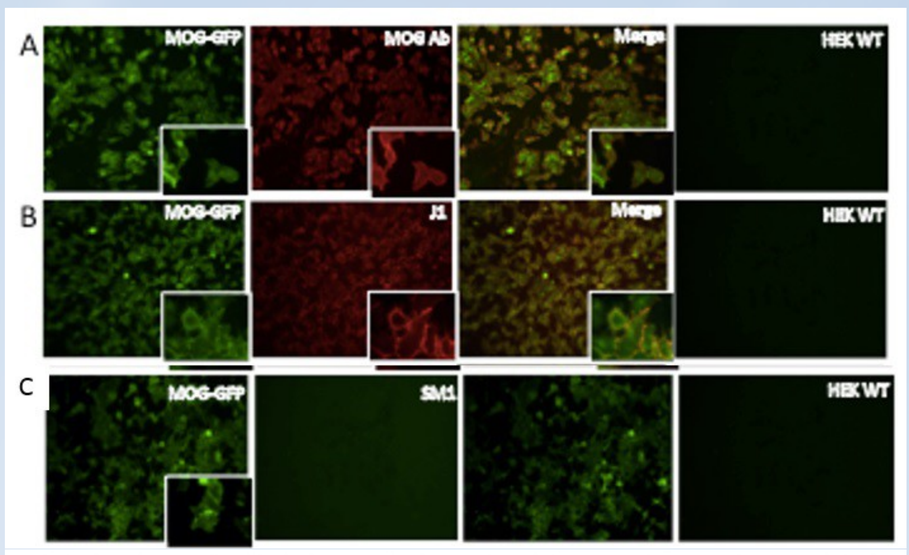
ANTI-MYELIN OLIGODENDROCYTE GLYCOPROTEIN (MOG) ANTIBODIES IN A PATIENT WITH RECURRENT OPTIC NEURITIS

V. FELICA[1], E. D'ERRICO[1], I. TEMPESTA[1], E. DISTASO[1], A. FRIGERI[1], M. MASTRAPASQUA[1], N. CAMPOBASSO[2], E. CAPUTO[3], M. RUGGIERI[1], I.L. SIMONE[1], AND C. TORTORELLA[1]

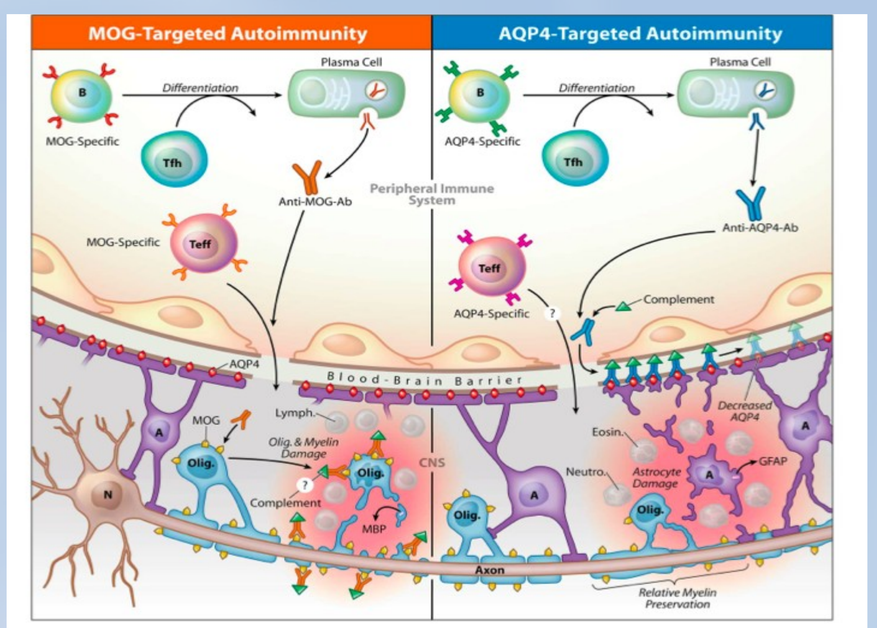
[1]*Department of Basic Medical Sciences, Neurosciences and Sense Organs, University of Bari "Aldo Moro", Bari, Italy*
[2]*Department of Emergency and Organ Tranplantation (DETO), Section of Endocrinology and Metabolic Diseases, University of Bari "Aldo Moro", Italy*
[3]*U.O. Neurology, OGR "F. Miulli", Acquaviva delle Fonti, Italy*

INTRODUCTION

Myelin oligodendrocyte glycoprotein (MOG) localizes on the outermost surface of the oligodendrocytes myelin sheath in the central nervous system (CNS). Anti-MOG autoantibodies are detected in patients with various inflammatory demyelinating diseases of the CNS, including acute disseminated encephalomyelitis, multiple sclerosis and neuromyelitis optica spectrum disorders. Recent studies emphasized an association between anti-MOG antibodies and recurrent optic neuritis, suggesting the possibility to include this phenotype in neuromyelitis optica spectrum disorder.



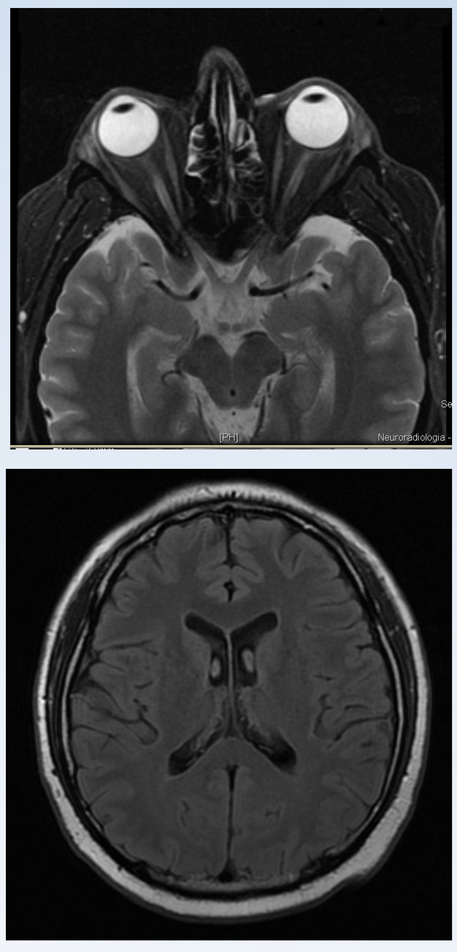
Cell based immunofluorescence assay (CBA) performed on HEK 293 expressing human MOG Alpha-1 fused to GFP. The GFP was inserted to evaluate the expression levels of MOG (green staining).
In red is shown the immunofluorescence staining using commercial antibody (A), our positive patient (J1) serum (B) and one negative serum of a Multiple Sclerosis patient. (SM1) (C). Non transfected cells (HEK WT) were used as negative control. Magnification 20X. Insert shows an higher magnification (100X) of the staining on a single transfected cell.

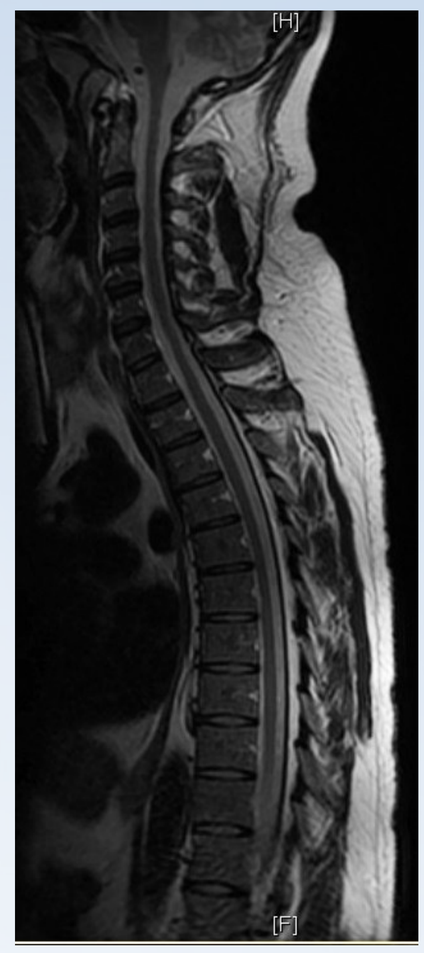


Zamvil S and Slavin AJ. Does MOG Ig-positive AQP-seronegative opticospinal inflammatory disease justify a diagnosis of NMO spectrum disorder? Neurol Neuroimmunol Neuroinfiamm 2015

CASE REPORT

A 35 years old caucasian man was admitted to our Department because of steroid dependent recurrent optic neuritis with serum anti-MOG antibodies. His prior medical and family history was unremarkable except for six relapsing inflammatory optic neuritis (in one case bilateral) all of which successfully treated with IV Methyl-Prednisolone (12 mg/kg/day for 5 days) followed by oral Prednisolone. New optic neuritis ever occurred during oral Prednisolone tapering. Serum autoantibodies test were unremarkable. The cerebrospinal fluid examination was normal, brain and spine MRI was negative except for slight hyperintensity in right optic nerve on STIR sequence, compatible with right optic neuritis. Slight nerve conduction disturbance in pattern reversal visual evoked potentials was showed in right optic nerve, whereas no response was detected in left optic nerve. At the admission to our Department, the patient was asymptomatic, but he was under Prednisone therapy (50 mg/day since more than three months). Search for anti-AQP4 antibodies was negative, whereas high anti-MOG antibodies titer was detected. In order to improve steroid dependency, the patient was treated with selective apheresis (IgG immunoadsorption), one session every other day for a total of ten sessions. A progressive reduction of serum anti-MOG antibodies titer was observed and the patient started a very slow oral Prednisolone tapering. After 50 days from the last apheresis session we documented an increase of serum anti-MOG antibodies titer and patient presented a new left optic neuritis. Immunosuppressive therapy with azathioprine was proposed.





CONCLUSIONS

Patients with isolated optic neuritis in absence of MRI features suggestive of MS can be candidates for analyzing anti-MOG antibodies.
Even if the pathogenic relevance of such antibodies is not fully understood they seems to predict optic neuritis recurrence similarly to anti-AQP4 antibodies, but with a better long-term prognosis.
Selective apheresis seems to result only in short-term reduction of antibody titer and may be useful to treat unresponsive clinical relapse, but it seems to be ineffective in long-term.
In recurrent and steroid dependent forms immunosuppressive therapy should be recommended.

References
1.Pröbstel AK, Rudolf G, Dornmair K, et al. Anti-MOG antibodies are present in a subgroup of patients with a neuromyelitis optica phenotype. J Neuroinflammation. 2015 Mar 8;12:46.
2.Chalmoukou K, Alexopoulos H, Akrivou S, et al. Anti-MOG antibodies are frequently associated with steroid-sensitive recurrent optic neuritis. Neurol Neuroimmunol Neuroinflamm. 2015 Jul 2;2(4):e131.
3. Wingerchuk DM, Banwell B, Bennett JL, et al. International Panel for NMO Diagnosis. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015 Jul 14;85(2):177-89.

Sin
XLVII CONGRESSO NAZIONALE
22-25 OTTOBRE 2016 – VENEZIA
WebPoster