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Neurophysiological Insights and Successful Aggressive Immunomodulation in Severe MOG-Associated Acute Disseminated Encephalomyelitis With Fulminant Bulbar Palsy: A Case Report

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Abstract

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) frequently presents as acute disseminated encephalomyelitis (ADEM) in pediatric and adolescent populations. While often steroid-responsive, severe cases with fulminant bulbar palsy and respiratory failure are rare and diagnostically challenging.

We report a 15-year-old female patient who presented with acute quadriparesis, urinary retention, and rapidly progressive bulbar palsy requiring mechanical ventilation.

Extensive laboratory evaluation, including cerebrospinal fluid (CSF) analysis and serum MOG-IgG testing using a live cell-based assay, was positive at a titer of 1:80, while cerebrospinal fluid oligoclonal bands were absent. Neurophysiological testing demonstrated absent F-wave responses in all tested nerves despite preserved distal motor and sensory conduction, indicating proximal motor pathway dysfunction.

The patient was successfully managed with a stepwise escalation of immunomodulatory therapy - including pulse corticosteroids, therapeutic plasma exchange (PLEX), intravenous immunoglobulin (IVIG), and rituximab - ultimately leading to complete respiratory weaning and decannulation.

This case underscores the diagnostic utility of early neurophysiological testing in predicting the severity of demyelination in MOG-ADEM. It highlights the efficacy of aggressive, sequential immunotherapy in achieving functional recovery in fulminant presentations.

Categories: Neurology, Internal Medicine, Allergy/Immunology
Keywords: acute disseminated encephalomyelitis, aggressive immunomodulation, bulbar palsy, electromyography, intravenous immunoglobulin, mogad, mog antibody disease, nerve conduction study, neurophysiology, plasma exchange

Introduction

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an antibody-mediated inflammatory demyelinating disorder of the central nervous system (CNS) that has emerged as a distinct clinical entity, separate from neuromyelitis optica spectrum disorders (NMOSD) and multiple sclerosis (MS) [1].

Acute disseminated encephalomyelitis (ADEM) is an inflammatory demyelinating disorder of the CNS and is a recognized clinical presentation of MOGAD, particularly in the pediatric population. MOGAD has an estimated prevalence of approximately one in 10,000 individuals and is more frequently reported in children [2].

Acute MOGAD attacks generally respond to high-dose corticosteroids; however, some patients develop severe neurologic involvement or show an inadequate response to initial treatment. In such cases, escalation of acute therapy with intravenous immunoglobulin (IVIG) or therapeutic plasma exchange (PLEX). In patients requiring long-term treatment, maintenance therapies may include steroid-sparing agents, including azathioprine and mycophenolate mofetil, as well as IVIG and rituximab, have been used. Careful assessment of the clinical course, serologic findings, and neuroimaging is important for distinguishing MOGAD from other inflammatory demyelinating disorders and identifying patients who may require treatment escalation [3].

Fulminant bulbar dysfunction with respiratory failure represents a severe and clinically significant

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manifestation of MOGAD [3]. In the present case, the coexistence of extensive myelitis and absent F-wave responses raised uncertainty regarding whether the neurophysiological abnormalities reflected proximal peripheral involvement or dysfunction related to spinal cord disease.

We present the case of a 15-year-old female patient with MOGAD presenting as ADEM who was successfully managed with a stepwise escalation of immunomodulatory therapy, including pulse corticosteroids, PLEX, IVIG, and rituximab, followed by successful weaning from mechanical ventilation and eventual clinical recovery.

Case Presentation

A 15-year-old female patient presented with a three-day history of non-specific febrile illness, rapidly followed by acute urinary retention and progressive ascending quadriparesis, with lower extremity weakness disproportionately more severe than upper extremity impairment.

On admission, the patient had a GCS of 15/15, a temperature of 99.6°F, a heart rate of 70/min with well-felt peripheral pulses and CRT <3 seconds, a respiratory rate of 27/min on room air, and an SpO_2 of 98% on room air.

Neurological examination demonstrated progressive ascending quadriparesis, with lower-extremity weakness more pronounced than upper-extremity weakness, accompanied by acute urinary retention. Within 24 hours, neurological deterioration was marked by profound dysphagia, an absent gag reflex, and severe phonation impairment, necessitating intubation and mechanical ventilation. Detailed documentation of muscle strength grading, tone, deep tendon reflexes, plantar responses, and sensory examination was not available in the medical records reviewed.

The baseline investigations done on admission are summarized in Table *1*.

Investigation	Patient result	Reference range
Hemoglobin	13 g/dL	12–16 g/dL
Total leukocyte count	18,900 cells/mm³	4,000–11,000 cells/mm³
Platelet count	4.08 × 10^5/mm³	1.5–4.0 × 10^5/mm³
MCV	79.8 fL	80–100 fL
MCH	27.3 pg	27–32 pg
MCHC	34.3 g/dL	32–36 g/dL
Sodium	141 mmol/L	135–145 mmol/L
Potassium	3.7 mmol/L	3.5–5.1 mmol/L
Chloride	110 mmol/L	98–107 mmol/L
Serum creatinine	0.41 mg/dL	0.5–1.0 mg/dL
Serum urea	27 mg/dL	15–45 mg/dL
Total protein	7.7 g/dL	6.0–8.3 g/dL
Albumin	4.6 g/dL	3.5–5.0 g/dL
Total bilirubin	0.25 mg/dL	0.2–1.2 mg/dL
Direct bilirubin	0.13 mg/dL	0–0.3 mg/dL
SGOT (AST)	11 U/L	5–40 U/L
SGPT (ALT)	22 U/L	7–56 U/L
ALP	185 U/L	44–147 U/L
Calcium	8 mg/dL	8.5–10.5 mg/dL
Magnesium	2.88 mg/dL	1.7–2.2 mg/dL
Phosphorus	6.4 mg/dL	2.5–4.5 mg/dL
Vitamin D3	5.5 ng/ml	30-100 ng/ml
PT	13.3 sec	~11–14 sec
aPTT	29.2 sec	~25–35 sec
INR	1.16	0.8–1.2

TABLE 1: Baseline laboratory investigations on admission.

ALP: alkaline phosphatase; ALT: alanine aminotransferase; aPTT: activated partial thromboplastin time; AST: aspartate aminotransferase; INR: international normalized ratio; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; MCV: mean corpuscular volume; PT: prothrombin time; SGOT: serum glutamic-oxaloacetic transaminase; SGPT: serum glutamic-pyruvic transaminase.

Within 24 hours of admission, her neurological status deteriorated precipitously, manifesting as fulminant bulbar palsy with profound dysphagia, an absent gag reflex, and severe phonation impairment, necessitating emergent intubation and mechanical ventilation.

Initial cerebrospinal fluid analysis revealed a non-inflammatory profile with minimal lymphocytic pleocytosis (2 cells/µL), no red blood cells, and normal glucose and protein levels.

Infectious panels, including Enterovirus PCR and routine cultures, were negative. Immunological testing demonstrated serum MOG-IgG positivity at a titer of 1:80 using a live cell-based assay; AQP4/NMO-IgG was negative, and cerebrospinal fluid oligoclonal bands were absent. Neurophysiological evaluation demonstrated preserved compound motor action potential and sensory nerve action potential amplitudes and conduction velocities across the bilateral tibial, peroneal, median, and ulnar nerves, while F-wave responses were absent in all tested nerves.

The key laboratory findings are summarized in Table 2.

Investigation	Patient result	Reference/expected result
CSF white blood cell count	2 cells/μL	0–5 cells/μL
CSF red blood cell count	0 cells/μL	0 cells/μL
CSF glucose	Normal	40–70 mg/dL
CSF protein	Normal	15–45 mg/dL
Enterovirus PCR	Negative	Negative
Routine bacterial cultures	No growth	No growth
Serum MOG-IgG antibody (live cell-based assay)	Positive, titer 1:80	Negative
Aquaporin-4 (AQP4/NMO-IgG) antibody	Negative	Negative
Oligoclonal bands	Absent	Absent
CMAP	Normal amplitudes and conduction velocities in bilateral tibial, peroneal, median, and ulnar nerves	Normal
SNAP	Normal amplitudes and conduction velocities in bilateral tibial, peroneal, median, and ulnar nerves	Normal
Minimum F-wave latencies	Absent in all tested nerves	Present

TABLE 2: Summary of key laboratory investigations at presentation.

AQP4, aquaporin-4; CMAP, compound motor action potential; CSF, cerebrospinal fluid; F-wave, late motor response; MOG-IgG, myelin oligodendrocyte glycoprotein immunoglobulin G; NMO-IgG, neuromyelitis optica immunoglobulin G; PCR, polymerase chain reaction; SNAP, sensory nerve action potential.

Additionally, brain and spinal magnetic resonance imaging (MRI) were performed, which demonstrated bilateral T2 hyperintense lesions involving the centrum semiovale and corona radiata (Figure 1A-1C).

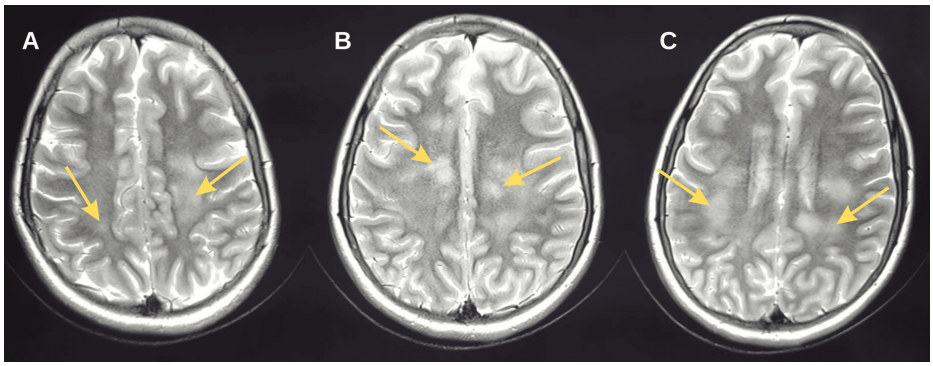


FIGURE 1: Axial T2-weighted brain magnetic resonance imaging.

(A-C): Serial axial images demonstrate bilateral T2-hyperintense lesions involving the centrum semiovale and corona radiata (arrows indicate the lesions).

Diffusion-weighted imaging revealed multifocal diffusion-restricting lesions in the bilateral centrum semiovale, corona radiata, and splenium of the corpus callosum (Figure 2A-2C).

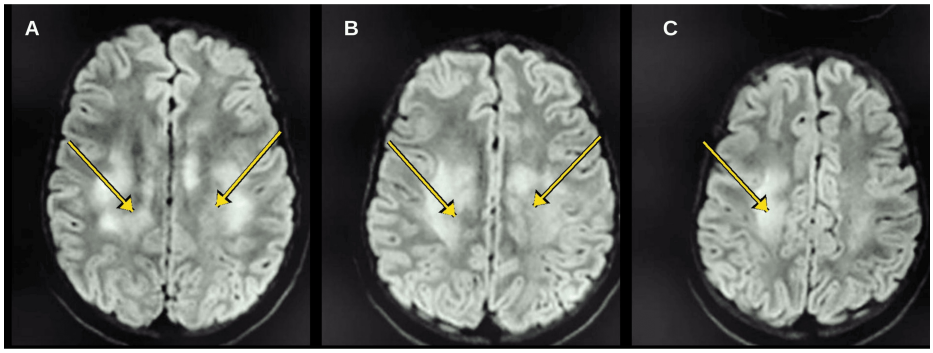


FIGURE 2: Axial diffusion-weighted brain magnetic resonance imaging.

(A-C): Serial axial images demonstrate multifocal hyperintense lesions involving the bilateral centrum semiovale and corona radiata (arrows).

Additional fluid-attenuated inversion recovery (FLAIR) imaging findings, as documented in the original radiological interpretation, included signal abnormalities involving the right thalamocapsular region and right putamen. Thickening of the right optic nerve was also noted (Figure *3*). The absence of dedicated orbital MRI sequences limited further characterization of the optic nerve abnormality.

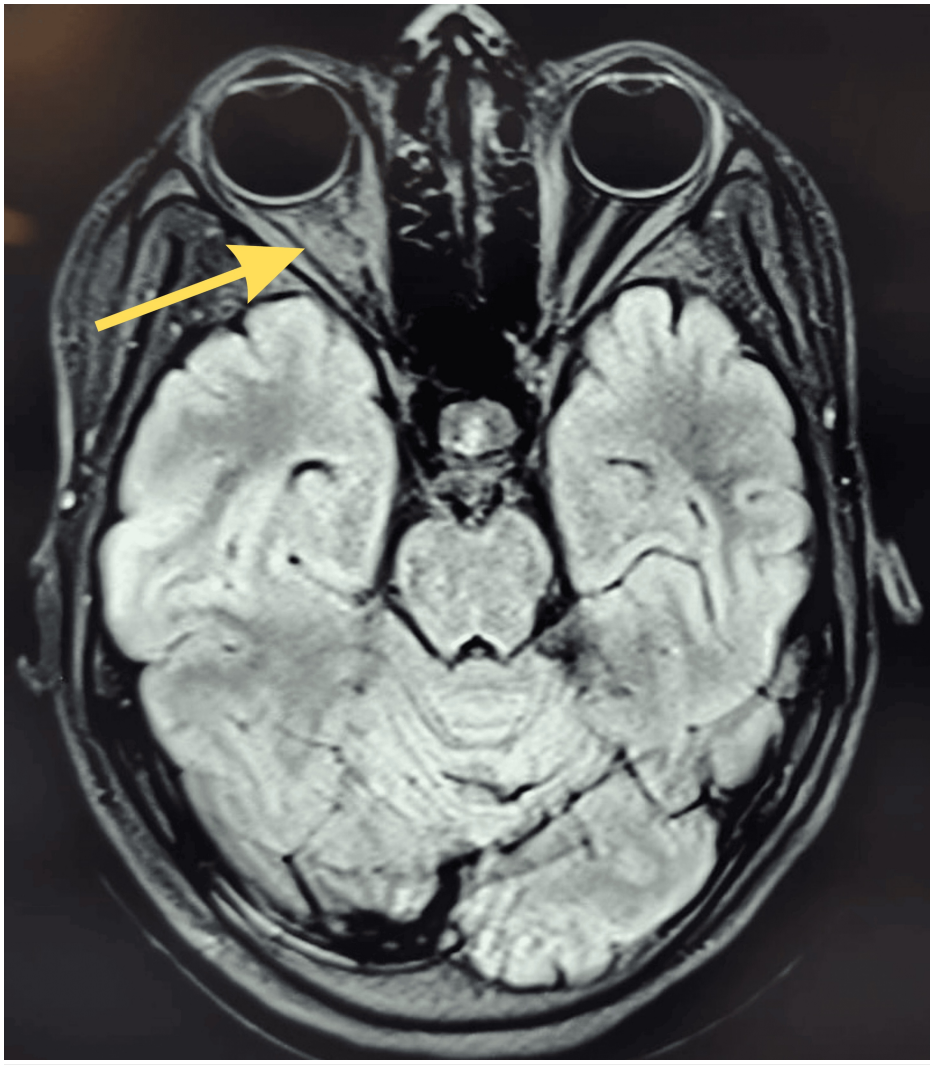


FIGURE 3: Axial FLAIR brain MRI demonstrating thickening of the right optic nerve (arrow).

FLAIR: fluid-attenuated inversion recovery.

Spinal MRI showed long-segment T2/STIR hyperintensities extending throughout the cervical, thoracic, and lumbar spinal cord to the conus medullaris at the L1 level (Figure *4A*-*4B*).

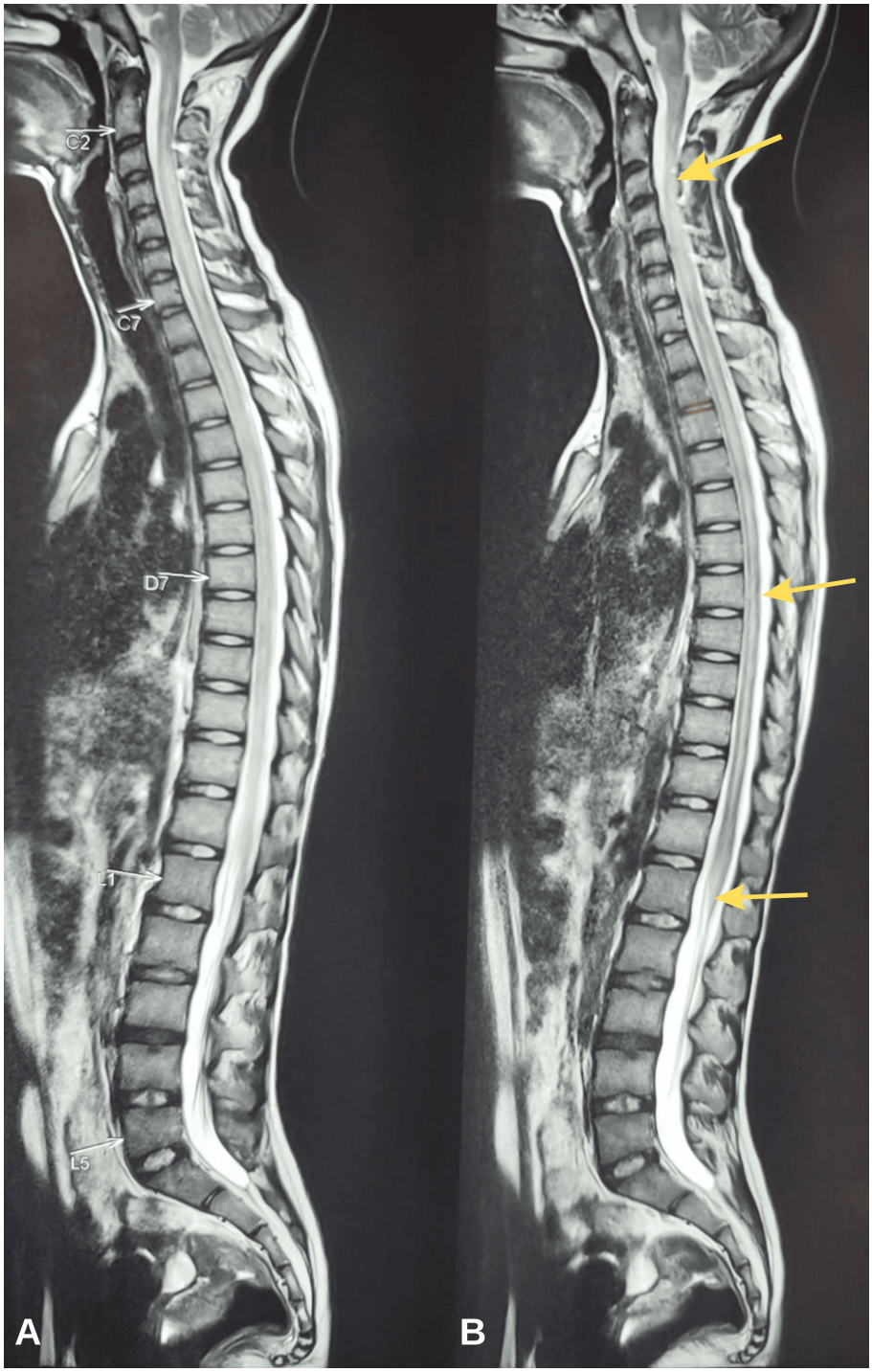


FIGURE 4: Sagittal T2/STIR spinal magnetic resonance imaging.

(A-B): Serial sagittal images demonstrate longitudinally extensive intramedullary hyperintense signal involving the cervical and thoracic spinal cord and extending to the conus medullaris at the L1 level (arrows indicate representative areas of abnormal intramedullary signal).

STIR: short tau inversion recovery.

Given the severity of the bulbar dysfunction, the patient required a tracheostomy on hospital day 12 for prolonged mechanical ventilation. We initiated a rapid, stepwise immunomodulatory regimen comprising intravenous methylprednisolone pulse therapy (30 mg/kg/day), five cycles of therapeutic plasma exchange (PLEX), and sequential intravenous immunoglobulin (IVIG).

During this therapeutic cascade, the patient developed a catheter-associated Enterobacter cloacae urinary tract infection, which was successfully treated with IV meropenem before the initiation of maintenance

therapy with intravenous rituximab (375 mg/m²). Table 3 summarizes the patient's clinical course and treatment timeline throughout hospitalization and follow-up.

Hospital day	Clinical course/treatment
Day 0	Admitted with acute urinary retention and progressive ascending quadriparesis. Serum MOG-IgG/AQP4 testing was obtained. IV methylprednisolone 30 mg/kg/day was initiated.
Day 1	Developed rapidly progressive bulbar dysfunction with respiratory failure, requiring intubation and mechanical ventilation. IV methylprednisolone was continued.
Days 1–5	Completed 5-day pulse IV methylprednisolone course.
Day 2	Due to severe neurological involvement, therapeutic plasma exchange (PLEX) was initiated.
Days 2, 4, 6, 8, and 10	Five PLEX sessions were completed on alternate days.
Days 11–15	After completion of the fifth PLEX session, IVIG (total 2 g/kg) was administered over five days. No further PLEX was performed after IVIG.
Day 12	Tracheostomy was performed because of prolonged mechanical ventilation.
During hospitalization	Developed a catheter-associated Enterobacter cloacae urinary tract infection, which was treated with IV meropenem before further immunosuppression.
~Day 16 onward, after infection control	Rituximab 375 mg/m² was administered as further immunomodulatory therapy.
Following weeks	Serial laryngoscopy and FEES demonstrated recovery of bulbar function. The patient was progressively weaned from mechanical ventilation.
~1 month after tracheostomy	Successfully decannulated and weaned from mechanical ventilation.
6-month follow-up	Remained clinically stable without further demyelinating events. No scheduled maintenance immunotherapy was prescribed at discharge; pediatric neurology follow-up continued for relapse surveillance.

TABLE 3: Clinical course and treatment timeline during hospital admission and follow-up.

AQP4, aquaporin-4; FEES, fiberoptic endoscopic evaluation of swallowing; IV, intravenous; IVIG, intravenous immunoglobulin; MOG-IgG, myelin oligodendrocyte glycoprotein immunoglobulin G; PLEX, plasma exchange.

Neurological recovery followed this aggressive immunomodulatory approach. Serial flexible laryngoscopy and fiberoptic endoscopic evaluation of swallowing (FEES) were utilized to track functional bulbar integrity objectively. Laryngoscopy confirmed the restoration of bilateral vocal cord mobility, laryngeal sensation, and soft palate elevation, while FEES demonstrated the absence of penetration or aspiration across all food and liquid consistencies. Guided by these functional milestones, the patient was successfully weaned from mechanical ventilation and decannulated one month post-tracheostomy, maintaining 99% oxygen saturation on room air at discharge.

At six-month follow-up, the patient remained clinically stable without further demyelinating events. She continued follow-up with pediatric neurology, with ongoing assessment for relapse and consideration of further maintenance immunotherapy. No maintenance therapy was given at discharge.

Discussion

MOGAD encompasses a clinically diverse array of demyelinating manifestations. While ADEM is the predominant phenotype in pediatric and adolescent cohorts, optic neuritis remains the most prominent clinical feature in adult populations [4].

Applying the 2023 international consensus criteria, patients with low-positive MOG-IgG results require at least one supporting clinical or radiological feature for a diagnosis of MOGAD [5]. In this patient, serum MOG-IgG was positive at a titer of 1:80 using a live cell-based assay. In the setting of AQP4-IgG negativity and supportive radiological features, including longitudinally extensive myelitis with conus involvement and deep gray matter involvement, the patient fulfilled the 2023 diagnostic criteria for MOGAD [1,5].

Despite being classically considered steroid-responsive, a significant subset of individuals follows a treatment-refractory or fulminant trajectory, frequently correlating with diminished functional outcomes [4].

The patient's precipitous clinical decline from a prodromal febrile illness to quadriparesis, neurogenic bladder, and subsequent fulminant bulbar palsy within 24 hours exemplifies the severe end of the disease spectrum. This underscores that low MOG-antibody titers do not preclude aggressive disease activity.

The neurophysiological data necessitate particular attention. F-wave responses were absent in all tested nerves despite preserved distal motor and sensory amplitudes and conduction velocities. Although absent F-waves may occur with proximal nerve-root dysfunction and are commonly described in peripheral demyelinating disorders, this finding is not specific for peripheral demyelination. In the setting of longitudinally extensive myelitis, anterior horn cell or spinal gray matter dysfunction and acute spinal cord shock may also contribute to F-wave abolition. Although MOG expression is primarily restricted to the central nervous system, emerging evidence documents combined central and peripheral demyelination (CCPD) in MOG-seropositive cases. Rinaldi et al. identified peripheral nervous system involvement in an MOGAD cohort where nerve conduction studies revealed conduction block and diminished F-wave persistence, indicating an acquired demyelinating process [6].

A review of MOG-associated CCPD further described peripheral neuropathies manifesting alongside classic CNS lesions, suggesting that antibody translocation across the blood-nerve barrier at the nerve-root transition zone may link these compartments [7].

Similarly, a case of multifocal motor neuropathy with MOG antibodies exhibited temporal dispersion and conduction block, leading the authors to propose MOG-IgG1 as a putative marker for combined demyelinating syndromes [8].

In our case, the absence of peripheral clinical symptoms and preserved distal nerve conduction findings makes a diagnosis of CCPD less probable. The absent F-wave responses may reflect either proximal peripheral involvement or dysfunction related to the extensive spinal cord disease. Therefore, these findings should be interpreted in conjunction with the clinical and radiological features rather than as an isolated marker of peripheral demyelination or disease severity.

Management guidelines, such as those from the Saudi expert panel, emphasize high-dose corticosteroids as primary therapy. However, in cases of inadequate clinical response, therapeutic plasma exchange (PLEX) is advocated, with five cycles often required for substantial recovery [9].

IVIG may be integrated into the regimen, though evidence for apheresis or IVIG in acute MOGAD episodes remains constrained to retrospective data [9].

Rituximab has proven effective as rescue therapy in pediatric ADEM cases failing standard acute treatments [10], paralleling the sequential immunomodulatory strategy that facilitated recovery in our patient.

Furthermore, tocilizumab may serve as a viable alternative for patients refractory to rituximab or those exhibiting high relapse frequency [9,11].

Functional recovery was objectively monitored through serial laryngoscopy and FEES, ensuring that decannulation and ventilator weaning were dictated by the resolution of aspiration risk rather than empirical timelines.

Cohort studies indicate that a higher relapse burden in MOGAD is associated with cumulative long-term disability, highlighting the necessity of proactive treatment to prevent recurrence [12].

Conclusions

This case highlights that MOGAD can manifest as a fulminant and life-threatening presentation, characterized by rapid progression to bulbar palsy and respiratory failure. It underscores the value of early neurophysiological assessment while emphasizing that F-wave abnormalities are not specific for proximal peripheral demyelination and should be interpreted in the context of the accompanying clinical and radiological findings, particularly in patients with extensive myelitis. The favorable outcome achieved through a stepwise, aggressive immunomodulatory approach, comprising pulse corticosteroids, PLEX, IVIG, and rituximab, suggests that intensive, multimodal intervention may contribute to recovery even in severe or refractory presentations. This case emphasizes the importance of integrating clinical, radiological, and neurophysiological findings when evaluating severe MOGAD presentations.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Acquisition, analysis, or interpretation of data: Kaandeeban Mohanraj, Vedant Bawa, Sakshi Taori, Naveen Punchayil Narayanankutty

Drafting of the manuscript: Kaandeeban Mohanraj, Sakshi Taori

Critical review of the manuscript for important intellectual content: Kaandeeban Mohanraj, Vedant Bawa, Sakshi Taori, Naveen Punchayil Narayanankutty

Concept and design: Sakshi Taori

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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