CLINICAL SPECTRUM OF MOGAD PATIENTS WITH DIVERSE PHENOTYPES- A SHORT CASE SERIES

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Myelin oligodendrocyte glycoprotein associated disease **(MOGAD)** is a rare, inflammatory demyelinating disorder of the CNS with various phenotypes starting from optic neuritis, via transverse myelitis to acute demyelinating encephalomyelitis (ADEM) and cortical encephalitis.[1]

Optic neuritis (ON) is the **most frequent clinical phenotype in older age patients**. The optic neuritis in MOGAD is typically **bilateral at onset** and can lead to blindness over hours to a few days. The **anterior optic pathway is predominantly affected** and fundoscopy often reveals optic disc edema, sometimes accompanied by retinal hemorrhages.

Myelitis in MOGAD is **longitudinally extensive** in approximately 70% to 80% of cases, but **shorter lesions often coexist** on MRI, which is different from the single myelitis lesion typically encountered in AQP4-NMOSD. **The conus medullaris is frequently affected**, often accompanied by sphincter or sexual dysfunction.

ADEM or ADEM-like phenotype is characterized by MRI evidence of **multifocal CNS involvement with or without encephalopathy.** Anti-MOG antibodies are reported in **40% - 68% of children with ADEM** diagnosis. But in adults with the positive anti-MOG test, ADEM presentation is less frequent, varies from a few up to 18% of cases. The spectrum of attack phenotypes in MOGAD is broader than in AQP4-NMOSD.

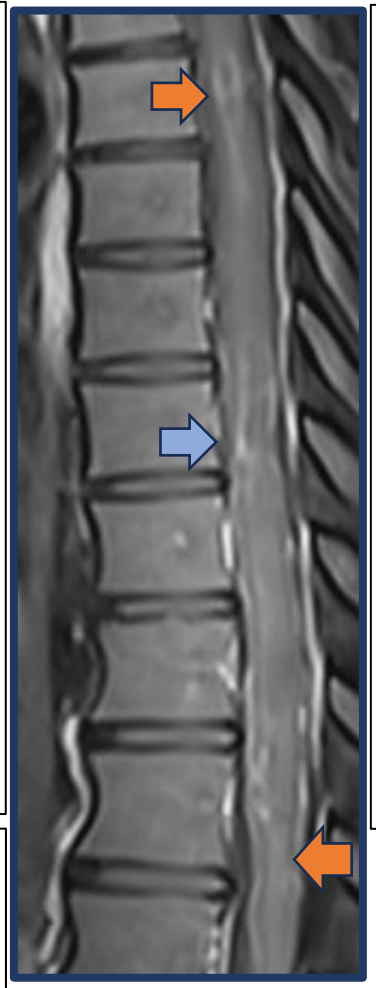
Critical element of reliable diagnosis is **detection of pathogenic serum antibodies MOG,** preferably with optimized **cell-based assay (CBA)**; **along with core clinical demyelinating event, supporting MRI & clinical features** (Intl MOGAD panel proposed criteria 2023).[2]

Acute immunotherapy is very effective in MOGAD, and severe disability (ambulatory and visual) is less frequent than in NMOSD; so it is critical to diagnose this condition to begin early, appropriate treatment.

CASE NUMBER 1

MOGAD presenting as longitudinally extensive myelitis with conus involvement.

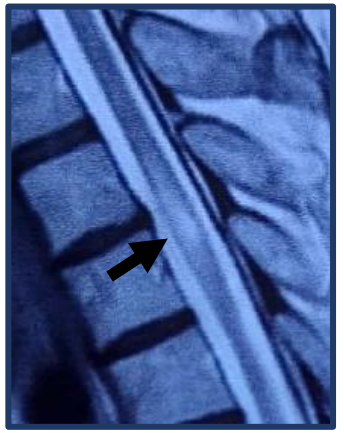
- Young adult male presented with subacute onset, asymmetrical UMN type paraparesis with urinary voiding difficulties of 1 month duration suggestive of a **cauda-conus syndrome**.
- MRI showing **LETM extending from C7 till conus** with minimal cord expansion, **heterogenous intramedullary enhancement** seen. Serum **MOG antibody positive** by cell-based assay.
- Patient was **treated with iv MPS and PLEX**, followed by Oral Prednisone & Rituximab, and improved from EDSS of 7.5 to 6 within 15 days of treatment.



CASE NUMBER 2

MOGAD presenting as recurrent short segment transverse myelitis- conus sparing.

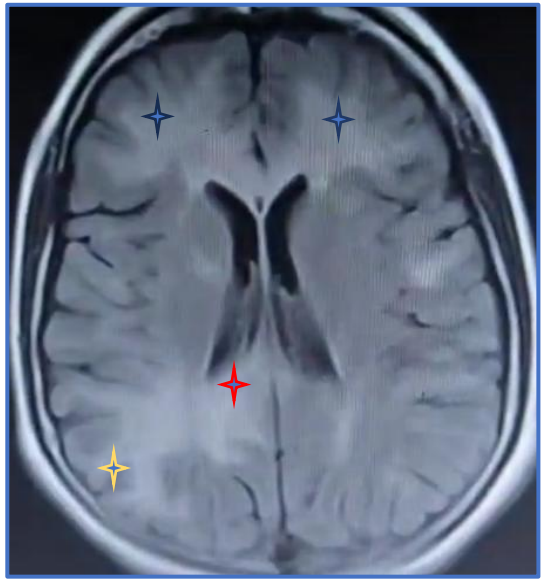
- Young male with past h/o **steroid responsive short segment transverse myelitis with funicular pains** on medial aspect of both forearm.
- MRI showing **non-enhancing T2 hyperintense signal involving lower cervical spinal cord**, and demyelinating plaques in periventricular & subcortical white matter. **MOG antibody (+) [1:32].**

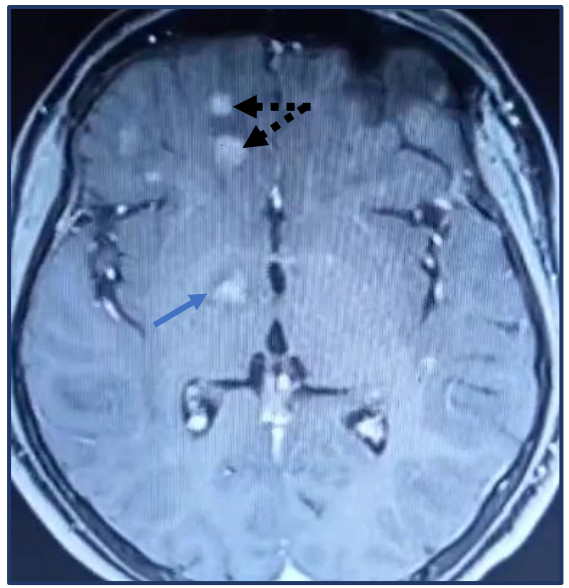


CASE NUMBER 3

MOGAD presenting as ADEM without encephalopathy in a pediatric patient.

- 10 year old girl with a history of **chronic fever along with headache and seizures**, was evaluated for PUO, clinically diagnosed as Tubercular Meningitis and initiated on ATT when she presented to our institute.
- MRI Brain with contrast demonstrated **extensive multifocal bilateral T2/FLAIR hyperintensities** involving the subcortical and periventricular white matter of both cerebral hemispheres, as well as corpus callosum, with multiple enhancing lesions s/o **Acute Disseminated Encephalomyelitis (ADEM)**. **Serum MOG antibody positive**.
- Patient was **treated with IVIG** and showed improvement to become symptom-free within one month.

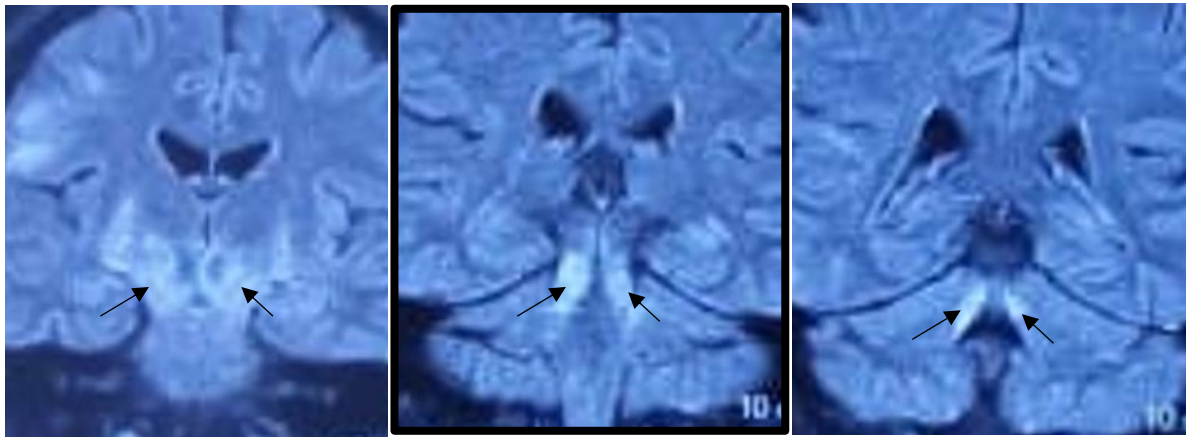




CASE NUMBER 4

MOGAD presenting with recurrent attacks of Optic Neuritis (sequential involvement).

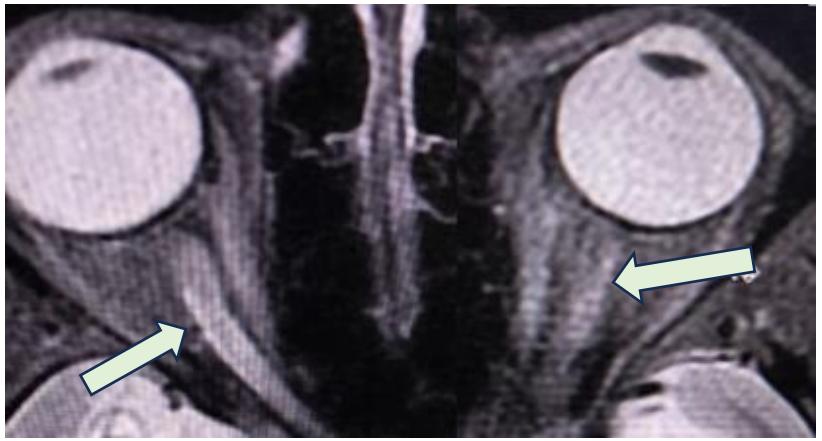
- 27 year old male presented with an **acute onset of painless DOV, left f/b right** in a span of 1 week; a/w colour desaturation & loss of contrast sensitivity of 1 month duration. Acute brainstem syndrome with dysphagia, dysarthria and hiccups; and cerebellar ataxia.
- **Past h/o Left sided optic neuritis in 2018, and right optic neuritis in 2023- both steroid responsive.**
- **VEP showing prolonged P100 latencies** of 149.1ms in left eye and 192.9ms in right eye. CSF showed lymphocytic pleocytosis with mildly elevated protein. NMO-MOG **panel positive for MOG antibody**.
- He was **treated with IV MPS, followed by IVIG** i/v/o poor improvement of V_A, **followed by Rituximab** for maintenance immunotherapy.



CASE NUMBER 5

MOGAD presenting as typical Optic Neuritis (painless unilateral DOV)

- 20/Male with acute onset left eye painful vision loss, associated with color desensitization. **Left eye RAPD (+)** and **V_A of P_L** present. Prolonged VEP of 131ms in right eye. **Serum MOG antibody strongly positive.**



He was **treated with IV MPS followed by Oral prednisone taper and Rituximab** i/v/o severe LOV in 1st attack. Repeat V_A testing after 1 month was 6/24.

CONCLUSION

MOGAD is a CNS demyelinating disease **with heterogenous disease manifesatations with both monophasic & recurrent clinical presentations**; which if diagnosed early and treated has a good prognosis.

REFERENCES

1. Sechi E. NMOSD and MOGAD. Continuum (Minneap Minn). 2024 Aug 1;30(4):1052-1087.
2. Banwell B et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. Lancet Neurol. 2023 Mar;22(3):268-282.