



ORIGINAL RESEARCH PAPER

Neurology

MAPPING THE MADURAI MYELIN PUZZLE: CLINICAL RADIOLOGICAL SPECTRUM OF CENTRAL NERVOUS SYSTEM DEMYELINATING DISORDERS IN SOUTHERN INDIA

KEY WORDS: Aquaporin-4, Longitudinally Extensive Transverse Myelitis, Modified Rankin Scale, Mycophenolate Mofetil, Myelin Oligodendrocyte Glycoprotein, Neuromyelitis Optica Spectrum Disorder, Visual Evoked Potential.

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ABSTRACT

Introduction: The nosology of central nervous system (CNS) demyelinating disease has changed considerably over the past decade, and antibody testing against aquaporin-4 (AQP4) and myelin oligodendrocyte glycoprotein (MOG) now separates entities that were previously grouped together (NEMOS et al., 2016). Data describing how this spectrum presents in public-sector hospitals in southern India remain limited (Pandit & Mustafa, 2021). **Aim:** To describe the demographic, clinical, electrophysiological, radiological and short-term outcome profile of patients admitted with CNS demyelinating disorders at a government tertiary care centre in southern Tamil Nadu. **Materials and Methods:** Fifty-five consecutive patients admitted to the Department of Neurology, Madurai Medical College and Government Rajaji Hospital with a clinically and radiologically supported CNS demyelinating event were studied. Serum AQP4-IgG and MOG-IgG were tested by cell-based assay where feasible. All patients underwent magnetic resonance imaging (MRI) of the relevant neuraxis and pattern-reversal visual evoked potentials (VEP). Disability was graded using the modified Rankin Scale (mRS) at admission and at discharge. Categorical variables were compared using the Fisher exact test, continuous variables using the Mann-Whitney U test, and paired mRS scores using the Wilcoxon signed-rank test; $p < 0.05$ was taken as significant. **Results:** The cohort comprised 35 women and 20 men (female-to-male ratio 1.75:1) with a mean age of 31.3 ± 13.7 years (range 13-65 years); nine patients (16.4%) were below 18 years of age. 40 patients (72.7%) presented with a first demyelinating event. AQP4-IgG seropositive NMOSD was the commonest diagnosis ($n=20$, 36.4%) and occurred exclusively in women, followed by MOGAD ($n=16$, 29.1%), seronegative NMOSD-phenotype disease ($n=6$, 10.9%), multiple sclerosis ($n=5$, 9.1%) and six patients (10.9%) in whom antibody testing could not be completed. One patient each had post-infectious cerebellitis and tapentadol-associated toxic demyelination. The female preponderance in AQP4-IgG disease was significantly greater than in MOGAD (20/20 versus 9/16, $p=0.001$), and patients with multiple sclerosis were significantly older than the rest of the cohort (median 53 versus 29 years, $p=0.009$). Myelitis, alone or combined with optic neuritis, accounted for 30 presentations (54.5%). VEPs were abnormal in 24 patients (43.6%), most often in the AQP4-IgG group (15/20, 75.0%). Longitudinally extensive transverse myelitis was the single commonest MRI abnormality (24/55, 43.6%). All patients received intravenous methylprednisolone; 39 (70.9%) were started on mycophenolate mofetil and 7 (12.7%) received rituximab. Among the 53 survivors the median mRS improved from 5 (IQR 5-5) at admission to 2 (IQR 1-2) at discharge ($p < 0.001$), and 45 (84.9%) were functionally independent (mRS 0-2) at discharge. Two patients died (3.6%). **Conclusion:** Antibody-defined NMOSD and MOGAD together accounted for roughly two-thirds of demyelinating admissions in this South Indian cohort, whereas multiple sclerosis was uncommon and occurred in an older age group. Most patients were severely disabled on admission yet recovered functional independence with corticosteroids and early oral immunosuppression, which suggests that a serology-guided approach is both feasible and worthwhile even where monoclonal therapies are not routinely affordable.

INTRODUCTION

Inflammatory demyelinating disorders of the central nervous system (CNS) are no longer regarded as variants of a single disease. The demonstration that aquaporin-4 immunoglobulin G (AQP4-IgG) is a pathogenic antibody in neuromyelitis optica spectrum disorder (NMOSD), and the subsequent recognition of myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) as a separate entity, have redrawn the boundaries between conditions that used to be labelled together as "optico-spinal" or "atypical" multiple sclerosis (MS) (Banwell et al., 2023; Wingerchuk et al., 2015). The distinction matters at the bedside because the three disorders differ in attack severity, relapse risk and, most importantly, in their response to disease-modifying therapy: agents that benefit MS may worsen NMOSD, and the interval to starting maintenance immunosuppression influences long-term disability (Wingerchuk et al., 2007). Indian data on this spectrum are still sparse. The only population-based survey available, from urban Mangalore, reported a prevalence of 8.3 per 100,000 for MS and 2.6 per 100,000 for NMOSD, with NMO and its spectrum disorders making up about 14% of all demyelinating disease (Pandit & Kundapur, 2014). Hospital-

based series from various parts of the country have consistently described a higher relative burden of NMOSD than is seen in Western cohorts, along with a younger age at onset and a marked female preponderance in the AQP4-IgG seropositive group (Jagtap et al., 2015; Pandit & Mustafa, 2021; Singhal et al., 2014). What is less clear is how this spectrum behaves in a government tertiary care hospital serving a predominantly rural, low-income population, where antibody testing is not universally available, delays to presentation are common and long-term monoclonal antibody therapy is largely beyond the reach of most families. The Department of Neurology at Government Rajaji Hospital, Madurai, is a referral centre for the southern districts of Tamil Nadu and receives a substantial number of patients with acute demyelinating events. We undertook this study to document the clinical presentation, antibody profile, electrophysiological and imaging findings, treatment received and short-term functional outcome of consecutive patients admitted with CNS demyelinating disease, and to examine whether the phenotypic separations described in international cohorts are reproduced in our setting.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based observational study conducted in the Department of Neurology, Madurai Medical College and Government Rajaji Hospital, Madurai, a tertiary care teaching institution in southern Tamil Nadu. Fifty-five consecutive patients admitted with a clinically suspected and radiologically supported CNS demyelinating event were enrolled. Written informed consent was taken from every patient or, for those below 18 years of age, from a parent or legal guardian.

Inclusion and exclusion criteria

Patients of either sex and any age presenting with an acute or subacute neurological deficit attributable to CNS demyelination, supported by characteristic MRI abnormalities, were included. Patients with compressive myelopathy, spinal or cerebral infarction, neoplastic or infective lesions mimicking demyelination, nutritional myelopathy and peripheral nervous system disease alone were excluded after appropriate investigation.

Clinical evaluation

A structured proforma was used to record age, sex, comorbid illness, drug and substance history, the presenting syndrome and whether the event was a first attack or a relapse. Presenting syndromes were classified as isolated optic neuritis, isolated myelitis, combined optic neuritis with myelitis, brainstem or cerebellar syndrome, and cerebral or hemispheric syndrome (including acute disseminated encephalomyelitis and tumefactive demyelination). Disability was graded by the modified Rankin Scale (mRS) at admission and again at discharge by the treating neurologist (Swieten et al., 1988).

Investigations

Serum AQP4-IgG and MOG-IgG were tested by cell-based assay in a referral laboratory. Where the antibody result was unavailable because of cost or because the sample could not be dispatched, the patient was recorded as untested rather than as seronegative, and the syndromic diagnosis was retained. Cerebrospinal fluid analysis including oligoclonal band estimation was performed where indicated. All patients underwent MRI of the brain, with dedicated orbit sequences and contiguous spinal cord imaging as clinically dictated, using contrast where renal function permitted. Longitudinally extensive transverse myelitis (LETM) was defined as a cord lesion extending over three or more contiguous vertebral segments. Pattern-reversal visual evoked potentials were recorded in all patients and reported as normal or abnormal on the basis of P100 latency and amplitude, with optic atrophy on funduscopy noted separately.

Diagnostic classification

NMOSD was diagnosed using the 2015 International Panel for NMO Diagnosis criteria (Wingerchuk et al., 2015), MOGAD using the 2023 International MOGAD Panel proposed criteria (Banwell et al., 2023), and MS using the 2017 revisions of the McDonald criteria (Thompson et al., 2017). Patients fulfilling NMOSD criteria who tested negative for both AQP4-IgG and MOG-IgG were designated as seronegative NMOSD-phenotype disease.

Treatment

All patients received intravenous methylprednisolone 1 g daily for five days followed by an oral corticosteroid taper. Maintenance immunosuppression with mycophenolate mofetil or azathioprine was initiated in patients with a relapsing course, AQP4-IgG seropositivity or a severe index attack. Rituximab was reserved for patients with breakthrough relapses on oral immunosuppression or with a particularly severe presentation, subject to affordability.

Statistical analysis

Data were entered in Microsoft Excel and analysed using

standard statistical software. Continuous variables are expressed as mean $\pm$ standard deviation or as median with interquartile range (IQR), and categorical variables as frequencies and percentages. Between-group comparison of proportions was performed using the Fisher's exact test and of continuous variables using the Mann-Whitney U test, given the small subgroup sizes. Admission and discharge mRS scores were compared using the Wilcoxon signed-rank test. A two-tailed *p*-value below 0.05 was considered statistically significant.

RESULTS

Demographic profile

Fifty-five patients were studied. Thirty-five (63.6%) were women and 20 (36.4%) were men, giving a female-to-male ratio of 1.75:1. The mean age at presentation was 31.3 ± 13.7 years, with a median of 30 years and a range of 13 to 65 years. Nine patients (16.4%) were below 18 years of age, and the youngest three were 13 years old. Forty patients (72.7%) were seen during a first demyelinating event and 15 (27.3%) during a relapse; the highest number of previous attacks in any single patient was 4. Comorbidity was uncommon. Two patients had type 2 diabetes mellitus with systemic hypertension, one had systemic lupus erythematosus preceding the demyelinating event, one had undergone atrial septal defect closure, and one young man had a documented history of tapentadol abuse. The demographic and clinical characteristics of the cohort are summarised in [Table/Fig-1].

[Table/Fig-1]: Demographic and clinical characteristics of the study cohort (n=55).

Variable	Value	Percentage
Total patients	55	100.0
Female	35	63.6
Male	20	36.4
Mean age (years)	31.3 ± 13.7	–
Median age (years)	30 (range 13–65)	–
Age < 18 years	9	16.4
First demyelinating event	40	72.7
Relapse at presentation	15	27.3
Any comorbid illness	5	9.1
mRS 4–5 at admission	53	96.4

mRS: modified Rankin Scale

Diagnostic categories

AQP4-IgG seropositive NMOSD was the single largest group, accounting for 20 patients (36.4%), and every one of these patients was female. MOGAD was diagnosed in 16 patients (29.1%), among whom the sex distribution was almost equal (9 women, 7 men). Six patients (10.9%) fulfilled NMOSD criteria but were negative for both antibodies, 6 (10.9%) could not have antibody testing completed and were retained under their syndromic label, and 5 (9.1%) were diagnosed with multiple sclerosis, of whom 3 had cerebrospinal fluid oligoclonal bands. One adolescent presented with bilateral cerebellar demyelination following a febrile illness and was classified as post-infectious cerebellitis, and 1 young man with a history of tapentadol abuse had brainstem demyelination attributed to toxic leukoencephalopathy [Table/Fig-2].

The female preponderance in AQP4-IgG disease (20 of 20) was significantly greater than in MOGAD (9 of 16; Fisher exact test, *p*=0.001). Patients with multiple sclerosis were considerably older than the remainder of the cohort, with a median age of 53 years against 29 years (Mann-Whitney U test, *p*=0.009). There was no significant difference in age between the AQP4-IgG and MOGAD groups (*p*=0.51). Eight of the nine children and adolescents belonged to the MOGAD or seronegative groups, and no patient below 18 years was diagnosed with multiple sclerosis.

[Table/Fig-2]: Distribution of diagnostic categories with age and sex (n=55).

Diagnostic category	n (%)	Fem ale	Male	Mean age (years)
AQP4-IgG NMOSD	20 (36.4)	20	0	32.0 ± 9.9
MOGAD	16 (29.1)	9	7	31.1 ± 15.7
Seronegative NMOSD phenotype	6 (10.9)	2	4	19.2 ± 6.9
Antibody testing incomplete	6 (10.9)	2	4	29.7 ± 9.4
Multiple sclerosis	5 (9.1)	2	3	50.4 ± 14.8
Post-infectious cerebellitis	1 (1.8)	0	1	22.0
Toxic demyelination	1 (1.8)	0	1	19.0

AQP4: aquaporin-4; NMOSD: neuromyelitis optica spectrum disorder; MOGAD: myelin oligodendrocyte glycoprotein antibody-associated disease

Clinical presentation

Myelitis dominated the clinical picture. Eighteen patients (32.7%) presented with isolated myelitis, most often as an acute paraparesis or quadriparesis with sphincter involvement, and a further 12 (21.8%) had simultaneous or sequential optic neuritis and myelitis, so that some form of spinal cord involvement accounted for 30 presentations (54.5%). Isolated optic neuritis was the presenting syndrome in 11 patients (20.0%), bilateral in eight of them. Brainstem and cerebellar syndromes were seen in seven patients (12.7%) and included two visually striking presentations, a one-and-a-half syndrome in a 20-year-old woman and an eight-and-a-half syndrome with bilateral facial palsy, trigeminal involvement and ataxia in an 18-year-old girl. One 13-year-old girl with AQP4-IgG seropositive disease presented with intractable vomiting and hiccups characteristic of the area postrema syndrome, and a 42-year-old woman presented with isolated left hemichorea arising from a brainstem demyelinating lesion. Cerebral and hemispheric presentations accounted for the remaining seven patients (12.7%), comprising two children with acute disseminated encephalomyelitis, three patients with tumefactive lesions and two with a cognitive-behavioural presentation [Table/Fig-3].

Two unusual sensory manifestations deserve mention. A 36-year-old woman in her fourth relapse of AQP4-IgG disease reported neuropathic pruritus with myelopathy hand, and a 32-year-old woman with underlying systemic lupus erythematosus presented with paraparesis, optic neuritis and a coexisting peripheral neuropathy.

[Table/Fig-3]: Presenting clinical syndrome (n=55).

Presenting syndrome	n (%)	AQP4-IgG	MOGAD
Isolated myelitis	18 (32.7)	7	5
Optic neuritis with myelitis	12 (21.8)	8	3
Isolated optic neuritis	11 (20.0)	3	6
Brainstem or cerebellar syndrome	7 (12.7)	2	1
Cerebral or hemispheric syndrome	7 (12.7)	0	1

Visual evoked potentials

VEP were abnormal in 24 patients (43.6%), the abnormality being prolongation of P100 latency in 23 and a pattern of optic atrophy in one adolescent girl with four previous episodes of bilateral optic neuritis. Abnormal VEP were most common in the AQP4-IgG group, where 15 of 20 patients (75.0%) had a delayed response, compared with 7 of 16 (43.8%) in the MOGAD group. Notably, all five patients with multiple sclerosis had normal VEP, which is explained by the fact that none of them presented with an optic nerve syndrome. In nine

patients, the VEP were abnormal in the absence of any current visual complaint, indicating subclinical optic nerve involvement.

Magnetic resonance imaging:

Spinal cord abnormalities were the most common imaging finding, present in 29 patients (52.7%), and LETM extending over three or more vertebral segments was documented in 24 patients (43.6%). Cervical cord involvement predominated, and two patients had a hemicord lesion producing a Brown-Séquard syndrome. Optic nerve contrast enhancement was the dominant abnormality in 12 patients (21.8%) and was bilateral in most of the MOGAD group. Brainstem or cerebellar demyelination was seen in seven patients (12.7%), tumefactive lesions in three (5.5%), periventricular lesions with Dawson fingers in two (3.6%), and an ADEM-like pattern of confluent subcortical lesions in two (3.6%). Three patients had simultaneous LETM and optic nerve enhancement. The distribution of imaging and electrophysiological findings across diagnostic groups is shown in [Table/Fig-4].

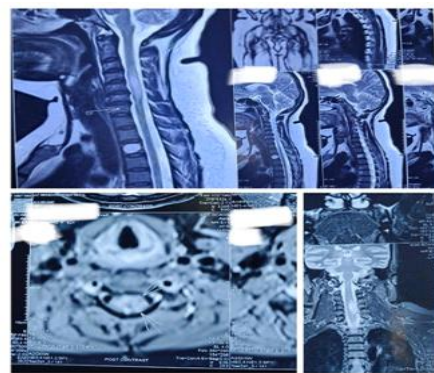


Fig 4-A shows T2 Flair hyperintense intramedullary long segment lesion in case of NMO Aquaporin Antibody positive patient.



FIG 4 -b The above image shows T2 Flair hyperintensity and DW Restriction in left thalamus in a case of Tapentadol induced Demyelination in a young male.

[Table/Fig-4]: Visual evoked potential and magnetic resonance imaging findings by diagnostic group.

Finding	AQP4-IgG (n=20)	MOGAD (n=16)	MS (n=5)	Others (n=14)	Total
Abnormal VEP	15	7	0	2	24
Spinal cord lesion	12	8	0	9	29
Optic nerve enhancement	5	6	0	1	12
Brainstem/cerebellar	3	1	0	3	7
Tumefactive lesion	0	0	3	0	3
Dawson fingers	0	0	2	0	2
ADEM-like pattern	0	1	0	1	2

VEP: visual evoked potential; MS: multiple sclerosis; ADEM: acute disseminated encephalomyelitis

Treatment

All 55 patients received intravenous methylprednisolone for the acute attack. Maintenance immunosuppression with mycophenolate mofetil was started in 39 patients (70.9%), azathioprine was used in one patient with systemic lupus

erythematosus, which also treated the underlying disease, and seven patients (12.7%) received rituximab in addition to oral immunosuppression. Rituximab was given predominantly to patients with a relapsing course, including four with three or more previous attacks. Plasma exchange was not administered to any patient in this series.

Outcome

Fifty-three patients (96.4%) survived the index admission and two (3.6%) died. Disability at admission was severe: 47 patients (85.5%) had an mRS of 5 and six (10.9%) an mRS of 4, so that 96.4% of the cohort was bedbound or worse at the time of presentation. Among survivors the median mRS fell from 5 (IQR 5-5) at admission to 2 (IQR 1-2) at discharge, a difference that was highly significant on the Wilcoxon signed-rank test ($p<0.001$). The mean improvement was 3.2 mRS points. Forty-five survivors (84.9%) were functionally independent at discharge with an mRS of 0 to 2, and 24 (45.3%) had minimal or no residual symptoms with an mRS of 0 to 1. Eight patients (15.1%) remained moderately disabled with an mRS of 3, and these were predominantly patients with recurrent optic neuritis and established optic atrophy or with long-standing LETM. Fifty-one survivors remained on regular follow-up and two were lost to follow-up after discharge [Table/Fig-5].

Both deaths occurred in patients whose demyelinating process was atypical. The first was a 19-year-old man with a history of tapentadol abuse who presented with right hemiparesis and brainstem demyelination consistent with opioid-related toxic leukoencephalopathy; he deteriorated despite corticosteroids and supportive care. The second was a 60-year-old man who presented with a seizure and no focal deficit but was found to have a large tumefactive demyelinating lesion; he was awaiting brain biopsy for confirmation when he died.

[Table/Fig-5]: Modified Rankin Scale at admission and discharge among survivors (n=53).

mRS score	At admission, n (%)	At discharge, n (%)	p-value
0-1	0 (0)	24 (45.3)	<0.001
2	0 (0)	21 (39.6)	
3	0 (0)	8 (15.1)	
4	6 (11.3)	0 (0)	
5	47 (88.7)	0 (0)	
Median (IQR)	5 (5-5)	2 (1-2)	<0.001

IQR: interquartile range. p-value by Wilcoxon signed-rank test comparing paired admission and discharge scores.

DISCUSSION

The most striking feature of this cohort is the composition of the demyelinating spectrum. Antibody-defined disease, taking AQP4-IgG NMOSD and MOGAD together, accounted for 36 of 55 patients (65.5%), whereas multiple sclerosis constituted only 9.1%. This inversion of the Western pattern, in which MS is by far the commonest inflammatory demyelinating disorder, has been reported repeatedly from Asian populations and from Indian hospital series (Pandit & Kundapur, 2014; Pandit & Mustafa, 2021; Singhal et al., 2014). Pandit and Kundapur found that NMO and its spectrum disorders made up about 14% of all demyelinating disease in an urban South Indian population, and hospital-based series have described proportions ranging from 9% to 24% (Pandit & Kundapur, 2014; Pandit & Mustafa, 2021). Our figure is higher still, but this almost certainly reflects referral and admission bias rather than true prevalence: patients with relapsing-remitting MS are often managed in the outpatient department or in the private sector, while the severe myelitis and bilateral optic neuritis that characterise NMOSD and MOGAD lead almost invariably to inpatient admission at a government hospital. A study confined to admitted patients therefore enriches for the severe end of the spectrum.

The absolute female predominance in the AQP4-IgG group, with all 20 patients being women, is consistent with the literature, where female-to-male ratios of 8:1 to 10:1 are usual (Singhal et al., 2014; Wingerchuk et al., 2015). The near-equal sex distribution in MOGAD in our series (9 women, 7 men) also mirrors published cohorts, in which MOGAD shows little sex predilection (Banwell et al., 2023; Pedapati et al., 2020). The difference between the two groups was statistically significant in our data ($p=0.001$), and although the numbers are small, sex remains a useful and freely available clinical pointer while awaiting serology in a resource-limited setting. Patients with multiple sclerosis in our cohort were substantially older than the rest (median 53 versus 29 years, $p=0.009$). This is at odds with the usual second-to-third decade onset of MS and is best explained by the small number of MS patients and by the nature of their presentations: three of the five had tumefactive or hemispheric disease with cognitive change, presentations that are more likely to prompt admission and neurological referral in an older patient in whom stroke or tumour is first suspected. It should not be read as evidence that MS presents late in this population.

Myelitis, whether isolated or combined with optic neuritis, was the presenting syndrome in more than half of our patients, and LETM was the commonest single MRI abnormality at 43.6%. This is in keeping with Indian NMOSD series in which myelitis is the most frequent initial attack (Jagtap et al., 2015; Pandit & Mustafa, 2021). The clinical corollary is important for general physicians in our region: an acute non-compressive myelopathy in a young woman with a cord lesion spanning three or more segments should prompt AQP4-IgG testing before it prompts a search for tuberculosis or a nutritional cause, and should not be treated as clinically isolated syndrome awaiting dissemination in time.

The pattern of VEP abnormality is worth emphasising. Three-quarters of AQP4-IgG patients had a prolonged P100, and in nine patients across the cohort the VEP was abnormal without any current visual symptom. VEP remains an inexpensive, widely available and non-invasive test in centres such as ours where optical coherence tomography is not routinely accessible, and the detection of subclinical optic nerve involvement can materially influence the decision to commit a patient to long-term immunosuppression. That all five MS patients had a normal VEP simply reflects their non-optic presentations and the small denominator; it should not be generalised.

Functional recovery in this cohort was substantially better than the severity at admission would have predicted. Ninety-six percent of patients were mRS 4 or 5 on admission, yet 84.9% of survivors were independent at discharge and the median mRS improved by three points. Two factors are likely to be responsible. The first is prompt high-dose corticosteroid therapy, the value of which is well established in demyelinating attacks and which was administered to every patient in this series (Kleiter et al., 2016). The second is the early initiation of oral immunosuppression, most often mycophenolate mofetil, in seven of every ten patients. Mycophenolate has been shown to reduce relapse frequency and stabilise or improve disability in NMOSD, with acceptable tolerability, and its low cost makes it a rational first-line maintenance agent in Indian public hospitals (Huh et al., 2014; Montcuquet et al., 2016). Rituximab was used selectively in relapsing disease, in keeping with comparative treatment data showing superior relapse prevention with B-cell depletion (Mealy et al., 2014). The newer approved biologics for AQP4-IgG NMOSD, including eculizumab, inebilizumab and satralizumab, have transformed relapse prevention in trial settings but remain financially inaccessible to the great majority of our patients (Cree et al., 2019; Pittock et al., 2019; Traboulsee et al., 2020).

Two deaths occurred, both outside the classical autoimmune categories. The patient with tapentadol-associated brainstem demyelination is a reminder that toxic leukoencephalopathy can closely mimic inflammatory demyelination on imaging, that opioid-related white matter injury carries a poor prognosis, and that tapentadol abuse is a growing problem in India (Filley & Kleinschmidt DeMasters, 2001). The second death, in a patient with a tumefactive lesion awaiting biopsy, illustrates the diagnostic difficulty that tumefactive demyelination continues to pose. Both cases argue for a careful drug and substance history and for a low threshold for tissue diagnosis when the radiological picture is atypical.

Limitation(s)

This study has several limitations. The sample size of 55 is modest and the subgroups, particularly the multiple sclerosis group with five patients, are too small to support firm statistical inference; the comparisons presented should be regarded as descriptive. Antibody testing could not be completed in six patients because of cost and logistic constraints, so a proportion of the cohort remains serologically undefined and some AQP4 or MOG positive cases may have been misclassified. Cerebrospinal fluid oligoclonal band testing was not performed uniformly. Outcome was assessed only at discharge, so the analysis captures attack recovery rather than long-term disability or relapse risk, and two patients were lost to follow-up. Finally, being a single-centre study of admitted patients, the case mix is weighted towards severe disease and cannot be extrapolated to community prevalence.

CONCLUSION

In this cohort of 55 patients admitted with CNS demyelinating disease at a government tertiary care hospital in southern Tamil Nadu, AQP4-IgG seropositive NMOSD and MOGAD together accounted for approximately two-thirds of cases, while multiple sclerosis was distinctly uncommon. Myelitis with longitudinally extensive cord involvement was the dominant presentation, AQP4-IgG disease occurred exclusively in women, and visual evoked potentials detected subclinical optic nerve involvement in a useful proportion of patients. Despite severe disability at admission, most patients regained functional independence with intravenous methylprednisolone followed by early oral immunosuppression. These findings support a serology-guided but pragmatic treatment approach, in which mycophenolate mofetil serves as an affordable maintenance agent and rituximab is reserved for relapsing disease, and they underline the need for wider access to antibody testing in public-sector neurology practice in India.

Declaration by Authors

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