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Case Report

Anti-Myelin Oligodendrocyte Glycoprotein Antibodies Demyelinating Disorder in an Iraqi child: A Case Report

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Abstract

Anti-MOG associated demyelination is increasingly being recognized as a cause of recurrent CNS demyelination in the last decade. This is a case report of a 5 years old male child presented with disturbed level of consciousness and seizures where he admitted and treated as a case of CNS infection then the multi-focal neurological deficits points to a polyfocal CNS demyelinating disorder found to be an Anti-MOG associated demyelination with recurrent relapsing and remitting events.

***Keywords:** Case report; CNS; Central Nervous System; MOG; Myelin Oligodendrocyte Glycoprotein.*

Abbreviations

abs = antibodies
ADEM = Acute disseminated encephalomyelitis
ADEM-ON = ADEM with recurrent optic neuritis
ANA = Anti-Nuclear Antibodies
AQP4 = Aquaporin-4
ADS = Acquired demyelinating syndromes
CNS = Central nervous system
CRION = Chronic relapsing inflammatory optic neuropathy
CSF = Cerebrospinal fluid
EM = Encephalomyelitis
Gd = Gadolinium
IA = Immunoadsorption
IgA = Immunoglobulin A
IgG = Immunoglobulin G
IgM = Immunoglobulin M
IVIG = Intravenous immunoglobulin
IVMP = Intravenous methylprednisolone
LETM = Longitudinally extensive transverse myelitis
LP = Lumber puncture
MOG = Myelin oligodendrocyte glycoprotein
MOGAD = Myelin oligodendrocyte glycoprotein associated demyelination
MRI = Magnetic resonance imaging
MS = Multiple sclerosis
NMO = Neuromyelitis optica
NMOSD = NMO spectrum disorder
OCB = Oligoclonal IgG bands
ON = Optic neuritis
TM = transverse myelitis
VEP = Visual evoked potentials
VS = Vertebral segments
WCC = White cell count

Introduction

Paediatric acquired demyelinating syndromes (ADS) consist of many diseases induced by the immune system causing demyelination in different parts of the CNS, like ADEM, ON, TM, NMOSD and MS. The diagnosis of which type is difficult at first, but with time can be made with more accuracy [1-4].

When anti-aquaporin-4 antibodies (AQP4-abs) were identified in 2004, AQP4-ab-positive NMOSD became more practical to be diagnosed with confidence [5]. Taking more lights in the last years, were the anti-myelin oligodendrocyte glycoprotein antibodies (MOG-abs) as an important cause in CNS demyelination [6,7]. Myelin oligodendrocyte glycoprotein are found on the outer surface of the myelin sheath and plasma membrane of oligodendrocytes, where it is attacked by those antibodies [8,9].

MOG represents a small component of myelin (less than 1%), but due to the high CNS specificity and outer location make it more prone for those antibodies' attacks [6,9]. After their discovery, MOG-abs were thought to be related to MS, but with advances in the assay techniques [10], many studies had showed that those antibodies only found in little patients with MS [11-13]. Now those MOG-abs accepted to be a special disease entity different from MS [12,14]. Those antibodies are more encountered in the paediatric population as compared to adults [15-17].

MOGAD is more commonly encountered in paediatric. When reviewing the newest studies, we found that the major prevalence of MOG-abs in paediatric (40%) compared to adult (22%) cohorts [16]. The corner stone of detecting the MOG-abs is the live cell-based assay (CBA), which is used in many studies [11-14,17-24]. Depending on results from those studies, 34% of the paediatric patients with ADS were found to have MOG-abs (34%; Fig. 1).

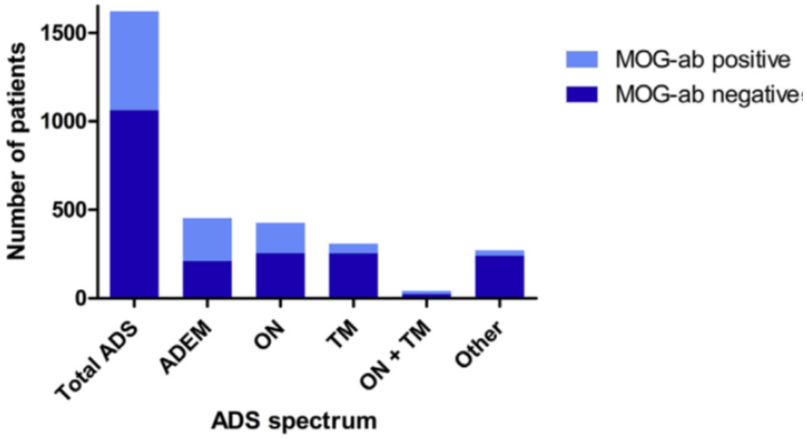

Figure 1: *ADS presenting phenotype, divided for MOG-ab-positive and negative patients [11-14,17-23].*

ADEM = acute disseminated encephalomyelitis, ADS = acquired demyelinating syndrome, MOG-ab = myelin oligodendrocyte glycoprotein antibody, ON=optic neuritis, TM = transverse myelitis.

According to the studies, MOG-abs are found mostly in patients with ADEM (53%), ON (40%), and TM (18%). Those MOG-abs positive patients presenting with ON and TM at the same time simulating a NMOSD represent a limited number of patients in different studies [11,13,14,21,23].

Looking to Fig. 2, we can see a summary of 13 studies about the multiple presentations of MOGAD in paediatrics at the start of the disease [11-14,17,20-27].

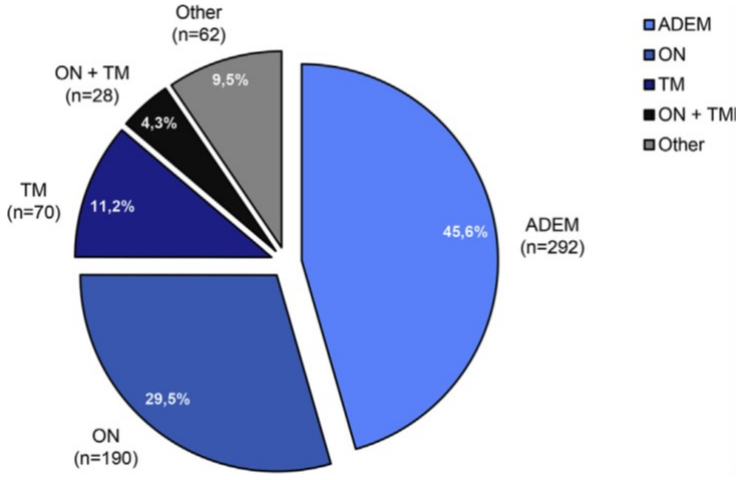

Figure 2: *Presenting clinical phenotypes within the paediatric MOGAD [11-13,17,20-27].*

Most of those patients in the studies are from Caucasian origin [12,17,28,29]. Females outnumber males in some studies but in the majority of studies there is equal male to female ratio [11-14,17,20,23,26,28,29,31]. In MS and NMOSD there is clear female predominance as compared to MOGAD [30,32,33].

The incidence of MOG-abs in paediatric ADS is more common in younger age groups than those with negative MOG-abs which could be explained by the higher incidence of ADEM in those younger age groups [11,13,14,20,28, 31, 33-37].

It is generally accepted that which clinical presentation of those paediatric patients who are tested positive for MOG-abs will be clearly dependent of the age group with ADEM being the most common in younger (less than 9 years and ON with or without NMOSD in older (more than 9 years) children [13,16,25,28], and adults [17, 38-41].

Case Report

A 5 year old male child named Muhamad Ahmed presented with abnormal body movement for half an hour before admission to the emergency room of the Child's Central Teaching hospital, one of the major Pediatric hospitals in the capital Baghdad.

The condition started as a flu-like illness 2 weeks before the admission with runny nose and low-grade intermittent fever mostly at night. The patient was kept at home for symptomatic treatment.

Within the next 4 days he developed recurrent attacks of bi-temporal headache with no vomiting, and relieved slightly by sleep and simple analgesia. Next day there was increased headache frequency and intensity and became associated with bouts of vomiting about 3-4 times daily for the next 2 days.

The child became lethargic, sleepy, spending most of his time at bed. At the 7th day of illness, he developed abnormal body movement as upward rolling of eyes with clenching teeth lasting for about 1 minute followed by secondarily generalized tonic-clonic seizure lasted for about half an hour reaching the hospital to be stopped by intravenous diazepam.

Following the convulsion, the patient didn't regain his consciousness and lapse in a comatose state during which he was only responding to painful stimuli by withdrawal.

Random blood sugar, complete blood count, C-reactive protein, renal function test, liver function test, serum electrolytes, and toxicology screen were all normal.

A brain CT was requested which showed bilateral ill-defined brain parenchyma hypodensities with area of exaggerated white/gray matter interface and suggest the need for brain MRI which was very difficult to be done according to the patient condition.

The patient was admitted to the ward of infectious diseases as a case of meningo-encephalitis (without LP and CSF analysis as he was in a critical state of illness).

He was started empirically on anti-meningeal antibiotics with acyclovir and dexamethasone. He remained in comatose state for the first 24 hours when brain MRI done then after.

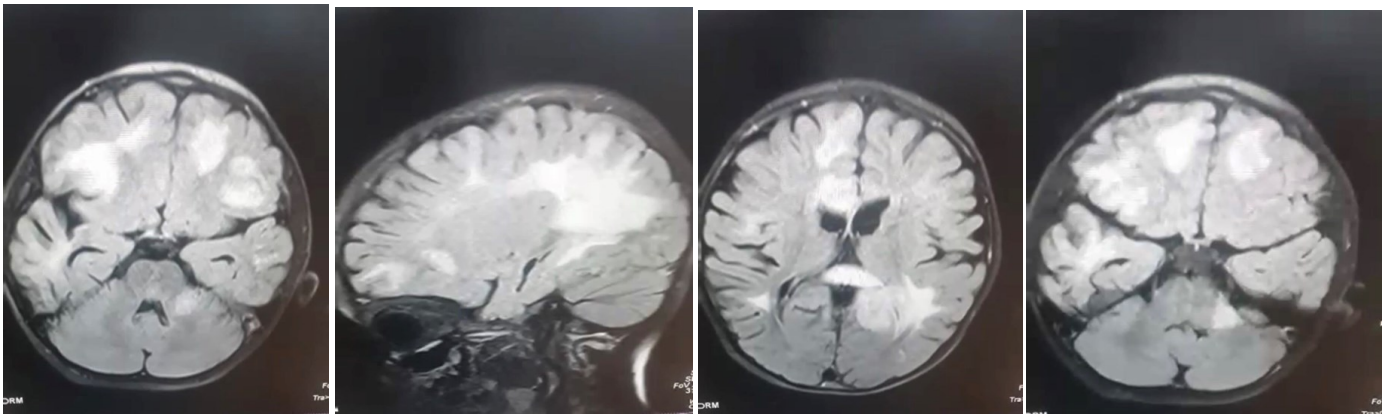

Figure 3: *First brain MRI showing bilateral asymmetrical large hazy hyperintense lesions involving cortical, subcortical, deep white and grey matters where he is regarded as a case of ADEM.*

Before start treatment the patient was regarded as a case immune mediated encephalopathy syndrome so sent for following investigations before starting immune modulating agents:

- ANA, Anti-dsDNA.
- Anti-c-ANCA Anti-Neutrophil Cytoplasmic Ab anti PR3.
- Anti-p-ANCA Anti-Neutrophil Cytoplasmic Ab anti-MPO.

- Anti-NMDA (N-methyl-D-aspartate)- Ab.
- Anti-MOG (Myelin Oligodendrocyte Glycoprotien) Ab.
- Anti-Aquaporine 4 Ab.

The patient was started on methyl prednisolone pulse steroid as 30 mg/kg per day for the next 5 successive days.

At about the 3rd day of treatment the patient started to regain his conscious level but he can't see his parents and only identify them by their voices.

At the 4th of November 2018 when the patient was receiving the fifth dose of pulse steroids, he was conscious and alert but had the following complains:

- Bi-frontal headache associated with pain behind both eyes and visual changes (although improving).
- Behavioral changes as he became aggressive with periods of shouting and hitting his family members on the reverse of his previous quiet nature.
- Slow and monotonus speech only single words.
- Difficulty swallowing for both solids and liquids with drooling of saliva and sometimes chocking.

On examination:

General medical and systemic examinations were unremarkable. Vital signs were normal. Growth parameters within the median values. His neurological examination as follows:

- Mental status: alert and cooperative.
- Speech: single slurred words with monotonus quality but can comprehence, name objects, and repeat some words although with difficulty.
- Meningeal signs were negative.
- Cranial nerves: patient can't effectively cooperate for visual acuity and visual field examinations and have normal pupillary light reflex but bilateral congested optic discs.
- Both eyes tracks moving objects in all directions without limitation, fragmentation, nystagmus, and no claimed diplopia.
- Jaw jerk just present.
- No facial asymmetry.
- Uvula midline and moves slowly with diminished gag reflex.
- Tongue midline without atrophy or fasciculation.
- Motor system examination show normal tone in the 4 limbs with power up to the grade of 3+ to 4- with normal reflexes in all limbs and upgoing planter responses.
- Cerebellar signs were negative.
- Sensation: needs more patient cooperation but roughly the patient feels the pinprick and resist this test said it hurts.
- Gait: the patient was ambulatory with no abnormal gait pattern.

At the 7th day the patient had shown dramatic improvement and discharged home on oral prednisolone taper over 2 weeks pending the results of investigations.

About 16 days after discharge (2 days after end of steroid), patient came back with recurrent headache with pain behind both eyes and within the next 2 days there was progressive visual loss, that increased gradually to the level of only light perception over about 36 hours.

Patient was admitted again and neurological examination was only significant for bilateral afferent pupillary light response, congested optic discs and markedly reduced visual acuity.

Patient admitted again at 26th of November 2018 and at the time of admission his investigations had arrived as follows:

- ANA 1:80 (normal range up to 1:40).
- Anti-NMDA-IgG antibodies 1:10 (normal range < 1:10).
- Anti-Aquaporine 4 negative.
- Anti-MOG-IgG antibodies 1:100 (normal range < 1:10).
- Other antibodies were negative.

New brain MRI with gado with special orbital view was recommended especially looking for optic nerve enhancement and new white matter enhancing lesions. It showed the same previous lesions with even signs of improvement of some lesions and unfortunately the orbital view with fat suppression was not done.

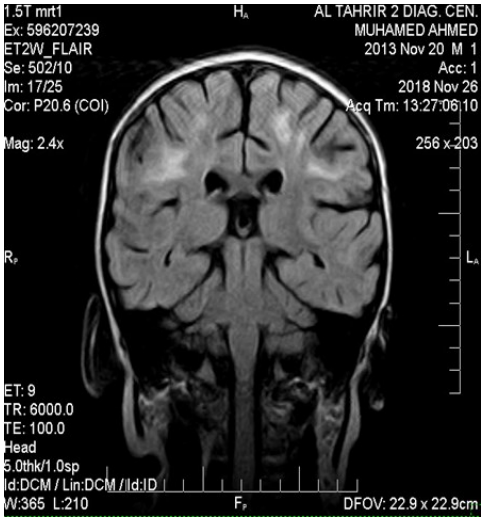

Figure (2) brain MRI done 3 weeks after receiving treatment showing marked improvement in demyelinating lesions, although the patient presented with optic neuritis.

The patient started again on pulse steroids with rapid improvement in the next 2 days and discharged after the third dose of pulse steroids with normal visual acuity.

This time discharged on oral steroids and mycophenolate as adjuvant drug because frequent relapses are expected.

Follow up

First visit after 7 days, the patient was doing well on a low steroid dose (20 mg prednisolone/day) and mycophenolate.

Second visit after 14 days, patient doing well and start to taper steroids gradually.

About 4 days from starting steroid taper there were attacks of headache although mild with rapid response to analgesia, and the mycophenolate dose increased.

About 21 days from discharge and 7 days from start of steroid taper there was recurrence of visual disturbance and at about 23 days after_discharge and 9 days from the steroid taper the patient can see about only 1 meter with headache and painful eye movements.

Second relapse admission and workup

- CSF analysis and CSF for oligoclonal bands and IgG index.
- Visual evoked potential.
- Stop the oral steroids and mycophenolate and restart pulse steroids.

Patient rapidly respond to pulse steroids and from the second dose about had a normal visual acuity.

CSF analysis show: sugar 83 mg/dl (RBS 180 mg/ dl), protein 22 mg/dl, WBCs 28 cells (90% lymphocytes, 8% neutrophils, 2% monocytes), negative Gram stain, negative ZN stain, negative film for fungus, no abnormal cells. CSF IgG index 0.74 (mild elevation as normal value < 0.7). CSF IgG oligoclonal bands negative.

Visual evoked potential showed evidence of demyelinating lesion of sever degree on both sides but left > right chiasmatic and pre- chiasmatic.

Patient decided to be started on Rituximab but the family refuse and so kept on oral steroids for next 3 months and was doing well.

Then lost follow up and the family stopped treatment and the patient came back again with disturbed level of consciousness and frequent seizures where he admitted in a critical state of illness. The brain MRI at that presentation shown large hazy lesions.

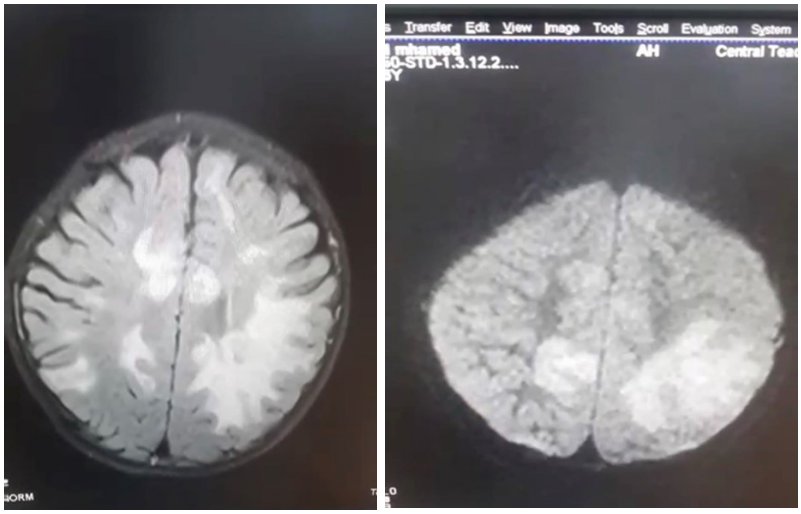

Figure (3) Brain MRI after the lost follow up about 6 months from the first encephalopathy attack showed multiple new lesions cortical and subcortical with large left sided temporo-parietal confluent lesion.

At that admission his Anti-MOG result was 1:320. Patient respond well on pulse steroids and started on Rituximab 4 doses on 4 successive weeks. Also started on levetiracetam to control the recurrent seizures as a result of that attack. The patient was doing well for next 3 months then he relapsed again with optic neuritis.

Clearly the patient was responding only to steroids and any aim to stop steroids and even decrease the dose ends with recurrent optic neuritis with the risk of having ADEM like presentations. So, in the last one year from Jan 2021 the patient started on IVIG with successful steroid taper then stop and the patient is now about 1 year free from attacks on IVIG doses every 2-3 months and developing normally.

Unfortunately, from those frequent attacks he developed epilepsy controlled on levetiracetam and right sided optic atrophy.

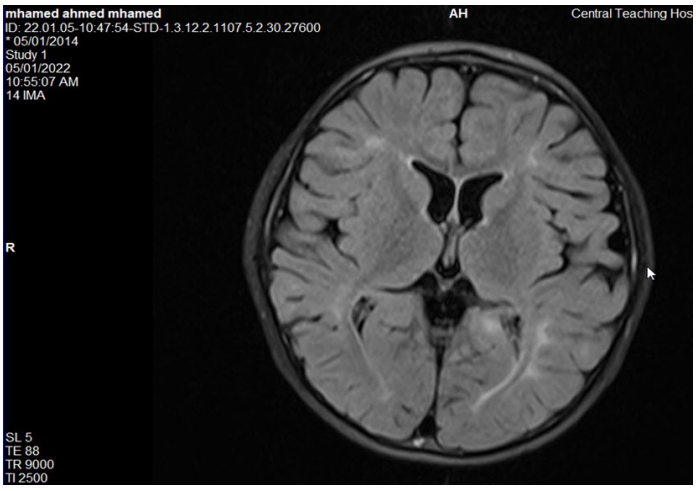

Figure (4) Last brain MRI on the 5th of January 2022 showing clear improvement of lesions on IVIG bolus doses every 2-3 moths without steroids.

Discussion

Due to the advances in the development of the cell-based assays aid in the discovery of new antibodies targeting the human myelin oligodendrocyte glycoprotein (MOG-IgG) called MOG-abs, presenting with different phenotypes but mostly with ADEM, ON, ADEM-ON, NMOSD, and brain stem encephalitis. Now MAGAD is considered a separated disease from MS and other CNS demyelinating syndromes [42].

Because of similarities of presentation (especially in patients with recurrent ON) with MS patients and to prevent over-diagnosis, special criteria to diagnose MOGAD were indicated [42].

Criteria for MOG-IgG testing in patients presenting with acute CNS demyelination: [42]

First attack or recurrent ON, or to lesser extent, myelitis, brainstem encephalitis, encephalitis, or any combination of them:
plus radiological or VEP support of CNS demyelination (note VEP support required in patients presented with ON) **plus** one of the followings:

MRI

a. Longitudinally extensive spinal cord lesion or spinal cord atrophy (LETM ≥3 VS, contiguous) on MRI or lesions of Conus medullaris.

b. Extensive optic nerve lesion (e.g., > half of the length of the pre-chiasmal optic nerve, on T2 or T1 with gadolinium enhancement) or Perioptic gadolinium enhancement during acute ON

c. The Brain MRI lesions are not periventricular, not ovoid, and not involving the corpus callosum

d. The lesions are large, and confluent.

Fundoscopy

e. Papilledema or papillitis when there is attack or relapse with ON

CSF

f. Neutrophilic predominance or WCC pleocytosis of more than 50/μl

g. Absence of OCB at presentation and any next follow up analysis in the CSF

Histopathology

h. Demyelinating lesions with deposition of IgG antibodies and complement.

Clinical findings

i. Presentation with recurrent sever unilateral but especially bilateral ON at the same time leading to sever impairment of visual acuity or even loss of vision in one or both eyes during or after the attacks of ON.

j. Severe or recurrent attacks of acute myelitis or brainstem encephalitis
k. Presentation with ADEM, and especially with recurrent ADEM or ADEM-ON.
l. Acute encephalopathy presenting with either seizures, behavioural changes, or disturbed level of consciousness with radiological support of demyelination.
m. Symptoms and signs following a recent vaccination within 4 days to 4 weeks of illness.
n. Presentation like an area postrema syndrome with resistant nausea, vomiting and intractable hiccups.

Treatment response

o. Steroid-dependent symptoms (including CRION)

If a patient presented with ON and LETM, it is better to test AQP4-IgG and MOG-IgG at the same time as such presentation is common in both MOGAD and NMOSD and not in MS [43, 49].
The time of doing the MRI study is strongly related to the probability of finding LETM with shorter lesions may be detected at the onset or the recovery periods indicating the need for MRI follow up which is better to be in axial and sagittal sections. However, children who tested positive for MOG-IgG frequently have LETM at the disease onset [50].

MOG-IgG-positive patients mostly will have longitudinal extensive optic nerve lesions (4-5 segments = anterior intra-orbital segment, posterior intra-orbital segment, canalicular, intracranial, chiasmal) [52] in contrast to the MS patients which involve only over 1 segment (70%) or 2 segments (30%)[51].

Regarding brain MRI with contrast, in MOG-IgG-positive patients, lesions agreed to be non-ovoid/round periventricular, and non-Dawson's finger-type lesions which was also found in 100% of mixed adult ($n = 15$) and paediatric ($n = 6$) cohort and juxtacortical U fiber lesions in 95.2% [47, 48].

In an exclusive paediatric cohort, it had been found that a lack of Dawson's finger-type lesions confirmed in MOG-IgG-positive patients (98.6%); and U fiber lesions were absent in 94.2% of MOG-IgG-positive paediatric patients in the same study [50]

Regarding CSF analysis neutrophilic pleocytosis is found in MOGAD as well as in NMOSD. However, it is absent in MS [53].

CSF oligoclonal bands are present in a minority of MOGAD patients (10-13%) in some studies in contrast to their presence in 98% of MS patients [54]. This indicate that the presence of OCB doesn't preclude the diagnosis of MOGAD but follow up will show their absence or alternative diagnosis with taking into consideration also that not all MS patients have CSF OCB especially in Asian patients (e.g., 40–80% in Japan) [43,55].

Multiple studied shows frequent relapses of symptoms when starting taper of steroids including also this case report [43, 44, 45, 46, 56].

Conclusion

Anti-MOG associated demyelination is a special disease entity that is presented in children under 9 years of age, mostly with ADEM, recurrent ADEM, or ADEM-ON and over 9 years old especially with recurrent myelitis, ON and NOMSD-like phenotypes.

All children with Clinical presentation of ADEM especially those under 9 years of age and with large confluent and hazy demyelinating lesions on brain MRI should be tested for MOG antibodies with serial evaluation for those who were positive at first presentation.

Best treatment options for Anti-MOG associated demyelination, are steroids and IVIG.

The patient is now regarded typical case of Anti-MOG demyelinating disorder from the following collected criteria:

1. Simultaneous bilateral acute relapsing optic neuritis following ADEM.
2. MRI findings consistent with ADEM and then improving even with recurrence of ON and non-consistent of MS MRI findings.
3. VEP showing evidence of demyelination.
4. Optic disc swelling during each relapsing acute ON.
5. CSF negative for oligoclonal bands.
6. Negative serum aquaporin-4 IgG Ab.
7. Positive Anti-MOG IgG Ab in serum.
8. Frequent flare-ups after stop steroids, or steroid-dependent symptoms.

Conflict of Interest

The author declares no conflict of interest.

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