Int. J Med. Pharm. Res. (P-ISSN: 2958-3675 | E-ISSN: 2958-3683)
Available on: https://ijmpr.in/

International Journal
of Medical and Pharmaceutical Research

CASE REPORT **OPEN ACCESS**

Unravelling the Mimicker of Autoimmune Disease: Myelin Oligodendrocyte Glycoprotein (MOG)-Associated Demyelinating Disease in Males

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Received: 13-01-2025
Accepted: 15-02-2025
Available online: 23-02-2025

A B S T R A C T

Myelin oligodendrocyte glycoprotein (MOG), a member of the immunoglobulin (Ig) superfamily, is a protein found exclusively on the outer surface of myelin sheaths and oligodendrocyte membranes. Because of this specific localization, MOG can become a target for both cellular and humoral immune responses in inflammatory demyelinating diseases. Its expression occurs later in postnatal development, making it a key marker for the maturation of oligodendrocytes. Autoantibodies targeting myelin oligodendrocyte glycoprotein (MOG) can result in various autoimmune disorders, including optic neuritis, transverse myelitis, brainstem encephalitis, and acute disseminated encephalomyelitis.In this case report we present a 19 year old male patient presented with complaints of bilateral lower limb numbness with acute urine retention since 1 day.

Keywords: MOG- Myelin Oligodendrocyte Glycoprotein. MOGAD- MOG antibody-associated disease Ig- Immunoglobulin.

INTRODUCTION

Autoimmune diseases often present with a wide range of clinical manifestations, making diagnosis a challenging and complex process. Among these, demyelinating diseases such as multiple sclerosis (MS) and other central nervous system (CNS) disorders are frequently misdiagnosed, as their symptoms can overlap with those of infectious, metabolic, or genetic conditions. One such emerging entity is Myelin Oligodendrocyte Glycoprotein (MOG)-associated demyelinating disease (MOGAD), a condition characterized by the presence of autoantibodies targeting MOG, a protein found on the surface of myelin sheaths and oligodendrocytes. While MOGAD can present similarly to classical autoimmune disorders like MS, it has distinct clinical and radiological features, making it a significant mimicker in the differential diagnosis.

MOG antibody-associated disease (MOGAD) primarily impacts the central nervous system, causing a range of neurological deficits such as optic neuritis, transverse myelitis, encephalitis, and acute disseminated encephalomyelitis (ADEM). While it affects both genders, recent research suggests that certain subtypes of MOGAD may occur more frequently or present differently in male patients. This case report examines the diagnosis and clinical progression of MOGAD in a male patient, emphasizing the need to consider MOG antibodies as a potential cause of inflammatory demyelinating diseases, particularly when an autoimmune origin is suspected.

The growing recognition of MOGAD as a distinct disease entity is critical, as it has implications for management and treatment, which may differ from those of other autoimmune demyelinating diseases. Early identification and accurate diagnosis are essential for ensuring appropriate therapeutic interventions, preventing long-term disability, and improving patient outcomes. This report aims to contribute to the understanding of MOGAD as a mimicker of autoimmune diseases, with a focus on its presentation in male patients and the diagnostic approach required to differentiate it from other similar conditions.

METHODS

Study Design:A single-patient case study aimed at identifying and characterizing radiological features of MOG-associated demyelinating disease (MOGAD).

MRI scans were done using standardized protocols:

- Brain MRI: Include T1-weighted, T2-weighted, FLAIR, and post-contrast sequences.
- Spinal MRI: Assess cervical and thoracic spinal cord for longitudinally extensive transverse myelitis (LETM).
- Orbital MRI: Focus on optic nerve involvement, including enhancement and thickening.

Case Presentation- A 19 year old male presented with acute urine retention since 1 day.On CNS examination following were the findings-

Lower Limbs-

	Right side	Left side
Muscle power	5/5	5/5
Sensory system (Pain and temperature)	Decrease perception	Decrease perception
Deep tendon Reflex	Present	Present

Upper Limbs-

	Right side	Left side
Muscle power	5/5	5/5
Sensory system (Pain and temperature)	Present	Present
Deep tendon Reflex	Present	Present

Patient underwent Serology tests and following were the Lab findings-

Test	Result values	Normal Reference Interval
Haemoglobin	16	13-17 g/dL
Neutrophils	70	40-70 %
Lymphocytes	24	20-45 %
Eosionophils	01	0.0-6.0 %
Basophils	00	0.0-2.0%
Monocytes	05	0.0-10.0%
MCV	105	82- 98/ fL
MCH	37	26-34/pg
ESR	20	0.0-15 mm
Bilirubin Total	0.89	0.20-1.3 mg/dL
SGOT	78	17-59 U/L
SGPT	54	0-50 U/L
A/G Ratio	1.5	1.1- 2.5
Urea	21	20-43 mg/dl
Creatinine	0.7	0.6-1.25
Anti Nuclear Antibody by IFA	Negative	
NMO, Anti-Aquaporin -4 IgG	Negative	
Myelin Oligodendrocyte Glycoprotein (MOG) Antibodies	**Positive**	

Patient underwent MRI Brain with Contrast Enhanced – Dorso Lumbar spine with Whole spine screening.

Following were the findings-

MRI Findings of Spine reveals H-shaped hyperintensity involving grey matter of spinal cord from D12 to L3 vertebral levels.

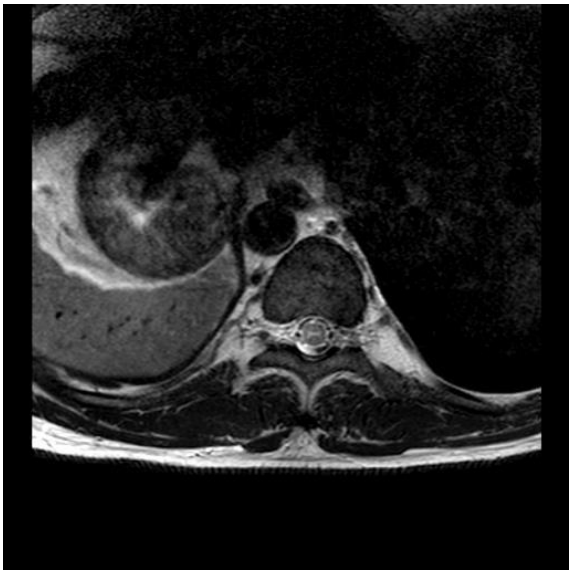

Figure 1Axial T2 Weighted image of spine showing hyper intensity within the cord in the Dorso- Lumbar region.

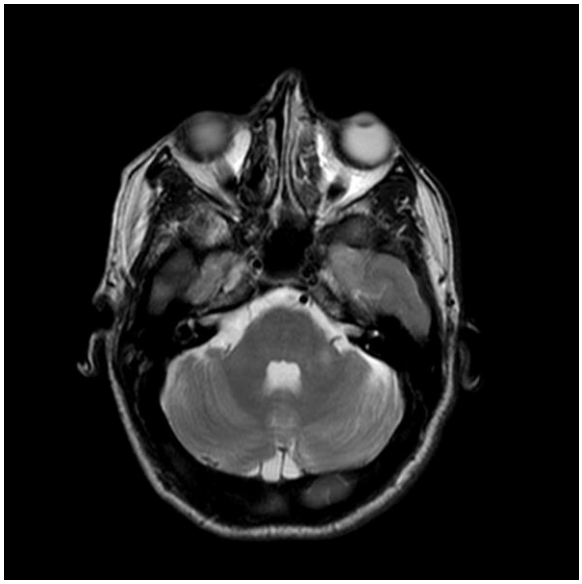

Figure 2 AXIAL T2 weighted sequence of Brain showing hyper intensity in left cerebellar peduncle.

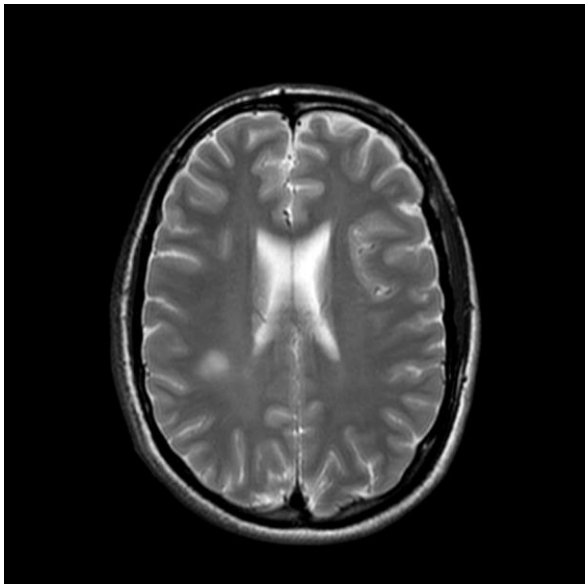

Figure 3 AXIAL T2 weighted sequence of Brain showing hyper intensity in right parietal region- likely suggestive of Demyelinating changes.

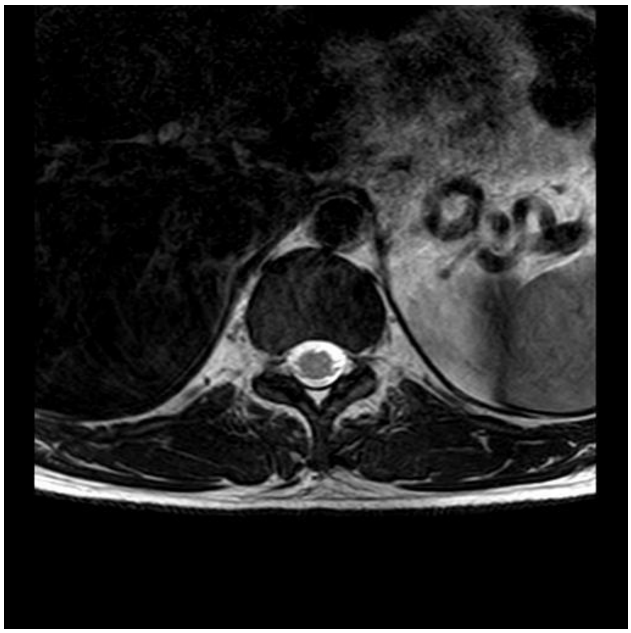

Fig 4- Axial T2 W sequence of spine showing resolution of hyperintensity after the treatment.

Now by combining Radiological, Serological and Clinical findings Diagnosis of Myelin Oligodendrocyte Glycoprotein Demyelination Disorder was made.

DISCUSSION

Antibodies against myelin oligodendrocyte glycoprotein (MOG-IgG) are linked to a diverse range of inflammatory demyelinating disorders of the central nervous system (CNS). In adults, the most common manifestations include optic neuritis (45.8%), transverse myelitis (22.8%), and brainstem encephalitis (17.1%), whereas in children, acute disseminated encephalomyelitis (ADEM) is more prevalent. Additionally, a minority of patients present with atypical conditions such as encephalitis, leukodystrophy, posterior reversible encephalopathy syndrome, multiple sclerosis, meningoencephalitis, or aseptic meningitis, with MOG-IgG detected in these cases(1).

The clinical presentation of MOG antibody-associated disease (MOGAD) varies by age and may include ADEM-like syndrome, focal neurological deficits, optic neuritis, transverse myelitis, or encephalitis. (2)

The use of a live cell-based assay to detect serum MOG IgG is both highly specific and sensitive for diagnosing MOG antibody-associated disease (MOGAD). In contrast, fixed cell-based assays show lower sensitivity and specificity. Serum

is the preferred biospecimen for MOG IgG testing. Although cerebrospinal fluid (CSF) may test positive for MOG IgG in 40-60% of MOGAD patients, relying solely on CSF testing may result in missed diagnoses in many cases.(3)
Longitudinally extensive spinal cord lesions on T2-weighted sagittal MRI, spanning 3 or more vertebral segments, are frequently observed in MOGAD and are present in the majority of cases (60 to 100 percent). These lesions can occur alone or in combination with other longitudinally extensive or short lesions. While isolated short lesions can occur, they are less common, and multiple sclerosis (MS) should be considered, especially if the lesions are located peripherally or show a ring of enhancement. On axial images, lesions in up to 75 percent of cases affect the central gray matter, often forming an "H-sign." In about one-third of cases, the lesions may be confined to the gray matter and appear as a continuous sagittal line, known as the "ventral sagittal line" sign. Myelitis with MOGAD has a predilection for the conus medullaris (image 8 and image 9), which may explain the high frequency of bowel, bladder, and sexual dysfunction. Acute disseminated encephalomyelitis (ADEM) is the most common presentation of MOGAD in children. Typical imaging findings in ADEM or ADEM-like presentations of MOGAD include multiple large, poorly defined ("fluffy") T2 hyperintensities in the white matter. Involvement of the deep gray matter, particularly with unilateral or bilateral T2 hyperintensities in the thalamus or basal ganglia, is commonly observed and helps differentiate MOGAD from multiple sclerosis (MS). However, these imaging features can also overlap with those seen in AQP4-IgG-positive neuromyelitis optica spectrum disorder (NMOSD). (4)

CONCLUSION

This case report highlights the importance of recognizing **Myelin Oligodendrocyte Glycoprotein (MOG)-associated demyelinating disease (MOGAD)** as a potential mimicker of other autoimmune conditions, particularly in male patients. MOGAD can present with a variety of neurological manifestations, including optic neuritis, transverse myelitis, encephalitis, and even ADEM-like symptoms, making it challenging to differentiate from other autoimmune or infectious demyelinating disorders. The variability in presentation, combined with the distinct but often subtle imaging and clinical features, underscores the need for heightened awareness and a thorough diagnostic approach.
Early identification of MOGAD is crucial, as it has significant implications for treatment and prognosis. With its unique response to immunotherapy, timely diagnosis can help guide appropriate management and potentially improve outcomes for affected patients. This report emphasizes the role of **MOG-IgG testing** and advanced imaging techniques, particularly **MRI**, in distinguishing MOGAD from other conditions like multiple sclerosis and neuromyelitis optica spectrum disorders (NMOSD).
In conclusion, MOGAD should be considered in the differential diagnosis of autoimmune demyelinating diseases, especially in male patients presenting with atypical or focal neurological deficits. Further research into the clinical, radiological, and immunological characteristics of MOGAD will aid in refining diagnostic strategies and enhancing the understanding of this emerging and complex disorder.

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