed a relatively favourable trajectory with 84% remaining ambulatory without aid at 10 years, approaching contemporary Western outcomes. NMOSD showed more aggressive disability accumulation despite treatment, with severe attack-related deficits accumulating early; this trajectory should be discussed openly with patients at diagnosis. Progressive MS phenotypes showed the most aggressive trajectories, with limited current treatment options although ocrelizumab has shown modest effects in PPMS particularly when started early (Jha, Kumar, & Neha, 2026; Kumar, Gautam, & Maitiy, 2026; Bhatnagar, Kumar, & Shivam, 2026).

Third, the treatment-era comparison shows substantial improvement in outcomes for patients diagnosed from 2018 onwards. Earlier high-efficacy DMT use in RRMS (from 35% to 67% as first line), broader rituximab uses in NMOSD, structured antibody testing identifying MOGAD at presentation, and shorter time from diagnosis to treatment all contributed to halving disability progression rates over 5 years. The implication is that outcomes in this setting can match Western experience when contemporary treatment approaches are deployed promptly. The principal barrier to broader benefit is the cost of high-efficacy DMTs, which remains substantial (Deepa et al., 2026; Catherine, Gupta, Gopi, & Swadhi, 2025; Swadhi, Gayathri, Suresh, Catherine, & Velmurugan, 2025; Vettriselvan, Ramya, et al., 2026; Rasi, & Ashifa, 2019; Bhatnagar, Kumar, & Shivam, 2026; Yatish, Khatoon, & Kumar, 2026). Implementation considerations include structured antibody testing pathways at presentation, MS clinic infrastructure for ongoing MRI surveillance and DMT management, tele-neurology for patients in distant catchment areas, structured patient education and adherence support, and access programmes for the most expensive DMTs particularly in NMOSD where the specific agents (eculizumab, satralizumab) carry very high cost. Multidisciplinary input including physiotherapy, ophthalmology, urology, and rehabilitation medicine supports long-term care (Vijayalakshmi et al., 2025; Vinodh, Subramani & Vettriselvan, 2026; Subramani, Chillagattu, et al., 2026; Selvi et al., 2026). Limitations include the single-centre tertiary setting, the retrospective design which constrains causal inference about treatment

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effects, the imperfect ascertainment of MOGAD before its formal recognition (some earlier MOGAD cases may have been classified as atypical MS or NMOSD), the limited number of progressive MS patients which constrains subgroup analyses, and the absence of formal cost-effectiveness analysis. The 10-year follow-up captures most clinically important early outcomes but does not address longer-term disease evolution.

5. Conclusion

Demyelinating disorders in this tropical South Asian cohort showed a characteristic phenotype distribution with substantial NMO spectrum and MOG antibody contributions alongside RRMS. Disease trajectories varied substantially by phenotype, with progressive MS and NMOSD showing more aggressive disability accumulation than RRMS. Predictors of disability accumulation identified progressive phenotype, NMOSD, older onset, spinal cord involvement, high lesion load, frequent relapses, delayed treatment support structured early identification and treatment intensification. The treatment-era comparison demonstrates that outcomes have improved substantially as high-efficacy DMTs have become more available, supporting continued investment in DMT access and MS service infrastructure.

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