1. INTRODUCTION

- Inflammatory demyelinating diseases of the central nervous system (CNS) represent a complex group of disorders traditionally dominated by the diagnosis of Multiple Sclerosis (MS). However, the last two decades have witnessed a paradigm shift with the discovery of pathogenic autoantibodies, fundamentally reshaping the diagnostic landscape. Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is a demyelinating disorder of the CNS, which can result in subacute inflammation of the optic nerve (optic neuritis, ON), spinal cord (transverse myelitis, TM), and brain (acute disseminated encephalomyelitis, ADEM); as well as cortical, brainstem, cerebellar, and leptomeningeal inflammation(1). NMOSD is an inflammatory disease that primarily affects the optic nerve and spinal cord; the brainstem, specifically the area postrema, can also be involved (2).

- The objective of this study is to contribute to this body of knowledge by describing the clinical phenotypes, radiological patterns, laboratory findings, and treatment responses in a cohort of MOGAD and NMOSD patients managed at our tertiary care centre in Visakhapatnam, India.

2. METHODS

- **Study Design and Population** A retrospective, observational cohort study was conducted. The study population included all patients with a serologically confirmed diagnosis of MOGAD (MOG-IgG positive) or NMOSD (AQP4-IgG positive) who presented to the Department of Neurology at our institution. A total of 15 patients were identified and included in the analysis.

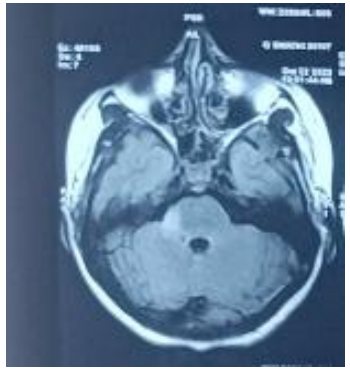
- **Data Collection and Variables** Data for each patient was meticulously extracted from the hospital's electronic medical records. The following variables were collected:

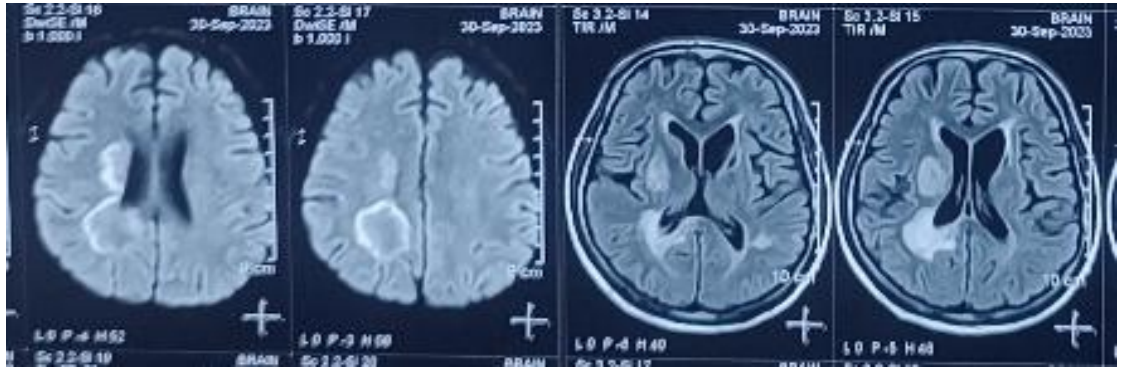
- **Demographics:** Age at onset, sex. **Clinical Presentation:** Primary neurological syndrome at first presentation (e.g., optic neuritis, transverse myelitis, brainstem syndrome).

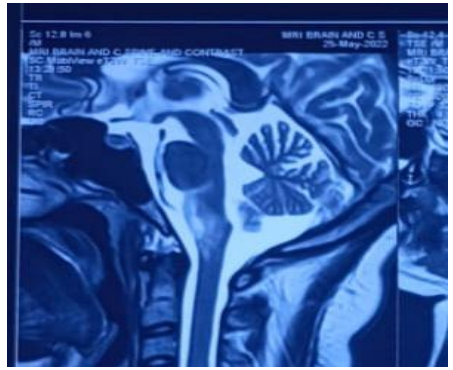
- **Disease Course:** Number of attacks (monophasic vs. relapsing). **Radiological Findings:** Key features from Magnetic Resonance Imaging (MRI) of the brain and spine. **Laboratory Findings:** Cerebrospinal fluid (CSF) analysis (cell count and protein levels) and Visual Evoked Potential (VEP) results.
- **Treatment Data:** Acute and long-term immunotherapies used (e.g., steroids, azathioprine, rituximab, plasma exchange).
- **Statistical Analysis** All data were analysed using descriptive statistics. Frequencies and percentages were calculated for categorical variables, while means and ranges were determined for continuous variables.

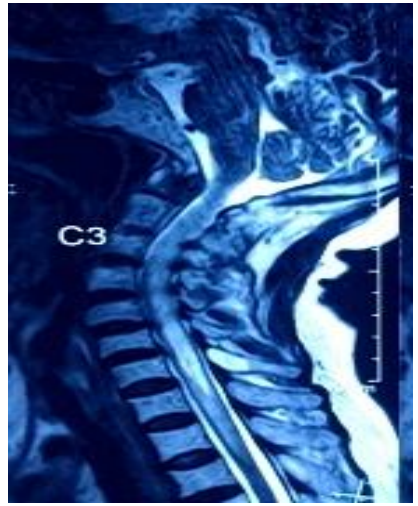
3. Results

- **Cohort Demographics and Serology** The final cohort consisted of 15 patients. The serological analysis revealed a striking predominance of MOGAD, which was diagnosed in 13 patients (87%). NMOSD was diagnosed in the remaining 2 patients (13%).
- The cohort had a female preponderance, with 10 female patients (67%) and 5 male patients (33%). The age of disease onset varied widely, with the youngest patient being 14 years old and the oldest being 70, yielding a mean age of onset of 37.5 years.
- **Clinical Phenotypes at Presentation**
- **Optic Neuritis (ON):** This was the most frequent presenting syndrome, occurring in 8 of 15 patients (53%). It was a common feature in the MOGAD subgroup.
- **Transverse Myelitis (TM):** TM was the presenting feature in 5 of 15 patients (33%). Critically, this presentation was often severe, with 4 of these patients demonstrating a longitudinally extensive transverse myelitis (LETM) on spinal MRI, leading to significant motor deficits such as paraplegia and urinary retention.
- **Brainstem & Encephalic Syndromes:** Six patients (40%) presented with syndromes related to brainstem or cerebral involvement. This included ataxia associated with Middle Cerebellar Peduncle (MCP) lesions in MOGAD patients and classic Area Postrema Syndrome in an NMOSD patient.
- **Paraclinical** and Treatment Data CSF analysis showed considerable variability. While some patients had significant pleocytosis (up to 90% lymphocytes) and elevated protein (up to 133 mg/dL), others had near-normal or benign CSF profiles. A relapsing disease course was observed in 4 of 15 patients (27%). All patients received high-dose intravenous steroids for acute attacks. Longer-term immunosuppression with agents such as azathioprine and rituximab was required in patients with a relapsing course or severe disease.









DISCUSSION

- This study provides valuable insights into the presentation of MOGAD and NMOSD in a Southern Indian cohort. The most significant finding is the pronounced predominance of MOGAD (87%) over NMOSD. This observation, while based on a small sample, raises important questions about the regional prevalence of these disorders and warrants further investigation in larger,multi-center studies.
- Our findings confirm the highly pleomorphic nature of MOGAD. The spectrum of disease in our cohort ranged from classic, steroid-responsive optic neuritis with excellent outcomes to severe, disabling LETM causing permanent neurological deficits. This challenges any perception of MOGAD as an invariably "mild" disorder and highlights that clinical severity at onset cannot reliably distinguish it from NMOSD.
- While certain radiological clues were observed, such as MCP involvement in a MOGAD patient, the significant overlap in major findings like LETM reinforces the limitations of relying on imaging alone for diagnosis. The variability in CSF findings further supports this conclusion.
- The limitations of our study include its retrospective design, the small sample size, and the potential for referral bias inherent to a single-center study at a tertiary care facility.

CONCLUSION

- In our cohort of patients with inflammatory demyelinating disease, MOGAD was the overwhelmingly predominant diagnosis. The clinical expression of MOGAD was highly variable, spanning a wide spectrum of severity. Given the significant overlap in clinical and radiological features with NMOSD, our study concludes that early, specific, and accurate serological testing is an indispensable and imperative step in the diagnostic pathway. A confirmed serological diagnosis is the cornerstone upon which to build an appropriate therapeutic strategy aimed at preventing long-term disability.

RECOMMENDATIONS FOR FUTURE DIRECTIONS

- Establish a Prospective Registry: A prospective, multi-center registry should be established in India to better understand the natural history, true prevalence, and long-term treatment outcomes for MOGAD and NMOSD.
- Standardize Protocols: Develop standardized diagnostic and treatment protocols to harmonize care for these patients across different centers.
- Investigate Regional Factors: Future research should explore potential genetic, ethnic, or environmental factors that may contribute

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2. Okada K, Kobata M, Naruke S. Neuromyelitis optica spectrum disorder with area postrema syndrome. Neurol Clin Pract. 2019 Apr;9(2):173-175. [PMC free article] [PubMed]