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A COMPREHENSIVE CASE STUDY OF MYELIN OLIGODENDROCYTE GLYCOPROTEIN ASSOCIATED ENCEPHALITIS

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ABSTRACT
A 54-year-old male was admitted on May 16th with a one-week history of giddiness, worsening headache, and blurred vision over the past three days, alongside a single episode of vomiting, mild right-sided walking difficulty, decreased memory, and altered behavior. He had no known prior medical conditions. On admission, his vitals were stable, and his GCS score was E4V5M6. Neurological examination showed no ataxia, and Romberg's test was negative. His lipid profile revealed low HDL (44 mg/dL) and high LDL (122 mg/dL), while liver function tests showed an elevated AST level (44 U/L). MRI of the brain indicated gyral thickening with FLAIR hyperintensity in the left posterior parieto-occipital and temporal lobes, with a chronic lacunar infarct in the right lentiform nucleus. The diagnosis included MOG-associated encephalitis, occipital blindness, and newly diagnosed type 2 diabetes mellitus. He developed hypertension (160/100 mm Hg), restlessness, and visual hallucinations, and was treated with antihypertensive and antidiabetic medications, eye drops, and antidepressants. Over the following days, he continued to experience headaches, reduced appetite, unformed stools, and blurred vision. His treatment was adjusted to include corticosteroids, NSAIDs, antibiotics, and additional antidiabetic drugs. EEG revealed mild focal dysfunction in the left occipital region. As his condition stabilized, certain medications were withdrawn or adjusted, including the addition of Vildagliptin and Mycophenolate mofetil. By the seventh day, his symptoms had improved, and he was discharged with a prescribed medication regimen that included antibiotics, corticosteroids, antidiabetics, eye drops, and pain relievers.

INTRODUCTION
Encephalitis refers to inflammation of the brain parenchyma, usually resulting from an infectious agent or an autoimmune response, which may be post-infectious, paraneoplastic, or idiopathic in nature. Patients may present with symptoms such as fever, changes in behavior, personality, cognition, and consciousness.[1] Additionally, they may experience focal neurological deficits, seizures, movement disorders, and/or autonomic instability. The global incidence of encephalitis is estimated to range from 3.5 to 12.3 cases per 100,000 individuals per year.[2] The range of autoimmune encephalitis syndromes has significantly broadened, with distinctive clinical profiles associated with specific pathogenic autoantibodies now being well-defined.[3] One of the autoantibody mediated encephalitis is MOG including acute disseminated encephalomyelitis. Myelin oligodendrocyte glycoprotein (MOG) antibody disease is a relatively recent category of autoimmune disorders characterized by the presence of antibodies against MOG. This condition primarily affects the optic nerve and spinal cord, often resulting in vision loss and paralysis.[4] Clinically, MOG antibody disease shares similarities with neuromyelitis optica spectrum disorder (NMOSD), particularly in the regions of the central nervous system (CNS) targeted by inflammatory attacks. It is noteworthy that up to 40% of NMOSD patients who are seronegative for aquaporin-4 (AQP4) antibodies are found to have MOG antibodies.[5] In this case report, we present and thoroughly discuss the case of a 54-year-old male patient who has been diagnosed with encephalitis associated with MOG antibodies.

CASE STUDY
A 54-year-old male patient was admitted to the hospital on the morning of May 16th with complaints of giddiness for one week, worsening headache, and blurred vision over the past three days, a single episode of vomiting, mild difficulty walking on the right side, decreased vision for the last three days, reduced memory, and altered behavior since that morning. He had no known prior medical history.

On admission, his vital signs were stable, and the Glasgow Coma Scale (GCS) score was E4V5M6. Neurological examination revealed no ataxia, and the

Romberg's test was negative. On May 16th, his lipid profile showed low HDL (44 mg/dL) and high LDL (122 mg/dL). Liver function tests revealed an elevated AST level (44 U/L). The complete blood count indicated low MCV (81 fl), high MCHC (35.4 fl), high polymorphs (72.2%), low basophils (0.7%), and elevated ESR (24 mm/hr). An MRI of the brain revealed gyral thickening with FLAIR hyperintensity along the left posterior parieto-occipital and temporal lobes, with faint post-contrast enhancement. A chronic lacunar infarct was also noted in the right lentiform nucleus. The diagnosis included MOG-associated encephalitis, occipital blindness, and newly onset type 2 diabetes mellitus.

On the first day, the patient developed increased blood pressure (160/100 mm Hg), mild restlessness, and visual hallucinations. He was treated with antihypertensive medications, antidiabetic drugs, eye drops, and antidepressants.

On the second day, the patient continued to experience headaches, reduced appetite, and unformed stools, although his blood pressure normalized, and the GRBS level was 239 mg/dL. CSF culture and antibiotic susceptibility tests showed no bacterial growth, and molecular biology testing confirmed no HSV infection. The autoimmune IFA test for Anti-MOG antibody was weakly positive. CSF fluid analysis showed increased protein (54 mg/dL) and sugar (123 mg/dL). HbA1c was elevated at 10.5%. The same medications were continued as on the first day, with the addition of another antidiabetic drug, NSAIDs, antibiotics, and corticosteroids.

On the third day, the patient continued to complain of headaches, blurred vision, and irrelevant speech. An EEG revealed mild focal, non-specific electrophysiological dysfunction over the left occipital region without epileptiform abnormalities or seizures. Renal function tests were normal, but the complete blood count showed an increase in RBCs (5.70 million/mm3), decreased MCV (82.8 fl), elevated total WBCs (11,940 cells/mm3), increased polymorphs (90.3%), decreased lymphocytes (8.8%), decreased monocytes (0.5%), decreased basophils (0.2%), and an elevated ESR (35 mm/hr). Glimepiride 1 mg was withdrawn, and methylprednisolone 1 g in 100 ml NS over one hour was administered instead of dexamethasone. The rest of the medications remained unchanged.

On the fourth day, the patient reported headaches, blurred vision, irregular sleep, and continued irrelevant speech. Blood urea levels increased to 60 mg/dL, while C-reactive protein remained normal. The same medications were continued, with the addition of sleeping medications, and the dose of Quetipine was increased to 50 mg due to the patient's sleep problems and irrelevant speech.

On the fifth day, the patient experienced giddiness and blurred vision. GRBS decreased to 251 mg/dL. RFT results showed a blood urea level of 48 mg/dL. The complete blood count indicated increased WBCs (14,440 cells/mm3), elevated polymorphs (90.3%), decreased lymphocytes (7.7%), decreased monocytes (0.7%), and decreased basophils (0.3%). Ceftriaxone 2 g and acyclovir 500 mg injections, as well as Flupirtine + paracetamol tablets, were withdrawn. The dose of Quetipine was reduced from 50 mg to 25 mg. Cefixime 200 mg twice daily was added.

On the sixth day, the patient continued to experience giddiness and blurred vision, with decreased irrelevant speech. Citicoline injections were withdrawn. Metformin SR 500 mg was replaced with metformin 500 mg + glimepiride 0.5 mg. Vildagliptin and Mycophenolate mofetil were added to the treatment regimen.

On the seventh day, the patient complained of blurred vision, and GRBS levels increased to 311 mg/dL. Lacosamide was withdrawn, and the metformin 500 mg + glimepiride 0.5 mg combination was replaced with metformin 500 mg + glimepiride 1 mg.

Following this, the patient's symptoms resolved, and he was discharged with the following medications: Cefixime 200 mg 1-0-1 for 4 days, prednisolone 80 mg 1-0-0 for 4 days (followed by 60 mg for the next days), pantoprazole 40 mg 1-0-0 before food for 7 days, Mycophenolate mofetil 500 mg 1-0-1, Vildagliptin 50 mg 0-1-0, melatonin 3 mg 0-0-1, Quetipine 25 mg 0-0-1, glimepiride 1 mg + metformin 500 mg 1-0-1 before food, carboxymethylcellulose eye drops 1-1-1, brimonidine tartrate eye drops 1-0-1 for 7 days, and Flupirtine + paracetamol for headache as needed.

Medications	**Dose**	**ROA**	**Frequency**	**1st day**	**2nd day**	**3rd day**	**4th day**	**5th day**	**6th day**	**7th day**
Inj. Citicoline	500mg in 100ml NS	IV	1-1-1	✓	✓	✓	✓	✓	✓	-
Inj. Pantoprazole	40mg	IV	1-0-1	✓	✓	✓	✓	✓	✓	✓
T. Metformin SR	500mg	P/O	1-0-1	✓	✓	✓	✓	✓	-	-
Carboxy methyl cellulose eye drops	0.5%		1-1-1	✓	✓	✓	✓	✓	✓	✓
Brimonidine tartrate eye drops	0.1%		1-0-1	✓	✓	✓	✓	✓	✓	✓
T. Cilnidipine	10mg	P/O	Stat	✓	-	-	-	-	-	-

T. Quetipine	25mg	P/O	0-0-1	✓	✓	✓	-	-	✓	✓
T. Quetipine	50mg	P/O	0-0-1	-	-	-	✓	✓	-	-
T. Glimepiride	1mg	P/O	1-0-0	✓	✓	-	-	-	-	-
Inj. Ceftriaxone	2mg	IV	1-0-1	✓	✓	✓	✓	-	-	-
Inj. Acyclovir	500mg with 100ml NS over 1hrs	IV	1-1-1	✓	✓	✓	✓	-	-	-
Inj. Dexamethasone	4mg	IV	1-1-1-1	✓	✓	-	-	-	-	-
Inj. Methylprednisolone	1gm in 100ml NS over 1 hrs	IV	1-0-1	-	-	✓	✓	✓	✓	✓
T. Melatonin	3mg	P/O	0-0-1	-	-	✓	✓	✓	✓	✓
T. Lacosamide	50mg	P/O	1-0-1	-	-	✓	✓	✓	✓	✓
T. Flupirtine + Paracetamol	100mg + 325mg	P/O	1-1-1	-	✓	✓	✓	-	-	-
T. Cefixime	200mg	P/O	1-0-1	-	-	-	-	✓	✓	✓
T. Metformin + Glimepiride	500mg + 0.5mg	P/O	1-0-1	-	-	-	-	-	✓	✓
T. Mycophenolate Mofetil	500mg	P/O	1-0-1	-	-	-	-	-	✓	✓
T. Vildagliptin	50mg	P/O	0-1-0	-	-	-	-	-	✓	✓

DISCUSSION

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is increasingly recognized as a critical differential diagnosis for demyelinating lesions in the central nervous system (CNS), particularly in the presence of MOG antibodies (MOG-AB). The diagnosis of MOGAD is complicated by significant clinical and radiological overlap with other classical demyelinating diseases, such as multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and acute demyelinating encephalomyelitis (ADEM).[6] Clinically, both classical ADEM and MOG antibody-associated encephalitis exhibit similar presentations. However, classical ADEM is more frequently associated with myelitis and/or optic neuritis at the initial presentation. Radiologically, classical ADEM is characterized by diffuse, confluent lesions involving both white and gray matter.[7] In contrast, lesions in MOG antibody-associated encephalitis are typically more well-defined, often cortically based, or in some cases, absent altogether. Demographically, MS patients tend to be slightly older than those with MOGAD and are predominantly of Caucasian descent. Additionally, MS disproportionately affects women, with a female-to-male ratio of approximately 3:1.[8]

A 30-year-old male presented with a five-day history of headache, fever, neck stiffness, and fatigue. Initial neurological and general examinations were unremarkable, and a normal CT scan was followed by cerebrospinal fluid (CSF) analysis showing pleocytosis, elevated protein, normal glucose, and negative oligoclonal bands (OCB). Empirical treatment with acyclovir and ceftriaxone was initiated for suspected acute meningitis, despite negative serological tests for common pathogens. Nine days later, the patient developed rapid tetraparesis, predominantly in the lower extremities, with hypoesthesia below the T8 level, neck stiffness, and gaze-evoked nystagmus. Repeated CSF analysis revealed persistent lymphocytic pleocytosis and elevated protein. MRI showed T2-weighted hyperintense signals in the pons and cerebellar peduncles, along with longitudinal extensive transverse myelitis (LETM) from C3 to C7, without blood-brain barrier disruption. Despite negative serology, methylprednisolone was continued, and IVIG therapy was added, leading to significant symptom improvement. Autoantibody testing revealed high MOG antibody titers in serum and CSF, leading to a diagnosis of MOG antibody-associated disease (MOGAD). The patient was discharged with residual neurogenic bladder dysfunction and mild hypoesthesia, with follow-up MRIs showing no abnormalities. Long-term cortisone treatment was tapered, followed by azathioprine therapy.[9]

In a 2018 Chinese study, 20.7% of MOG-positive patients experienced encephalitis during their disease course, with a median onset age of 22 years. The cohort included 10 men and 8 women. In contrast, a related Chinese cohort of AQP4 seropositive NMOSD patients showed that only 3.6% had encephalitis, all of whom were females in their late 30s.[10] Similarly, a 2018 French study found that 2.5% (5 out of 197) of MOG-positive patients had encephalopathy, with an additional 2% presenting with brain stem syndrome and encephalopathy.[11] A case report highlights a 46-year-old MOG-positive patient who initially presented with paraparesis due to bifrontal cortical encephalitis and later relapsed with optic neuritis, representing an older patient profile compared to other series.[12]

In a series of 18 patients from China, all exhibited typical symptoms of encephalitis, including reduced consciousness, headache, and behavioral changes. Seizures were reported in 9 of these cases. The study found that 72.2% of patients with MOG encephalitis

experienced a relapsing course of the disease. Two-thirds had encephalitis relapses at the onset, while one-third initially experienced optic neuritis attacks before progressing to encephalitis.[10] Additionally, 41.2% of the patients had elevated intracranial pressure during encephalitis attacks, and 64.7% displayed CSF pleocytosis. In comparison, the average white cell count in CSF was 251 cells/μL in the UK study[13] and 126 cells/μL in the Japanese study.[14]

In a Japanese case series, four patients who experienced generalized epileptic seizures, with or without altered consciousness, displayed unilateral cortical brain lesions on FLAIR imaging. These lesions corresponded with hyperperfusion observed on single-photon emission computed tomography (SPECT). Notably, the lesions were distinguished from seizure-induced changes by their presence on FLAIR rather than diffusion-weighted imaging (DWI), the presence of cerebrospinal fluid (CSF) pleocytosis, and a positive clinical response to high-dose steroids. These findings suggest an inflammatory origin for the cortical lesions.[14]

Acute management of MOG antibody encephalitis typically involves corticosteroids and/or plasma exchange or intravenous immunoglobulin (IVIG). Published case series indicate that most patients were initially treated with intravenous methylprednisolone, followed by a gradual tapering of oral prednisone, leading to complete recovery.[13] For those with a relapsing course, long-term immunosuppressive therapy is recommended. Data on long-term treatment are limited, with a single case series from the United Kingdom providing the majority of available information. Rituximab and mycophenolate mofetil are the most frequently used agents in these cases.[12]

CONCLUSION

Myelin oligodendrocyte glycoprotein (MOG) antibody disease is a relatively recent category of autoimmune disorders characterized by the presence of antibodies against MOG. A 54-year-old male was admitted on May 16th with symptoms of dizziness, worsening headache, blurred vision, vomiting, mild right-sided gait difficulty, memory issues, and altered behavior. Neurological and imaging studies revealed gyral thickening with FLAIR hyperintensity in the left posterior parieto-occipital and temporal lobes, and a chronic lacunar infarct in the right lentiform nucleus. The patient was diagnosed with MOG-associated encephalitis, occipital blindness, and newly diagnosed type 2 diabetes. He developed hypertension and visual hallucinations. His treatment included antihypertensives, antidiabetics, corticosteroids, NSAIDs, antibiotics, and adjustments in medication. By the seventh day, his condition improved, and he was discharged with a revised medication regimen.

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