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MOG ANTIBODIES ASSOCIATED BILATERAL OPTIC NEURITIS WITH DEMYELINATION: A CASE REPORT

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ABSTRACT

Myelin oligodendrocyte glycoprotein (MOG) is a regulatory glycoprotein involved in microtubule stability of myelin surface. Optic neuritis is an inflammatory condition of optic nerve, caused by damage and loss of protective sheath (myelin) surrounding the optic nerve which is so vital for good vision. Aquaporin-4 (AQP-4) will be negative for patients with MOG antibodies associated optic neuritis. This is a case of 39 years old male patient with complaints of diminished vision in left eye followed by right eye since 1 month, which is gradually in onset and progressed within 5 days. MRI scan of dorsal spine showed T_3 _T_{11} hyper intensity in retrobulbar region and MRI with contrast study showed altered signal intensity in cervical and dorsal spinal cord with patchy enhancement suspicious of demyelination. Serum analysis shows presence of anti-MOG IgG antibodies. Due to the poor prognosis of the disease, patient was advised to be in observation for 15 days and planned for 2 doses of Rituximab 500 mg. Meanwhile, symptomatic treatment was also given. This case implies that the early diagnosis of MOG-antibodies can differentiate the demyelination from AQP-4 antibodies positive demyelination and can treat accordingly. It is necessary to test patients longitudinally to assess the anti-MOG serostatus. Rehabilitative care is needed to prevent secondary complications of immobility and to improve functional status.

Key Words:- Myelin oligodendrocyte glycoprotein (MOG), Optic neuritis, demyelination, Rituximab.

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INTRODUCTION

Myelin oligodendrocyte glycoprotein(MOG) is a glycoprotein located on the myelin surface and found exclusively in the central nervous system(CNS) (Thomas RW *et al.*, 2019). Although it's exact role remains unclear, it is thought to act as a cellular adhesive molecule, to be involved as a regulatory of oligodendrocyte microtubule stability and to mediate complement cascade (Johns T, Bernard C, 1999).Patients with MOG antibodies associated disease (MOG-AD) may previously have been diagnosed with Neuromyelitis optica spectrum disorder(NMOSD), Transverse myelitis(TM), Acute disseminated encephalomyelitis(ADEM), Optic neuritis(ON) or Multiple sclerosis(MS) because of the pattern of inflammation (Weber MS *et al.*, 2018).

Optic neuritis is an inflammatory condition of the optic nerve, caused by damage and loss of the protective sheath (myelin) surrounding the optic nerve which is so vital for good vision. It usually affects young adults, especially females, between 18 and 45 years of age (Menon V *et al.*, 2011). Those with MOG antibody associated optic neuritis do not test positive for NMO antibody called Aquaporin 4(AQP-4). The diagnosis is confirmed when MOG antibodies in the blood using live cell based assays in patients who have repeated inflammatory attacks of the CNS (Kortvelyessy P *et al.*, 2017). MRI characteristics can help in differentiating MOG-AD from other neuro-inflammatory disorders

including MS and NMO. MOG antibody associated optic neuritis seems to predominantly affect the retrobulbar region, while AQP-4 associated optic neuritis found intracranially (Anonymous 1).The management of acute attacks includes high dose of oral or intravenous Methyl Prednisolone, plasma exchange, intravenous Immunoglobulin and Cyclophosphamide.

CASE STUDY

A 39 years old male patient admittedto Neurology department with complaints of diminished vision in left eye followed by right eye since 1 month which is gradual in onset and progressed within 5 days. Other complaints were weakness of both lower limbs associated with urinary incontinence since 5 days. Patient also presented with history of constipation, generalized body pains, and reduced capacity to work and fatigue. Patient is a not known case of hypertension or diabetes mellitus. At the time of admission patient was conscious and coherent, blood pressure was 110/70 mm of Hg, pulse rate was 70 bpm. On neurological examination, power of both lower limbs was 4/5 and relative afferent papillary defect, ataxic gait were evaluated.

Serum electrolytes, renal function tests and liver function tests were found within normal limit. MRI scan of dorsal spine showed T_3-T_{11} hyper intensity in retrobulbar region and MRI with contrast study showed altered signal intensity in cervical and dorsal spinal cord with patchy enhancement suspicious of demyelination. Anti-nuclear antibody (ANA) ELISA test revealed positive 2+ homogenous pattern for ANA by indirect immunofluorescence, indicating the presence of auto antibodies. Serum analysis for the presence of Anti-MOG antibodies was depicted in Table 1.

With the evidence of laboratory investigations, patient was diagnosed with “MOG antibodies associated bilateral optic neuritis with demyelination". Treatment was started with 5 cycles of plasmapheresis, also known as Plasma exchange (PLEX), which is often recommended for moderate to aggressive forms of optic neuritis. Due to poor prognosis of the disease, patient was advised to be in observation for 15 days and planned for 2 doses of Rituximab 500mg. Meanwhile symptomatic treatment was also given.

After giving 2 doses of Rituximab 500mg, patient was stabilized and symptoms were relieved. So patient was discharged with the medications: Prednisolone 20mg with prolonged taper in view of long term treatment, Pantoprazole 40mg once daily before breakfast, Calcium and multivitamin once daily and monthly Cyclophosphamide during follow up to reduce the number of relapses.

DISCUSSION

Even though MOG is a minor component of the CNS myelin sheath, accounting for less than 0.5% of its composition, many of its episodes have been demonstrated to be highly immunogenic both in rodents and humans (Passos GR, 2017). Most patients with MOG antibody associated diseases have favorable outcomes, but a subset are left with permanent disability, usually as a result of the initial attack (Reindl M, Waters P, 2019). Early detection of MOG antibodies may assist with the differentiation of this condition from AQP-4 antibody positive NMO and MS, and MOG antibodies may prove to be an essential bio-marker with diagnostic and therapeutic implications (Ramanathan S *et al*., 2014).Among patients with AQP-4 seronegative NMOSD, the frequency of a positive MOG antibody test ranges between 7.4% and 39% (Lechner C *et al*., 2016).

Our observation in the case study was the patient responded well to the steroid therapy and visual acuity outcomes were also favorable. Patients who fully recover vision after optic neuritis may experience transient returns of blurred vision during time of stress, exertion or heat exposure. One study found that the rate of treatment failure was lower in patients on maintenance steroids (5%; median treatment duration of 10 months) compared to non-steroidal maintenance therapies (Ramanathan S, *et al*.,).

The role of oral steroids as adjunctive treatment with immunosuppressants remains unclear. A study found that patients with MOG antibody ON and/or myelitis treated with Azathioprine, but not adjunctive with oral steroids, experienced relapses more commonly than patients who underwent combination (Jarius S, *et al*.,).But immunosuppressants carry a risk of infections, particularly upper respiratory tract infections and urinary tract infections. So, good hygiene and hand washing are important if on immunosuppressants. The MOG-seropositive patients show more benign clinical course with a lower relapse rate and a longer time to a second attack affecting different CNS region compared to AQP-4 seropositive and seronegative patients (Probstel AK *et al*., 2015).

Table1. **MOG-Antibody test**

MOG-NMOSD-SERUM	
Anti-myelin oligodendrocyte glycoprotein (MOG) IgG antibodies	**STRONGLY POSITIVE**
Anti-Aquaporin-4 (NMO) IgG antibodies	**NEGATIVE**

Table 2. Complete blood count

LAB PARAMETER	OBSERVED VALUE	NORMAL VALUE
Hemoglobin	**13.2 g/dL** ↓	14-18 g/dL
Red blood cells	**4.23 mill/cu.mm** ↓	4.5-6 mill/cu.mm
White blood cells	**12,900 cells/cu.mm** ↑	4000-11,000 cells/cu.mm
Platelets	2.33 lakhs/cu.mm	1.5-4 lakhs/cu.mm
Erythrocyte sedimentation rate	**114 mm/1st Hr** ↑	0-9 mm/1st Hr

Table 3. Pharmacological Treatment provided

S. No	DOSAGE FORM	MEDICATIONS	DOSE	ROUTE	FREQUENCY
1	Tablet	GABAPENTIN+NORTRYPTILINE	400/10 mg	Oral	BD
2	Tablet	SHELCAL (Calcium)	500 mg	Oral	OD
3	Tablet	PANTOPRAZOLE	40 mg	Oral	OD
4	Capsule	REJUNEX (Multivitamin)	-	Oral	OD
5	Injection	Methyl Prednisolone	1g in 500ml Normal saline	Intravenous	BD
6	Tablet	OROFER-XT (Ferrous ascorbate+Folic acid)	100 mg	Oral	BD
7	Tablet	CEFIXIME	200 mg	Oral	BD
8	Injection	RITUXIMAB	500 mg in 500ml Normal saline	Intravenous	2 doses in 15 days

CONCLUSION

This case implies that the early diagnosis of MOG-antibodies can differentiate the demyelination from AQP-4 antibodies positive demyelination and can treat accordingly. MOG antibody associated optic neuritis is an immunological entity with a characteristic clinical and therapeutic profile. MOG antibodies are increasingly recognized in adult patients with inflammatory CNS demyelination with a spectrum encompassing NMOSD, ADEM and unilateral or bilateral isolated optic neuritis. Currently, there are no evidence bases guidelines for the acute treatment of patients with MOG antibodies. For MOG antibody associated disease, evaluation of visual function as well as gait assessment and evaluation of bladder function are more relevant. It is necessary to test patients longitudinally to assess the Anti-MOG serostatus. Rehabilitative care is needed to prevent secondary complications of immobility and to improve functional status. Further studies with larger cohort should be needed to consolidate the findings and it potentially lead to therapeutic recommendations in majority of the MOG-seropositive patients.

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CONFLICT OF INTEREST

Nil.

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