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Case Report

Paediatrics Section

# Myelin Oligodendrocyte Glycoprotein Antibody Associated Acute Disseminated Encephalomyelitis Post Varicella Infection in a Child: A Case Report

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## ABSTRACT

Myelin Oligodendrocyte Glycoprotein Antibody associated Disease (MOGAD), an autoimmune demyelinating disorder, often overlaps with features of Acute Disseminated Encephalomyelitis (ADEM), optic neuritis and transverse myelitis in paediatric population. Recent advances have identified Myelin Oligodendrocyte Glycoprotein (MOG) antibody as a marker in a subset of paediatric demyelinating diseases. MOGAD is increasingly being recognised as a distinct entity among paediatric demyelinating disorders. Identification of MOG antibodies is important as it has diagnostic, therapeutic and prognostic implications particularly in post-infectious demyelination. Herein, the authors report a case of 10-year-old male who presented with fever and vesicular lesions for 10 days, suggesting chickenpox infection (varicella), which was followed by paraparesis. Neurological examination suggested hypotonia of lower limbs and absent deep tendon reflexes with rest of central nervous examination within normal limits. Radiological and biochemical investigations (serum MOG positive) confirmed the diagnosis of ADEM likely to be post varicella infection. He was managed successfully with intravenous immunoglobulin, high dose steroids and is currently doing well on follow-up. This case highlights the importance of early detection, diagnosis and testing for anti-MOG in children presenting with ADEM like features. Early recognition of MOG antibody associated ADEM and timely initiation of immunotherapy are crucial for improving neurological outcomes in affected children. This case emphasises the need to consider MOGAD in children presenting with acute demyelination after varicella.

**Keywords:** Chicken pox, Demyelinating, Hyperintensity, Optic neuritis, Paraparesis

## CASE REPORT

A 10-year-old previously healthy male child presented to the paediatric outpatient department with fever and vesicular skin lesions over the face which later spread to trunk and extremities over the next 10 days. The fever was documented, high grade, with two-three spikes per day. The rash resolved with crusting of lesions approximately in 10 days, consistent with diagnosis of chickenpox. Two days after resolution of the rash he developed fever followed by weakness in bilateral lower limbs, inability to walk and not passing urine since morning prior to admission. There was no family history of neurological disorder, no prior hospital admissions.

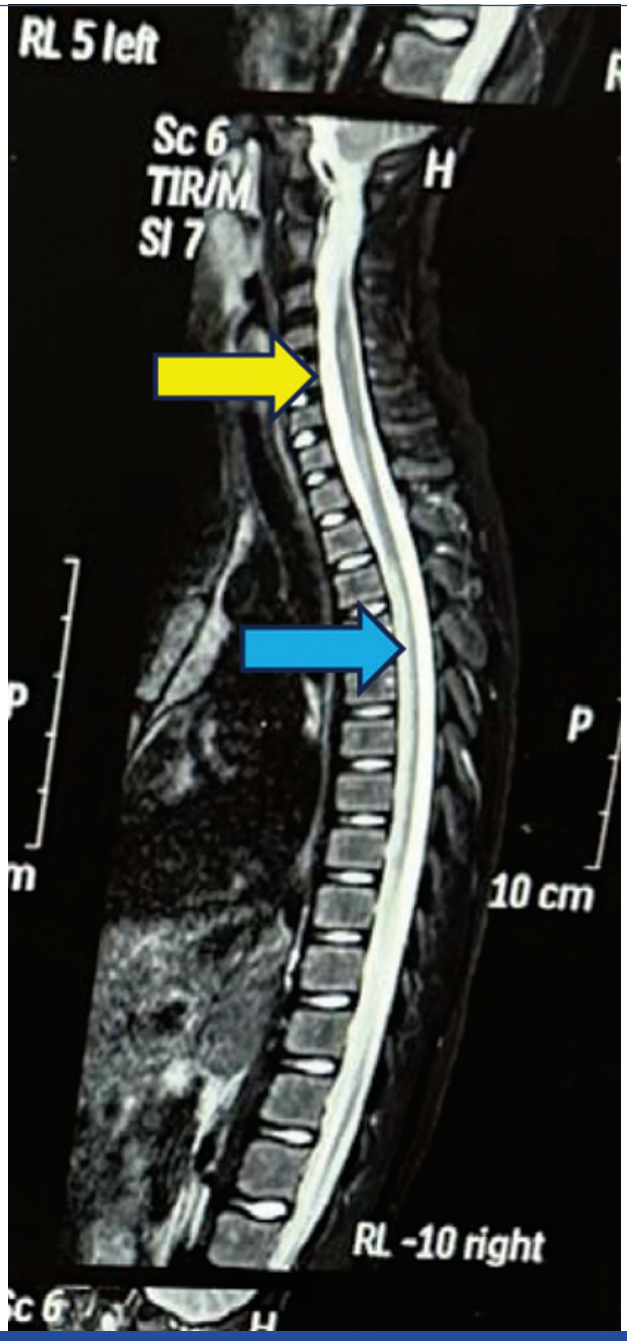
On physical examination, the child was febrile (102°F), and blood pressure, respiratory rate, pulse rate were normal. A detailed neurological examination revealed hypotonia and weakness in both lower limbs. Muscle power was graded 2/5 bilaterally. Deep tendon reflexes, including the knee and ankle jerks, were absent, and the Babinski sign was negative. There was no history of unconsciousness, abnormal body movements, no cranial nerve palsy or eye movement abnormalities. Upper limb power and deep tendon reflexes were normal. Rest of the systemic examination including cardiovascular, respiratory and abdominal system was normal. Considering above findings, a provisional diagnosis of post infectious acute transverse myelitis was kept.

The child was started on intravenous fluids (as per the Holliday–Segar formula), along with intravenous antibiotics—ceftriaxone (100 mg/kg/day) and vancomycin (80 mg/kg/day) for 10 days—and acyclovir (20 mg/kg/dose) administered eight-hourly. He was catheterised for monitoring of urinary output and investigations were planned. Routine investigations sent, were within normal limits [Table/Fig-1]. Neuroimaging by contrast enhanced MRI Brain and spine showed short segment T2 hyperintense signal in cervical cord from C5-C7 levels, long segment T2 hyperintense signal in thoracic cord post contrast enhancement and subtle white matter hyperintensities in pons and midbrain, indicating features of ADEM [Table/Fig-2]. Cerebrospinal Fluid (CSF) analysis revealed protein 47 mg/dL, glucose 62 mg/dL and no pleocytosis or oligoclonal bands. Serum MOG-IgG antibodies were positive by a cell-based immunofluorescence assay. CSF analysis showed no oligoclonal bands thereby ruling out Multiple Sclerosis (MS). Additionally Visual Evoked Potential (VEP) was normal suggesting no evidence of optic neuritis. The pattern of weakness ruled out Guillain Barre syndrome and preserved consciousness ruled out the possibility of viral encephalitis. Based on above findings, a diagnosis of post-chickenpox ADEM associated with MOG antibody positivity was made.

| Test | Values | Normal range |
|---|---|---|
| Haemoglobin (g/dL) | 12 | 12.5-16.1 |
| TLC (cells/µL) | 13700 | 4000-10500 |
| Platelet count (lacs) | 4.5 | 1.5-4 |
| Blood urea (mg/dL) | 24 | 15-45 |
| Serum creatinine (mg/dL) | 0.6 | 0.3-0.88 |
| SGOT (U/L) | 24 | 10-40 |
| SGPT (U/L) | 20 | 10-40 |
| S.ALP (U/L) | 116 | 150-560 |
| S.Ca (mg/dL) | 10.2 | 8.8-10.8 |
| S.Phosphorus (mg/dL) | 5.6 | 3.7-5.6 |
| S.Sodium (mEq/L) | 136 | 135-145 |
| S.Potassium (mEq/L) | 3.7 | 3.5-4.6 |
| CSF Protein (mg/dL) | 47 | 20-45 |

| | | |
|---|---|---|
| CSF Sugar (mg/dL) | 62 | >75% of serum glucose |
| CSF Cytology | Acellular | <5 cells |
| CSF culture | Sterile | No growth/ sterile |
| CSF Neuro-viral panel | Negative | Negative |
| ESR (mm/hr) | 22 | <20 |
| Serum NMO MOG panel | Anti MOG IgG antibody positive | |
| Anti Aquaporin-4 (NMO) IgG Antibodies | Negative | |

**[Table/Fig-1]:** Summary of investigations.




**[Table/Fig-2]:** Contrast Enhanced MRI Brain and spine showing (yellow arrow) short segment T2 hyperintense signal in cervical cord from C5-C7 levels, long segment (blue arrow) T2 hyperintense signal in thoracic cord post-contrast enhancement.

The patient was also managed with Intravenous Immunoglobulin (IVIG) (@2 g/kg) over five days and high dose intravenous methylprednisolone (@30 mg/kg/day) for five days followed by an oral steroids (2 mg/kg/day) to be tapered over six to eight weeks. The patient showed significant improvement during hospital stay with gradual recovery of power and bladder control. He was discharged after 14 days of hospitalisation and was followed up at three months post discharge, when he remained asymptomatic with no residual neurologic deficits.

## DISCUSSION

The ADEM is an inflammatory demyelinating event, typically monophasic, occurring predominantly in children preceded by viral infections such as varicella, measles mumps, rubella. Approx 50% of ADEM cases show MOG-IgG positivity. While varicella (chickenpox) infection in children is a recognised trigger for ADEM, neurological complications occur in less than 1% of varicella cases, post-varicella MOG-IgG-associated ADEM remains exceedingly rare with an incidence of 0.5-1 per 100,000 population [1-3]. Post-infectious immune activation is considered the key mechanism, where molecular mimicry between viral antigens and myelin components leads to an aberrant immune response [4].

Bhandari S et al., reported a case post-chickenpox MOG antibody–associated ADEM presenting with quadriparesis following varicella infection, similar to the present case where neurological symptoms followed a short latent period after chickenpox. However, their patient had involvement of all four limbs, whereas the present case predominantly involved the lower limbs with bladder dysfunction [5]. Yadav A et al., reported a teenage patient who developed ADEM following varicella infection with positive MOG antibodies, who showed diffuse white matter changes on MRI and responded well to corticosteroid therapy. Similar MRI features and favourable response to immunotherapy were observed in the present case, supporting the monophasic nature of post-infectious MOGAD [6]. Hancock KAM et al., reported a case of a six-year-old boy who developed MOG-antibody (MOG-Ab) associated demyelinating syndrome, after initially presenting with aseptic meningitis. In contrast, the present case did not show meningeal features but presented with acute myelopathy, highlighting the phenotypic heterogeneity of paediatric MOGAD despite a shared autoimmune mechanism [7]. In the present case, the temporal relationship between varicella infection and neurological symptoms, along with positive anti-MOG antibodies, supports a diagnosis of post-varicella MOGAD, a rare entity [8]. MRI findings were consistent with ADEM showing diffuse bilateral white matter involvement. The absence of CSF oligoclonal bands in this patient further supports MOGAD rather than MS, since OCBs are typically negative or transient in MOGAD (<30%) but persistently positive in MS (>90%) [9,10].

Similarly, normal VEP ruled out optic neuritis, which is another common phenotype of MOGAD but was not evident in this case. In the present case, the patient responded well to IVIG and corticosteroids with near-complete recovery and no relapse on follow-up, suggesting a monophasic course. Treatment of MOGAD includes high-dose intravenous methylprednisolone for 3-5 days followed by a slow oral steroid taper over 6-8 weeks [11]. IVIg or plasmapheresis is considered in steroid-refractory case [12]. Long-term prognosis in paediatric MOGAD is generally favourable; however, 20-30% of patients may experience relapses, particularly when steroids are tapered rapidly or discontinued early [13]. Persistent antibody positivity may predict higher relapse risk, emphasising the need for close follow-up [14]. The present case highlights the importance of considering MOGAD in the differential diagnosis of demyelinating syndromes following viral infections such as varicella. Early recognition and immunotherapy are crucial for preventing permanent neurological sequelae. Given the overlap between post-infectious ADEM and MOGAD, testing for anti-MOG antibodies is essential in all children presenting with ADEM-like features.

## CONCLUSION(S)

Authors report a case of MOG-antibody-associated ADEM following chickenpox in a child. This reinforces the need for MOG-IgG testing in post-viral demyelinating syndromes and highlights chickenpox as a potential autoimmune trigger. The presence of serum MOG-IgG supports classification within the MOG-associated demyelination spectrum, which presents with an ADEM phenotype in the paediatric

population. In this case the child demonstrated multifocal, bilateral white-matter involvement and concurrent spinal cord lesions following a recent viral illness. Early initiation of immunotherapy led to clinical improvement, highlighting the favourable outcome typically seen in paediatric MOG-associated ADEM when promptly treated. This case emphasises the need to consider MOGAD in children presenting with acute demyelination after varicella.

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