Pediatric optic neuritis with myelin oligodendrocyte glycoprotein (MOG) antibodies

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Introduction

Anti-myelin oligodendrocyte glycoprotein (MOG) antibodies have been described as being associated with several pediatric demyelinating syndromes, including ADEM, recurrent ON, NMO-like disease, and ADEM-like onset followed by monophasic or recurrent ON. Patients with MOG antibody-associated demyelination appear to have a unique clinical, radiological, and therapeutic profile, which represents a major advance in their diagnosis and management.
The purpose of this abstract is to describe the clinical and neuroimaging features of two pediatric patients with inflammatory demyelinating disease associated with positive MOG antibodies.

Methods

We select patients who came to our attention in the last year with diagnosis of inflammatory demyelinanting disease (IDD) and positive serum MOG antibodies (MOG-ab). For all patients the following examinations were required at onset: neurological exam, brain and spine MRI with gadolinium (gd), determination of oligoclonal bands (blood and CSF) and anti MOG dosage in serum. Patients with secondary causes of demyelination were excluded. Diagnosis of IDD subtypes was made according to International Pediatric Multiple Sclerosis Group (IPMSSG) 2012 criteria.

Results

In a period of one year (may 2014-may 2015) we saw 8 patients who received diagnosis of IDD. Two patients were classified as multiple sclerosis, two patients with optic neuritis-MOG positive, one patients with ADEM and two patients with clinically isolated syndrome.
The two patients with MOG antibody included a 3 years old boy and a 7 years old girl. Both patients presented optic neuritis at onset, (bilateral and monolateral respectively) confirmed by visual evoked potentials, negative chemical and physical LCR analysis, negative BOG and AQ4. Spine MRI was normal for both patients while brain MRI showed lesions of thalami and basal ganglia and post gd hyperintensity with thickening of optic nerves. Patients were both treated with 5 days of endovenous methylprednisolone 20 mg / Kg/ day followed by oral prednisone 2mg/Kg/day for two weeks followed by progressive discontinuation in other 2 weeks. Both presented complete clinically recovery into one month. The girl showed complete normalization of MRI at neuroradiological follow up after 3 months. The boy has not yet performed follow up MRI.

Conclusions

The spectrum of IDD-MOG related is in ongoing definition and the pathogenic role of MOG has not yet completely defined. We describe two patients with overlapping clinical and neuroradiological picture. Our suggestion is that patients with isolated optic neuritis (also without ADEM) and MRI showing involvement of basal ganglia or thalami should undergo to serum MOG ab detection.

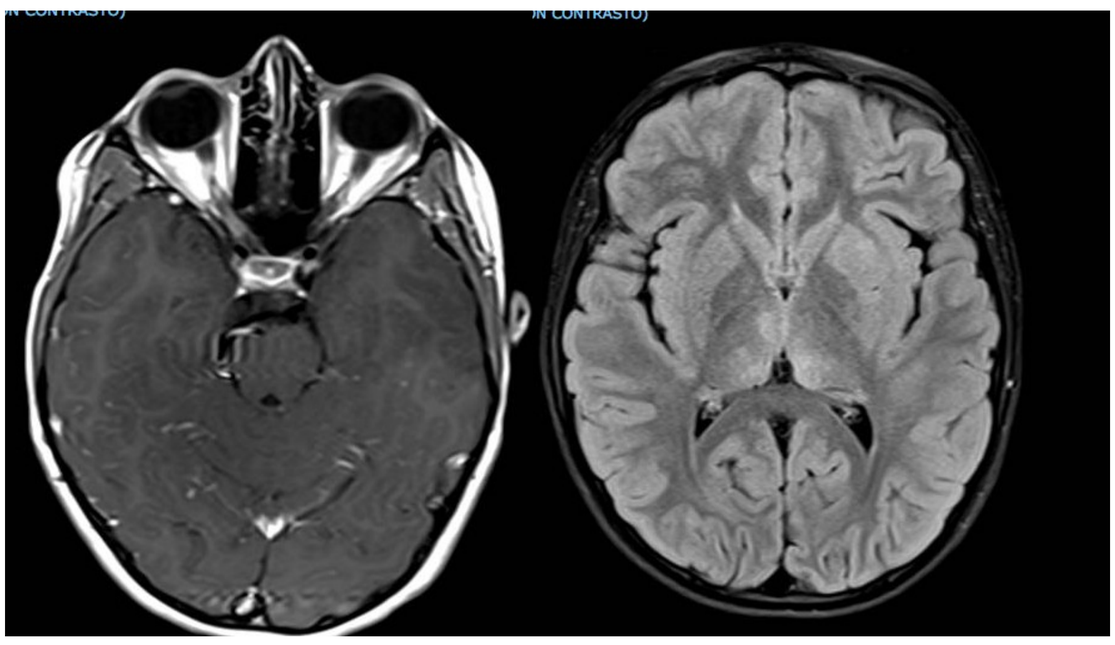

Fig. 1 Brain MRI of the 7 years old girl showed optic nerves thickened and tortuous with post-Gd enhancement and T2-Hyperintensive lesions of thalami and basal ganglia.

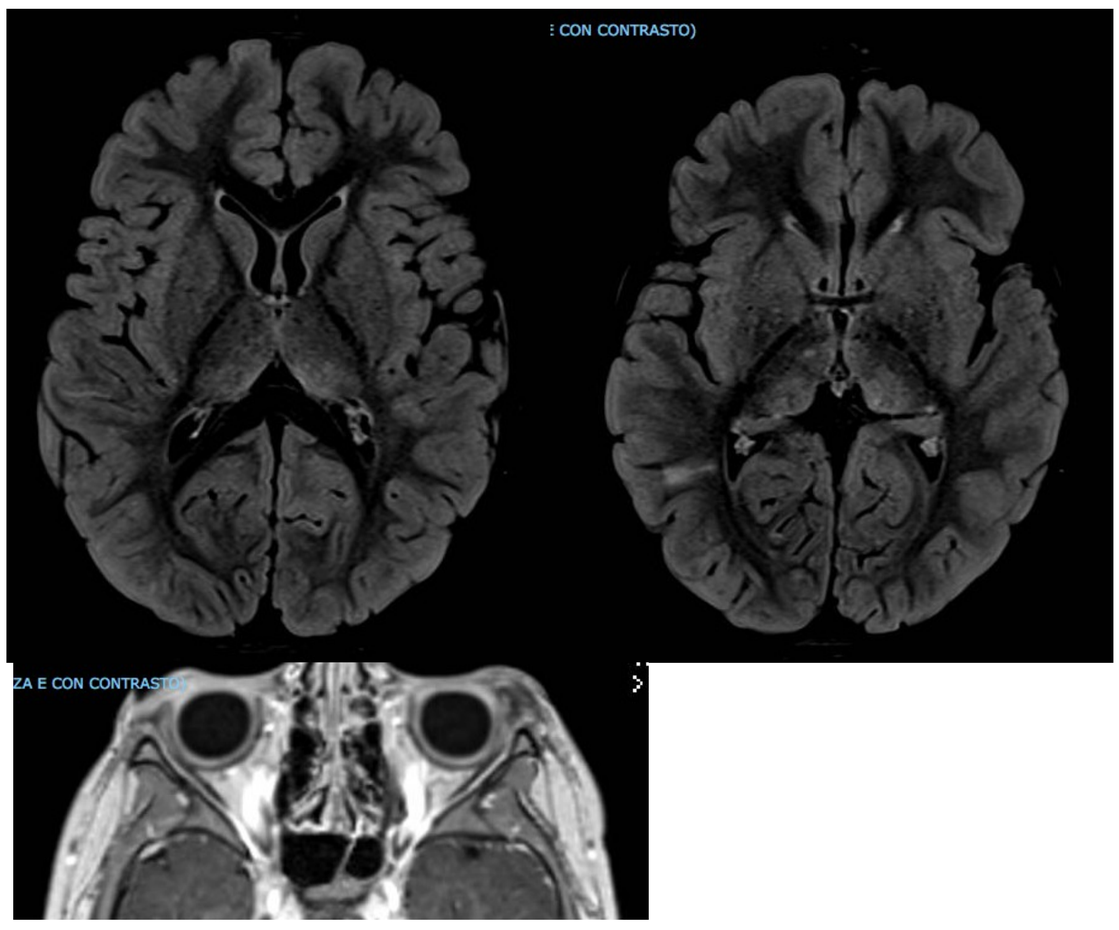

Fig. 2 Brain MRI of the 3 years old boy showed optic nerves thickened and tortuous with post-Gd enhancement and T2-Hyperintensive lesions of thalami and right parietal subcortical white matter.

References

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