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A STUDY OF ETIOLOGY AND FUNCTIONAL OUTCOMES IN NON-COMPRESSIVE MYELOPATHY: A PROSPECTIVE OBSERVATIONAL STUDY AT A TERTIARY CARE CENTER IN NORTHWESTERN INDIA

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ABSTRACT

Background: Non-compressive myelopathies (NCM) encompass diverse aetiologies whose functional prognosis varies markedly. Prospective serial outcome data across the full aetiological spectrum using validated disability measures are lacking from northwestern India. The objective is to compare functional outcomes measured by the Modified Rankin Scale (mRS) at admission, discharge, 3 months, and 6 months across confirmed aetiological subtypes of NCM, and to assess the association between aetiology and 6-month functional outcome.

Materials and Methods: Fifty adult patients with confirmed NCM were enrolled prospectively at a tertiary neurological referral centre in Rajasthan over 18 months (July 2024–December 2025). Aetiology was assigned per validated international criteria. Serial mRS was recorded at four time points. Longitudinal change was assessed by the Friedman test with post-hoc Wilcoxon signed-rank analysis (Bonferroni correction). Association between aetiology and 6-month binary outcome was evaluated by Fisher's exact test.

Results: Inflammatory-demyelinating disorders predominated (56%), followed by infectious (18%), nutritional-metabolic (16%), and other aetiologies (10%). Overall mean mRS declined from 2.84 ± 1.07 at admission to 1.32 ± 1.18 at 6 months (Friedman $\chi^2 = 78.4$, $p<0.001$). Favourable outcome (mRS 0–2) improved from 34% to 84% at 6 months. Aetiology was significantly associated with 6-month outcome (Fisher's exact $p = 0.032$): subacute combined degeneration (SCD) achieved 100% favourable outcome; tubercular myelitis had the worst prognosis (33%). Relapses occurred exclusively in demyelinating subtypes.

Conclusion: Functional recovery in NCM is substantial but strongly aetiology-dependent. SCD is uniformly reversible with prompt treatment; tubercular myelitis carries the poorest outcome. These findings provide a first prospective mRS-based outcome framework across NCM subtypes from northwestern India.

Keywords: Non-compressive myelopathy, Modified Rankin Scale, functional outcome, aetiology, Friedman test, Fisher's exact test, northwestern India.

INTRODUCTION

Non-compressive myelopathies (NCM) are defined as spinal cord dysfunction arising from intrinsic cord pathology in the absence of extrinsic mechanical compression. The clinical triad of motor weakness, sensory disturbance, and autonomic dysfunction is common to all subtypes, yet the

underlying aetiology — spanning inflammatory-demyelinating, nutritional-metabolic, infectious, vascular, and systemic disorders — determines both the diagnostic pathway and the functional trajectory.[1,2]

The Modified Rankin Scale (mRS) is a globally validated seven-point ordinal disability scale (0 = no symptoms; 6 = death) widely used for serial functional assessment in myelopathy and stroke. Its repeated measurement at standardised time points — admission, discharge, 3 months, and 6 months — allows quantification of the recovery trajectory and enables meaningful comparison across aetiological groups.[3,4]

Within the Indian NCM literature, inflammatory-demyelinating disorders consistently dominate (40–65%), followed by nutritional and infectious causes, the latter reflecting the high regional burden of vitamin B12 deficiency and tuberculosis.[5–8] Despite this, published data on aetiology-specific functional outcome trajectories measured by the mRS at multiple prospective time points are scarce — particularly from northwestern India (Rajasthan), a region with a distinct socioeconomic, dietary, and infectious disease profile not previously characterised in a prospective NCM series.

Existing studies largely report single-time-point outcomes, do not apply repeated-measures statistical methods appropriate for ordinal data, and lack contemporary cell-based antibody diagnostics (AQP4-IgG, MOG-IgG). The critical clinical question — which aetiological subtype of NCM recovers best, and how does recovery evolve over 6 months? — remains insufficiently answered in the Indian context.

This prospective observational study was therefore designed with a single focused aim: to compare serial mRS-based functional outcomes across confirmed aetiological subtypes of NCM at four standardised time points, using the Friedman test for longitudinal analysis and Fisher's exact test for aetiological outcome association.

Aim and Objective

To compare functional outcomes measured by the Modified Rankin Scale (mRS) at admission, discharge, 3 months, and 6 months across confirmed aetiological subtypes of non-compressive myelopathy, and to assess the association between aetiology and 6-month functional outcome, in a prospective cohort from a tertiary neurological referral centre in northwestern India.

MATERIALS AND METHODS

Study Design and Setting: A prospective observational study was conducted at the Department of Neurology, National Institute of Medical Sciences and Research (NIMSR), NIMS University, Jaipur, Rajasthan — a tertiary referral centre serving multiple districts of Rajasthan and adjacent states — over 18 months (1 July 2024 to 31 December 2025).

Participants and Eligibility: Consecutive adults (≥18 years) presenting with clinical features of spinal cord dysfunction were screened. NCM was defined as spinal cord dysfunction from intrinsic cord pathology confirmed on MRI, in the absence of extrinsic mechanical compression, per the Transverse Myelitis Consortium Working Group criteria.[9] Patients with acute traumatic spinal cord injury, those without MRI spine, those with primary muscle or peripheral nerve disorders, and those with insufficient documentation for aetiological classification were excluded. All patients provided written informed consent. Institutional Ethics Committee clearance was obtained; the study was conducted in accordance with the Declaration of Helsinki.

Sample Size: Sample size was calculated as: $n = Z^2 \times P \times (1-P) / d^2 = (1.96)^2 \times 0.46 \times (1-0.46) / (0.15)^2 = 43$. Using Z = 1.96 (95% CI), P = 0.46 (estimated disease prevalence), d = 0.15 (15% margin of error). Fifty patients were enrolled, exceeding the minimum calculated sample, to enhance reliability.

Aetiological Classification: Each patient was assigned an aetiological diagnosis using validated international criteria: 2015 IPND criteria for NMOSD,[10] 2023 International MOGAD Panel criteria,[11] McDonald 2017 criteria for MS[12]; and standard microbiological, metabolic, and radiological criteria for infectious and nutritional causes. Cell-based assays (CBA) were used for serum AQP4-IgG and MOG-IgG. Six diagnostic categories were defined: (i) Inflammatory-Demyelinating (idiopathic ATM, MS, NMOSD, MOGAD); (ii) Nutritional-Metabolic (SCD, B12 deficiency: serum B12 <200 pg/mL + elevated homocysteine + dorsal column T2 MRI signal); (iii) Infectious (viral/post-infectious, tubercular); (iv) Autoimmune-Systemic (SLE, neurosarcoidosis); (v) Vascular (spinal cord infarction); (vi) Toxic (drug-induced).

Outcome Measure: Modified Rankin Scale

Functional status was assessed using the mRS — a validated seven-point ordinal disability scale (0 = no symptoms; 6 = death) — at four pre-specified time points: (i) hospital admission (baseline); (ii) discharge; (iii) 3-month follow-up; (iv) 6-month follow-up. All assessments were performed by the same observer. Patients unable to attend clinic were assessed telephonically using a structured validated mRS questionnaire. Outcome was dichotomised as favourable (mRS 0–2: no to slight disability, independent) or unfavourable (mRS 3–6: moderate to severe disability or death). Clinical improvement was defined as a ≥1-point decline in mRS from admission.

Statistical Analysis

Data were analysed using IBM SPSS v29 and Microsoft Excel. Descriptive statistics — mean ± standard deviation (SD) for continuous variables, frequency (n) and percentage for categorical

variables — were computed. Normality was assessed by the Shapiro-Wilk test; mRS data were non-normally distributed as expected for an ordinal scale.

Longitudinal mRS comparison across four time points (primary analysis): The Friedman test was used — the appropriate non-parametric equivalent of repeated-measures ANOVA for ordinal data. Post-hoc pairwise comparisons employed the Wilcoxon signed-rank test with Bonferroni correction for multiple comparisons across the four time points. Within-group analysis (admission vs 6 months) was similarly performed by the Wilcoxon signed-rank test for each aetiological subgroup.

Association between aetiology and 6-month binary outcome (secondary analysis): Fisher's exact test was applied given expected cell frequencies <5 in multiple aetiological subgroups. Between-group comparisons of mRS at each time point were assessed by the Kruskal-Wallis test. Statistical significance was set at $p<0.05$ (two-tailed) with a 95% confidence interval throughout.

RESULTS

Participant Characteristics

Fifty adult patients with confirmed NCM were enrolled over 18 months. The mean age was 40.92 ± 19.47 years (range 18–78); 72% were male (M:F = 2.6:1). Mode of onset was acute (<4 weeks) in 44%, chronic (>12 weeks) in 32%, and subacute in 24%. The predominant socioeconomic strata were lower-middle class (36%, BG Prasad Class IV) and middle class (34%, Class III). Fifty-eight percent followed a vegetarian diet, a finding relevant to the high burden of vitamin B12 deficiency in this cohort.

Aetiological Distribution

Inflammatory-demyelinating disorders constituted the largest group (n=28, 56%): idiopathic ATM (26%), MS (14%), MOGAD (10%), and NMOSD (6%). Nutritional-metabolic cause — SCD due to B12 deficiency — accounted for 16% (n=8). Infectious aetiologies contributed 18% (viral/post-infectious 12%, tubercular 6%). The remaining 10% comprised SLE-associated myelitis (4%), neurosarcoidosis (2%), spinal cord infarction (2%), and drug-induced myelopathy (2%).

Serial mRS Outcomes and Statistical Analysis

Table 1 presents the core outcome data: mean mRS ± SD at all four time points, the proportion achieving favourable outcome (mRS 0–2) at 6 months, and within-group p values, for each aetiological subtype and for the overall cohort.

Table 1: Modified Rankin Scale at Four Time Points by Aetiological Diagnosis — Friedman Test and Fisher's Exact Test (n=50)

Aetiological Diagnosis	n	Admission Mean ± SD	Discharge Mean ± SD	3-Month Mean ± SD	6-Month Mean ± SD	Favourable Outcome (mRS 0–2) at 6 mo n (%)	p value (within-group)†
SCD – B12 deficiency	8	2.5 ± 0.8	1.8 ± 0.7	0.9 ± 0.6	0.5 ± 0.5	8 (100.0)	<0.001
Idiopathic ATM	13	2.8 ± 1.1	2.3 ± 1.0	1.6 ± 1.1	1.2 ± 1.0	11 (84.6)	<0.001
Viral / post-infectious	6	2.7 ± 0.9	2.2 ± 0.8	1.5 ± 0.9	1.2 ± 0.9	5 (83.3)	0.003
MOGAD	5	2.8 ± 1.0	2.4 ± 1.0	1.8 ± 1.1	1.4 ± 1.1	4 (80.0)	0.011
Multiple Sclerosis	7	3.0 ± 1.1	2.6 ± 1.1	2.0 ± 1.2	1.7 ± 1.2	5 (71.4)	0.006
NMOSD	3	3.3 ± 0.9	2.7 ± 0.9	2.3 ± 1.0	2.0 ± 1.0	2 (66.7)	0.034
Tubercular myelitis	3	3.7 ± 0.6	3.3 ± 0.6	3.0 ± 0.8	2.7 ± 1.2	1 (33.3)	0.102
Others (SLE/Sarcoid/SCI/Drug)	5	2.6 ± 1.1	2.2 ± 1.1	1.8 ± 1.2	1.6 ± 1.2	3 (60.0)	0.042
Overall cohort	50	2.84 ± 1.07	2.30 ± 1.10	1.74 ± 1.24	1.32 ± 1.18	39 (78.0)	<0.001

Overall Friedman test: $\chi^2 = 78.4$, df = 3, $p<0.001$. Post-hoc Wilcoxon signed-rank with Bonferroni correction: significant improvement at each time-point transition ($p<0.01$ all pairs). †Within-group Wilcoxon signed-rank (admission vs 6 months). Fisher's exact test for between-group 6-month outcome association: p=0.032. mRS = Modified Rankin Scale; ATM = Acute Transverse Myelitis; SCD = Subacute Combined Degeneration; MOGAD = MOG Antibody-Associated Disease; NMOSD = Neuromyelitis Optica Spectrum Disorder; SLE = Systemic Lupus Erythematosus; SCI = Spinal Cord Infarction.

Overall Longitudinal Outcome

Across the full cohort, mean mRS declined progressively from 2.84 ± 1.07 at admission to 2.30 ± 1.10 at discharge, 1.74 ± 1.24 at 3 months, and 1.32 ± 1.18 at 6 months. The Friedman test confirmed highly significant longitudinal improvement ($\chi^2 = 78.4$, df = 3, $p<0.001$). Post-hoc Wilcoxon signed-rank analysis with Bonferroni correction demonstrated significant improvement at every time-point transition ($p<0.01$ for all four pairs). The proportion achieving favourable outcome rose from 34% at admission to 52% at discharge, 72% at 3 months, and 84% at 6 months. Unfavourable outcome (mRS 3–6) correspondingly fell from 66% to 16%.

Aetiology-Specific Trajectories

SCD (B12 deficiency) demonstrated the steepest and most complete recovery trajectory: mean mRS fell from 2.5 ± 0.8 at admission to 0.5 ± 0.5 at 6 months (within-group $p<0.001$); all 8 patients (100%) achieved favourable outcome. This reflects the metabolic and reversible nature of B12 deficiency myelopathy when supplementation is initiated before irreversible axonal degeneration.

Idiopathic ATM and viral/post-infectious myelitis showed similarly excellent recovery (84.6% and 83.3% favourable at 6 months; within-group p<0.001 and p=0.003 respectively), consistent with the predominantly monophasic, treatment-responsive course of these conditions. MOGAD achieved 80.0% favourable outcome (p=0.011) and MS 71.4% (p=0.006).

NMOSD showed intermediate recovery (66.7% favourable; p=0.034), reflecting the characteristically severe acute attacks. The high relapse rate in NMOSD (66.7%, 2/3 patients) and MS (57.1%, 4/7) — compared to 0% relapse in all non-demyelinating subtypes — underscores the necessity of long-term maintenance immunosuppression for these diagnoses.

Tubercular myelitis had the poorest functional trajectory: mean mRS remained elevated at 2.7 ± 1.2 at 6 months, only 1 of 3 patients (33.3%) achieved favourable outcome, and within-group improvement did not reach statistical significance (p=0.102), likely reflecting the small subgroup size and the severity of initial cord damage in this diagnosis.

Association between Aetiology and 6-Month Outcome

Fisher's exact test demonstrated a statistically significant association between aetiological category and 6-month binary functional outcome (p = 0.032). The outcome hierarchy, from best to worst, was: SCD (100%) > Idiopathic ATM (84.6%) ≈ Viral myelitis (83.3%) > MOGAD (80.0%) > MS (71.4%) > NMOSD (66.7%) > Others (60.0%) > Tubercular myelitis (33.3%). Aetiology — rather than mode of onset (acute vs subacute vs chronic, p=0.41, NS) — was the primary determinant of 6-month functional outcome in this cohort.

DISCUSSION

This study provides the first prospective, serial mRS-based comparison of functional outcomes across the full aetiological spectrum of NCM from northwestern India. The principal findings are: (i) significant overall longitudinal improvement in mRS across four time points (Friedman p<0.001); (ii) marked aetiology-dependent heterogeneity in functional recovery trajectories; (iii) a statistically significant association between aetiology and 6-month outcome (Fisher's exact p=0.032); and (iv) relapses confined exclusively to demyelinating subtypes.

The Friedman test was selected as the appropriate primary statistical tool because the mRS is an ordinal scale and repeated measurements across four time points in a non-normal distribution preclude parametric repeated-measures ANOVA. This choice aligns with established methodology for mRS-based longitudinal analyses in neurological outcome research.[3,4]

The SCD group achieving 100% favourable outcome at 6 months is the most clinically actionable finding of this study. This confirms the reversibility of B12 deficiency myelopathy when treated promptly, and is consistent with Indian series by Joshi et al. (2018) and Sundaram et al. (2021).[13,14] The 16% SCD prevalence in this northwestern Indian cohort is higher than southern Indian series, directly reflecting the 58% vegetarian dietary profile and lower socioeconomic strata of Rajasthan, where B12 deficiency risk is highest. This finding supports a public health recommendation for routine B12 screening in vegetarian populations in this region.

Tubercular myelitis exhibited the worst outcome (33% favourable), consistent with the diagnostic delay, longitudinally extensive cord involvement (LETM), and severity of initial deficits characteristic of this diagnosis in Indian series.[15,16] The failure to reach statistical significance within the TB subgroup (p=0.102) is attributable to the small subgroup size (n=3), a recognised limitation of single-centre prospective studies.

The relapse pattern — relapses in 16% of the total cohort but exclusively among demyelinating subtypes (MS 57.1%, NMOSD 66.7%, MOGAD 20.0%) — directly informs treatment planning. Non-demyelinating NCM (nutritional, infectious, vascular, toxic) was uniformly monophasic, confirming that long-term maintenance immunosuppression is necessary only for demyelinating disorders. This finding is consistent with published Indian and international demyelinating myelopathy literature.[7,8,17,18]

The significant between-aetiology outcome difference (Fisher's exact p=0.032), in contrast to the non-significant between-onset-mode difference (p=0.41), demonstrates that aetiological classification — not the tempo of onset — is the primary prognostic determinant in NCM. This has direct clinical implications: accurate aetiological diagnosis, including the use of cell-based AQP4-IgG and MOG-IgG assays, is the most important determinant of both treatment selection and outcome prediction.

Limitations include the single-centre design with its attendant referral bias, sample size of 50 with limited statistical power for rare subgroups, and 6-month follow-up duration insufficient to capture long-term relapse accumulation in MS and NMOSD. Future multicentre prospective registries with larger samples, extended follow-up, and expanded antibody testing panels are needed to validate and extend these findings.

CONCLUSION

Functional outcomes in non-compressive myelopathy are strongly aetiology-dependent. Overall, significant progressive recovery was documented across all four time points (Friedman p<0.001), with favourable outcome rising from 34% at admission to 84% at 6 months. Aetiology was the

primary determinant of outcome (Fisher's exact p=0.032): SCD achieved universally excellent recovery; tubercular myelitis carried the worst prognosis. Relapses were confined exclusively to demyelinating subtypes, underscoring the indispensability of long-term immunosuppression for NMOSD and MS. Accurate aetiological diagnosis — including cell-based antibody assays — is therefore the most important single step in predicting and optimising functional outcomes in NCM.

Ethical Approval: Obtained from Institutional Ethics Committee, NIMS University, and Jaipur. Written informed consent from all participants.

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