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RESEARCH ARTICLE
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A CHALLENGING CASE OF MOGADS (MYELIN OLIGODENDROGLIAL ANTIBODY DISEASE SPECTRUM) THAT CLOSELY MIMICS CAUDA EQUINA SYNDROME

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ABSTARACT
This case report describes the case of a young male who presented with subacute onset of cauda equina syndrome. MRI spine revealed enhancing lesions in the white matter on T2W imaging from T12 to L4 and patient was positive for MOG(myelin oligodendrocyte glycoprotein antibodies) suggestive of Myelin oligodendrocyte glycoprotein antibody disease(MOGADS). The patient was treated with steroids and made had a steady improvement in clinical features. This case report furthermore discusses the differential diagnosis of MOGAD syndrome including multiple scleroisis and anti NMO disease (neuromyelits optica syndrome).

CASE DESCRIPTION
Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD) is increasingly recognized as a distinct disease entity, with a wide spectrum of presentations.[1] Herein, we report a case with findings initially like cauda equina syndrome subsequently diagnosed with MOGAD. Though rare, it is now recognized that patients with MOGAD could have typical MS attacks at onset.[2-4] The major challenge, however, lies in the early recognition and correct diagnosis of such patients for an appropriate therapeutic strategy. Our case argues for the importance and proposed the rationale for a MOG antibody screen in selected patients with clinical phenotypes suggestive of MS. We also discussed rituximab as a therapeutic option in such patients.

Mr X a 23 years gentlemen from rural Rajasthan farmer by occupation presented with h/o progressive descending weakness involving both the lower limbs progressing over a few hours associated with difficulty in micturition and defecation for 2 days.

On examination the vitals of the patient were stable, he had a bilateral weakness of both lower limbs power was 3/5 in both lower limbs with absent knee ankle jerks flexor plantars and decreased perianal sensation with bowel and bladder affection resembling cauda equina syndrome without a clear sensory level demarcation. The patient denied any history of fever, cough, sore throat and diarrhea. Recent exposure to farm pesticides. The patient had a mixed diet with no significant past, personal and family history. The patient denied any history of addictions.

Biochemical examination was essentially normal. The patients serology for HIV/HbsAg/ HCV/ Enterovirus/HSV/HTLV were negative. The CSF examination was normal. MRI brain was normal, however, MRI spine revealed an hyperintense lesion on T2 imaging, extending from T12 to L4 suggestive of demyelination. Antibodies to oligodendrocyte glial protein (MOG) was positive, anti NMO (Neuromyelitis Optica) acquaporin-4 antibody was negative.

Patient was diagnosed as Acute Anti MOG syndrome, was catheterized started on iv methylprednisolone and transitioned to tapering course of oral methylprednisolone. Steroid tapering was planned over a period of 3to 4 months depending on response.

Patients symptoms started improving after 2 to 3 days of starting intravenous methylprednisolone, with improvement in bladder control, lower limb power and patient was discharged after 10 days with close watch and regular follow up.

DISCUSSION

Typical MOGAD has diverse presentations depending on age and can include ADEM-like presentation, focal neurologic deficits, optic neuritis, transverse myelitis, or encephalitic presentation.[4] Less common symptoms include cortical encephalitis or seizures.[1,5] Initial symptom onset of MOGAD with cauda equina like presentation enhancement seems to be rare.[2,3]

Neuroradiological findings in adult MOGAD patients are, typically poorly demarcated or tumefactive lesions with variable contrast enhancement. Spinal presentation ranged from transverse myelitis to LETM, while in the brain affections of the deep grey and white matter and the cortex were frequently detected.[5]

Though considered as distinct disorders, manifestations of MS and MOGAD may overlap. Among them, short-segment myelitis constitutes the initial presentation in more than half of multiple sclerosis (MS), up to 53% of MOGAD[1,6] and 15% of neuromyelitis optica spectrum disorder. In addition, around 6% to 17% of MOGAD patients have positive CSF OCB and 33% may full fill the McDonald diagnostic criteria. [1] Also, given their overlapping presentations, differentiation between the two disorders is extremely important based mainly on two reasons: 1) maintenance therapy of MS and MOGAD differs greatly: on the one hand, DMT is mainly used in patients

with MS; on the other, patients with MOGAD are particularly responsive to antibody-depleting treatments, but may even deteriorate with DMT;[1] 2) MOGAD is associated with a high risk of flare-ups after cessation of steroid treatment and maythus require close monitoring and careful steroid tapering at leastover 6 months.

Dilemma, however, lies in the selection of patientsbased on the trade-off between the highest diagnostic yield andunnecessary testing. The presence of short-segment myelitis or intrathecally restricted OCBs should not exclude a diagnosis ofMOGAD. Further, serum MOG antibody testing is warranted inpatients with the following features, even if the McDonald criteria were fulfilled: 1) recurrent myelitis with or without brain lesions; 2) conus medullaris involvement, especially I fpresent

at onset; 3) unsatisfied disease control with steroid or DMT, i.e., frequent flare-ups during steroid tapering or aftersteroid cessation, or relapses even during DMT use.[6]

Notably, the clinical clues become more important with a low-titer MOG antibody testing result to evaluate the clinical phenotype and diagnosis based solely onantibody testing should be avoided. It is the constellation of recurrent myelitis, lack of brain lesions at the first episode, involvement of conus medullaris, and a remarkable steroid dependence can lead to the final diagnosis of MOGAD.

The evolving identification of MOG patients with MS-likephenotypes also holds therapeutic implications. MOGAD exhibits a poor response to DMTs, including interferon-betaand glatiramer, hence making it likely that MOGAD and belong to distinct entities. Rather, a heterogeneous efficacy could be achieved with mycophenolate mofetil, AZA, RTX, ororal prednisolone. In particular, RTX, a B-cell depletingagent, was reported to reduce relapse rate by around 40% when used in treatment-naive patients. However, around 80% of relapses continued despite roof relapses bust B-cell depletion. Therefore, identifying those RTX-responsive patients when determining thetherapeutic strategy would be of great value. Interestingly, in the eight patients with clinical phenotypes suggestive of MS but ultimately diagnosed with MOGAD there is a The persistent positive MOG-AB titer are an indicator of an relapsing disease course.[3] Therapy of acute MOGAD consists of corticosteroids, intravenous immunoglobulins or plasma exchange. In patients with a high risk for relapses, long term immune modulating therapies include intravenous immunoglobulins, rituximab, azathioprine, and mycophenolate mofetil.[2]

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