Turkish Journal of Neurology

Turk J Neurol 2025;31(1):76-79
Doi: 10.55697/tnd.2025.149

CASE REPORT

Chronic progressive behavioral changes associated with headache: An atypical presentation of myelin oligodendrocyte glycoprotein (MOG)-associated disease

Neetu Sinha[1], Pawan Prakash[2], Abhay Ranjan[2]

[1]Department of Radiodiagnosis, Indira Gandhi Institute of Medical Sciences, Patna, India
[2]Department of Neurology, Indira Gandhi Institute of Medical Sciences, Patna, India

ABSTRACT

Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease does not have clinical features that are disease-specific, and its clinical spectrum has yet to be defined. Herein, we described an atypical presentation of MOG-related disease. A 26-year-old female presented with behavioral changes in the past seven months in the form of agitation, anger, anxiety, fear of death, panic attacks, and an obsession to repeatedly perform daily activities. The patient also had headache for six months. The patient had a similar history of behavioral abnormality and four episodes of painful visual blurring. Neurological examination was unremarkable except for truncal and gait ataxia. Magnetic resonance imaging of the brain showed hyperintense signals in bilateral subcortical white matter, middle cerebral peduncles, cerebellum, thalamus, and pons with contrast enhancement. Myelin oligodendrocyte glycoprotein antibody was found to be positive by cell-based indirect immunofluorescence assay. The patient was managed with an intravenous methylprednisolone pulse. The patient had complete resolution of symptoms and magnetic resonance imaging brain abnormalities at the one-month control.

Keywords: Behavioral symptom, myelin oligodendrocyte glycoprotein associated disorder, psychiatric symptom.

Myelin oligodendrocytes glycoprotein (MOG) antibody-associated disease (MOGAD) is an inflammatory disorder of the central nervous system characterized by attacks of immune-mediated demyelination targeting the optic nerves, spinal cord, and brain. It is a differential diagnosis of growing significance for demyelinating lesions in the cerebral nervous system and occurs in the presence of MOG antibodies. In adults, MOGAD commonly presents with optic neuritis and myelitis but may rarely present with altered consciousness, behavioral changes, and epileptic seizure.[1] Herein, we reported a case of MOGAD with atypical presentation.

CASE REPORT

A 26-year-old female presented to our hospital with behavioral changes in the past seven months in the form of agitation, anger, anxiety, fear of death, panic attacks, disturbed sleep-wake cycle with insomnia, disinhibition, and obsession with repeatedly doing daily activities such as bathing and offering prayer. The patient also had an episodic, bifrontal headache, which was mild in intensity and relieved by vomiting, for six months. Twenty days before presentation to our hospital, the patient developed acute onset headache (holocranial, moderate grade, and continuous), vomited 33 times, and had gait imbalance in the past 20 days. The patient needed the support of two people to stand and walk. The patient had a similar history of behavioral abnormality two years back, which improved with psychiatric medications in six months. The patient had four episodes of painful visual blurring since 2018, and the last episode was in January 2023. Each time,

Correspondence: Abhay Ranjan, MD. Department of Radiodiagnosis, Indira Gandhi Institute of Medical Sciences, Patna, India.
E-mail: drabhayranjan97@gmail.com
Received: March 06, 2024 **Accepted:** May 03, 2024 **Published online:** March 05, 2025
Cite this article as: Sinha N, Prakash P, Ranjan A. Chronic progressive behavioral changes associated with headache: An atypical presentation of myelin oligodendrocyte glycoprotein (MOG)-associated disease. Turk J Neurol 2025;31(1):76-79. doi: 10.55697/tnd.2025.149.

the patient had a complete recovery of vision with steroid treatment. A written informed consent was obtained from the patient.

On examination, the patient was conscious and well-oriented to time, place, and person. The motor and sensory examinations were unremarkable. The patient had truncal and gait ataxia. The higher mental function was normal. The cranial nerve examination was regular. Magnetic resonance imaging (MRI) of the brain depicted multifocal, asymmetrical, patchy, and some punctate hyperintense signals in the subcortical white matter of the bilateral frontoparietal regions, bilateral superior temporal regions, bilateral middle cerebral peduncles, bilateral cerebellar regions, bilateral thalami, and pons. Contrast-enhanced, T1-weighted imaging of the brain showed patchy and punctuate enhancement in the above areas (Figures 1a-c).

The laboratory examinations, including hematological, biochemistry, and thyroid profile, anti-thyroid peroxidase antibodies, and vitamin B12 were normal. Visual evoked potential showed normal bilateral P100 latency. Lumbar puncture with cerebral spinal fluid analysis was normal, with no oligoclonal bands. Anti-neuromyelitis

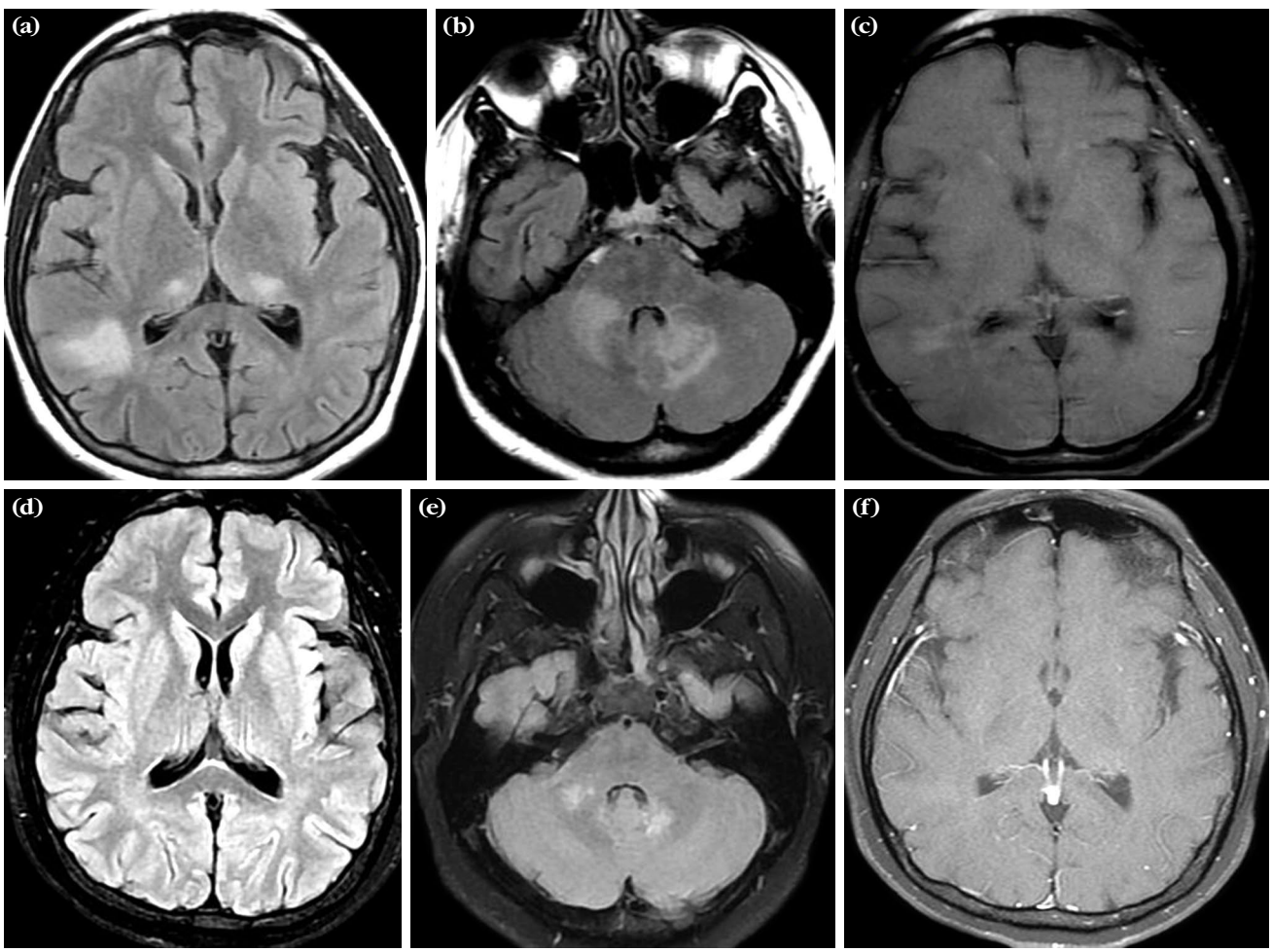

Figure 1. (a) Axial T2-FLAIR MRI demonstrates asymmetrical, hyperintense, and poorly demarcated areas in the subcortical and deep white matter of the right temporal lobe and bilateral thalami (more on the left side). **(b)** Axial T2-FLAIR MRI shows a hyperintense signal in the pons, bilateral middle cerebellar peduncles, and cerebellum. **(c)** Gadolinium-enhanced, T1-weighted axial MRI shows patchy and punctate enhancement in the cerebellum, middle cerebellar peduncles, and right temporal region (not shown here). In addition, hyperintense areas are also observed in the subcortical white matter of the left frontoparietal and right medial temporal region on T2-FLAIR sequences. **(d-f)** In the follow-up MRI at one month, the lesions significantly decreased in size and contrast enhancement.

FLAIR: Fluid-attenuated inversion recovery; MRI: Magnetic resonance imaging.

optica (NMO) and MOG antibodies were assessed. The MOG antibody was found positive by using cell-based indirect immunofluorescence assay, and anti-NMO antibodies were not found. Cerebrospinal fluid and serum examinations were performed for autoimmune encephalitis, and the panel was negative. Electroencephalogram was normal. The abdomen was normal on ultrasonography.

The patient was managed with intravenous methylprednisolone pulse therapy 1,000 mg/day for five days and improved with complete resolution of the headache and vomiting. However, at the time of discharge, the patient had mild ataxia while walking but did not require support. The patient was on oral prednisolone 60 mg daily. At the one-month follow-up, there was a complete resolution of ataxia. The repeat MRI showed a significant reduction in white matter hyperintensities, and contrast enhancement was observed in the supratentorial white matter, cerebellum, thalamus, and pons (Figures 1d-f).

DISCUSSION

The behavioral and psychiatric changes associated with MOG antibodies are generalized anxiety disorder, dissociative entity disorder, suicidal ideation, amnesia, insomnia, agitation, hallucinations, panic attacks, and schizophrenia.[2,3] In this case, the patient also presented with behavioral symptoms in the form of agitation, anger, fear of death, panic attacks, disinhibition, and obsession with doing things repeatedly. The patient's obsessive behavior had not been previously reported to the best of our knowledge. The patient used to bathe repeatedly and offer prayers at frequent intervals.

Baljinder Singh et al.[2] reported two cases of anti-MOG disease who presented with psychiatric symptoms. The first patient presented with sudden onset visual loss along with generalized anxiety disorder, schizophrenia, dissociative entity disorder, and suicidal ideation, which resolved with intravenous steroids. The second patient also presented with auditory hallucinations, anxiety, major depression, and ablutophobia. The patient received plasmapheresis, which improved the acute symptoms, including psychiatric symptoms.

In a study done by Pedapati et al.[4], the most common syndrome at onset was unilateral optic neuritis (40%), followed by bilateral optic neuritis (35%), transverse myelitis (15%), optic neuritis plus transverse myelitis (5%), and cerebral syndrome (5%). In the study by von Zedtwitz et al.,[3] they reported two cases with anti-MOG antibody positivity and psychosis. Cherian et al.[5] reported a case who developed headaches, episodic memory loss, and behavioral disturbances suggestive of an autoimmune anti-NMDAR (N-methyl-D-aspartate receptor) encephalitis with positivity for NMDAR, CASPR2 (contactin-associated protein-like 2), and MOG. Yilmaz et al.[6] described a case of MOG-related disease with behavioral and personality changes following a seizure episode. Following intravenous immunoglobulin therapy, the patient showed complete recovery.

In conclusion, this case highlights the importance of considering MOGAD as a differential diagnosis in adults presenting with subacute to chronic behavioral symptoms. In MOGAD, patients can have obsession as a behavioral symptom. Early diagnosis and treatment with steroids lead to complete to near complete clinical improvement and prevent long-term morbidity.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea/concept, writing the article: A.R., Literature review: A.R., P.P., N.S.; Data collection: P.P., N.S.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

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