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MOG ANTIBODY ASSOCIATED ACUTE DISSEMINATED ENCEPHALOMYELITIS; A CASE STUDY BASED CLINICAL ANALYSIS

BY

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Abstract

Acute disseminated encephalomyelitis (ADEM) occurs mostly in children and can be triggered by infections and vaccinations. Recently, 40% of ADEM patients were found to be seropositive for myelin oligodendrocyte glycoprotein antibodies (MOG-abs)[1]. In addition, a subset of adult patients were negative for aquaporin-4 antibody, meeting the diagnostic clinical and radiological criteria for neuromyelitis optica- spectral disorder. NMOSD) contains high titer serum MOG-antibodies[2]. MOG antibody disease (MOGAD) is a neurological, immune-mediated disease involving inflammation of the optic nerve, spinal cord, and/or brain. Myelin oligodendrocyte glycoprotein (MOG) is a protein located on the surface of myelin sheaths in the central nervous system. Although the exact function of this glycoprotein is unknown, MOG is a target of the immune system in this disease. The diagnosis is confirmed when MOG antibodies are found in the blood of patients with recurrent attacks of central nervous system inflammation. The specific symptoms and severity of MOGAD can vary from person to person but include vision problems, symptoms related to spinal cord damage, and seizures. Treatment is given initially and is usually intravenous steroids, plasma exchange (PLEX), or intravenous immunoglobulin (IVIG). Those with MOG antibody disease should consider continuing treatment with immunosuppressive medications.

KEYWORDS: *IV Immunioglobulin, Myelin oligodendrocyte glycoprotein, ADEM*

1. INTRODUCTION

Antibodies are proteins that circulate in the bloodstream. They are part of the body's immune system (defense system) and are produced by a type of white blood cell (B-cell lymphocytes) that help attack and destroy viruses and bacteria and protect us from infection. Bacteria, viruses, and other. bacteria have proteins on their surface, called antigens. The immune system is programmed to recognize these antigens as "foreign" and the B-cell lymphocytes in turn produce many antibodies that attach to the infectious bacteria to destroy them. Sometimes the immune system is incorrectly programmed and produces antibodies. against brain protein. This can sometimes be caused by a simple infection, especially in children. Myelin oligodendrocyte glycoprotein (MOG) is a glycoprotein that is part of normal myelin and is found on the surface of the myelin sheath of neurons. Myelin is a fatty protein that insulates and protects nerves and helps maintain the speed at which messages are transmitted from the brain to the rest of the body. Antibodies to MOG loosen the myelin sheath, a process called demyelination. Demyelination means that messages cannot travel efficiently along the nerve, and those messages can slow down or stop altogether. This in turn can cause neurological (brain and nervous system) symptoms.

Acute disseminated encephalomyelitis (ADEM) is an acute autoimmune demyelinating disease of the central nervous system thought to be caused by a previous stimulus such as a viral infection or immunization. It is considered one of the most common demyelinating diseases of childhood, with an incidence of approximately 0.3–0.6 per 100,000 children per

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year[3] The average age of onset is 5–8 years, with a slight male predominance. According to studies, the male-to-female ratio varies from 1.2:1 to 2.6:1.1 2 Mortality is low (1% to 3%), but motor and neurocognitive deficits can persist 1 The presentation of ADEM can be variable and can exist constitutionally. various combinations of symptoms, altered mental status, and motility. and sensory disturbances and other neurological symptoms. Several studies have shown that although lesions improved on MRI, some children had milder cognitive deficits in certain domains, such as attention and visuospatial/visuomotor function[4,5]. Although ADEM is usually uniphasic, anti-myelin oligodendrocyte glycoprotein (anti-MOG antibodies) antibodies. antibodies are present,showed that it has prognostic value for relapsing multiphasic ADEM[6].The current literature only describes ADEM associated with anti-MOG antibody after influenza or Epstein-Barr virus (EBV) infection.6 To our knowledge, no cases related to MOG ADM. According to our knowledge, no antibodies caused by Mycoplasma pneumoniae infection have been reported.

2. DISORDERS WITH SIMILAR SYMPTOMS

The following disorders may have similar symptoms to MOGAD. Comparisons can be useful in differential diagnosis. MOGAD can be misdiagnosed as

- ✓ Multiple Sclerosis (MS)
- ✓ Neuromyelitis Optica Spectrum Disorder (NMOSD)
- ✓ Transverse Myelitis (TM)
- ✓ Acute Disseminated Encephalomyelitis (ADEM) because it causes inflammation, including damage to the brain, spinal cord, and optic nerve.

3. DIAGNOSIS

There are blood tests that can test for MOG antibodies. Cell-only assays are considered reliable for diagnosing MOGAD because they have improved over older ELISA tests. Lumbar puncture CSF analysis may show an increase in white blood cells during relapse in some patients, and oligoclonal bands are usually not found.Unlike anti-AQP4 antibodies, anti-MOG antibodies may decrease over time and may not be detectable early in the disease or during remission, especially in ADEM associated with MOG antibody disease. Those with persistently detected anti-MOG may be more likely to have recurrent rather than monophasic disease. There appears to be no overlap between people who are anti-MOG positive and AQP-4 positive, although isolated cases have been reported using the older ELISA test.MRI findings are similar to MS and NMOSD, but there may be some differences. Optic neuritis in MOG antibody disease appears to primarily affect the retrobulbar region, whereas AQP-4-related optic neuritis is seen intracranially. In addition, MOGAD lesions in the brain may resemble those seen in patients with ADEM.

4. STANDARD TREATMENT

4.1. ACUTE TREATMENTS

Treatment recommendations for MOG antibody disease have not been confirmed. Below are possible treatments for an acute event.

4.1.1. Intravenous Steroids

Although there are no clinical trials to support a unique approach to treating patients with MOG antibody disease, high-dose intravenous methylprednisolone is known to cause acute myelitis or optic neuritis. Usually, 3-5 days, unless there are good reasons not to continue. The decision to continue steroid therapy or add a new regimen is often based on clinical cost and MRI at the end of five steroid days. Those with MOG antibody disease respond well to steroids. Tapering off oral steroids can help prevent steroid withdrawal symptoms.

4.1.2. Plasma Exchange (PLEX)

PLEX is thought to act in autoimmune diseases of the central nervous system by removing specific or non-specific soluble factors likely to mediate, cause, or contribute to inflammatory organ damage. PLEX is often recommended for moderate to aggressive forms of TM and ON, as is very often the case with MOG antibody disease when there is little improvement after treatment with intravenous steroids. If symptoms are severe, PLEX therapy can be started at the same time as steroids. The efficacy of PLEX in MOG antibody disease has not been studied, but retrospective studies of TM treated with IV steroids followed by PLEX have shown a beneficial outcome. PLEX has also been shown to be effective in other autoimmune or inflammatory diseases of the central nervous system. Early treatment is beneficial - PLEX is usually started within a few days of steroid administration, very often before the end of steroid treatment. Particular benefit has been shown when initiated in the acute or subacute phase of myelitis or when active inflammation persists on MRI.

4.1.3. Intravenous immunoglobulin (IVIG)

Another option for treating acute inflammation caused by anti-MOG is intravenous immunoglobulin (IVIG). Immunoglobulin comes from blood donated by thousands of healthy people. As the name suggests, IVIG is administered intravenously. IVIG is generally well tolerated. Possible side effects are rare but usually occur during or shortly after the infusion, including headache, nausea, muscle pain, fever, chills, chest discomfort, skin, and anaphylactic reactions. Post-infusion reactions can be more severe and include migraine headaches, aseptic meningitis, kidney failure, and blood clots. As with corticosteroids and PLEX, there are no data to support the value of IVIG in acute events. Although most studies support the use of corticosteroids and/or PLEX

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in acute demyelinating syndromes, IVIG may be considered in certain cases.

4.1.4. Other acute treatments

If there is no response to steroids or PLEX therapy and active inflammation of the spinal cord persists, other immune-based interventions may be needed. In some patients, the use of immunosuppressants or immunomodulatory agents may be considered. Aggressive immunosuppression is considered when aggressive forms of myelitis are present at the onset or when it is particularly resistant to steroid and/or PLEX therapy. People should be carefully monitored because immunosuppression can lead to potential complications. As with all medications, the risks and benefits of aggressive immunosuppression should be considered and discussed with the clinical care team.

4.2. LONG-TERM TREATMENT

Anti-MOG was initially thought to be associated with fewer relapses and better outcomes in patients with AQP-4-positive NMOSD, but studies with longer follow-up periods show a higher rate of relapse. as previously reported.

A 2016 cohort study found that 80% of the cohort had multistage disease and an annualized relapse rate (AAR) of 0.9. They found that a third of patients with optic neuritis and about half of patients with spinal inflammation made a complete recovery. In contrast, two other studies showed that retinal neuroaxonal damage observed after an episode of acute optic neuritis was as severe in anti-MOG individuals as in individuals with AQP-4-positive NMOSD. People with MOG antibody disease should consider continued treatment with immunosuppressive medications. There are no FDA-approved medications to treat MOG antibody disease, so all prescriptions are off-label. The main treatments used in the United States are mycophenolate mofetil (CellCept), rituximab (Rituxan), azathioprine (Imuran), and repeated IVIG infusions or subcutaneous immunoglobulin. A number of UK studies have supported the use of IVIG to prevent relapse. Some patients with optic neuritis or transverse myelitis who also test positive for MOG antibodies may begin treatment after the initial event if the attack was severe and the person does not want to risk recurrence. All of these medications carry the risk of infection, especially upper respiratory infections and urinary tract infections (UTIs). Good hygiene and hand washing are important when taking immunosuppressants, as is a good urologist if you are at risk for UTIs. All of these drugs also carry the risk of developing a rare brain infection called progressive multifocal leukoencephalopathy, or PML. PML is an infection caused by the reactivation of the JC virus that lives in the kidneys. In an immunocompromised person, this virus can escape from the kidneys, cross the blood-brain barrier, and enter the brain, causing profound inflammation. Although treatable, it is very destructive and sometimes fatal. It is important to know that exposure to these drugs in MOG antibody disease has not resulted in a known case of PML. The known incidence of PML is estimated to be 1 in 25,000 with Rituxan and 1 in 6,000 with CellCept, based on the use of these drugs for immunosuppression for other purposes. The manufacturer of Imuran also warns of the risk of PML with Imuran, but the incidence of PML with Imuran has not been documented. Clinical care and early intervention are important when PML is suspected.Chronic immunosuppression requires regular skin examinations by a dermatologist because the immune system is the best defense against the development of cancer cells, and all these treatments can interfere with its normal function.Mycophenolate mofetil and azathioprine are both twice-daily pills that severely suppress the immune system. Both drugs were originally approved by the FDA for the prevention of transplant rejection, although azathioprine is now indicated for the treatment of rheumatoid arthritis, and both have been widely used in several autoimmune diseases. These medications often require blood tests beforehand, usually twice a year thereafter, to monitor liver toxicity and ensure optimal immunosuppression (absolute lymphocyte count around 1 and total white blood cell count between 3 and 4).

Azathioprine is the drug that has been used the longest. Although the AAR appears to be low with azathioprine, one of the complications of this drug is that some cannot stay in remission on azathioprine alone and must also receive steroids (complications of steroids are discussed below). In addition, a long-term study of azathioprine found that the risk of lymphoproliferative cancers was 3%. Common side effects are gastrointestinal disorders, which may include bloating, constipation, nausea, diarrhea, and may change during the course of treatment. Azathioprine is contraindicated during pregnancy, so pregnancy planning is very important. It is FDA Class D (meaning do not take this drug during pregnancy unless it is lifesaving) and is associated with an increased risk of miscarriage, a 7% rate of birth defects, and severe bone marrow suppression that reverses after birth. It is the cheapest of the drugs. In one study of patients with MOG antibody disease, the mean ARR for azathioprine was 0.99, with 41% of seizures occurring within the first 6 months, and most of these early seizures were in those who were also not treated with corticosteroids.

Mycophenolate mofetil has similar effects on the gastrointestinal tract, although many report that symptoms are milder with mycophenolate than with azathioprine. In addition, some patients complain of headaches induced by mycophenolates, especially at the beginning; they usually get worse with continued use. Lymphoma may be a risk of this drug; However, no cases have been reported in patients with MOG antibody disease while taking this drug, so the risk is likely to be small. Mycophenolate is also contraindicated during pregnancy, so planning is again very important. It is also FDA Class D (do not take this drug during pregnancy unless it is a life saver) and has a 45% chance of miscarriage. Of those who do not terminate the pregnancy, 22% have birth defects mainly on the face (mouth, ear).

Rituximab is an intravenous infusion that works differently than the other two agents above. Rather than being a broad immunosuppressant, rituximab completely depletes one type of white blood cell called B cells, which has an effect on the rest of the immune system. Although protocols vary slightly,

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it is usually administered twice a year (4 infusions total) and in an outpatient infusion center.

Low-dose prednisone is also used, more often outside the US as mentioned above. Some doctors also use it with azathioprine for those who continue to have flare-ups on azathioprine alone. Its use is not commonly recommended for maintenance therapy in the United States due to potential complications associated with long-term steroids, including diabetes, osteoporosis, weight gain, mood instability, hypertension, skin changes, etc.

IVIG has also been used as a maintenance treatment. treatment for MOG antibody disease. One retrospective study looked at treatment, AAR symptoms, and disability in 59 patients with MOG antibody disease. This study included 7 patients using IVIG as maintenance therapy. Of these 7 patients, 3 became ill during IVIG therapy and 3 of 7 patients (43%) failed. Half of the exacerbations occurred when IVIG doses were withdrawn or when the dosing interval was extended. Another prospective study examining AARs and disability in 102 children with MOG antibody disease found that maintenance IVIG reduced the median AAR from 2.16 to 0.51. They also found that 4 of 12 patients treated with IVIG (33.3%) relapsed. Some doctors may also prescribe subcutaneous immunoglobulin. After the acute phase, rehabilitation includes both psychological and physical adaptation to improve functional abilities and prevent secondary complications of immobility. Very little has been written in the medical literature specifically about rehabilitation after MOGAD. However, much has been written about spinal cord injury (SCI) recovery in general, and this literature is valid. Physical problems include visual impairment, bladder dysfunction, bowel dysfunction, sexual dysfunction, maintaining skin integrity, spasticity, pain, depression, and fatigue. Rehabilitation and learning how to do daily activities (ie dressing) with mobility problems is an important part of MOGAD treatment and recovery.

The clinical phenotype of MOG antibody disease is diverse, and ON is the most common phenotype of MOG antibody disease, accounting for more than 80%, followed by TM and ADEM. Recently, some clinicians have identified rare cases of MOG Ab-associated demyelinating disease. One case is that the patient can show brainstem encephalitis with pontine trigeminal root entry zone abnormality[2]. The second case is the patient with aseptic meningitis and optic neuritis [3]. And the third case is similar to typical multiple sclerosis [4]. The UK survey found that the clinical manifestations of MOG antibody disease in adults are not the same as in children. In children with MOG antibody disease, ADEM type is more common, accounting for about 40%, and relatively few in adults, about 9% [5]. Adult MOG antibody disease clinical manifestations are closer to the NMOSD. Patients with MOG antibody disease showed a higher count of cerebrospinal fluid white blood cell, content of protein, cerebrospinal fluid pressure, and larger nuclear magnetic lesions [6].

CASE PRESENTATION

Child presented with fever and one episode of seizure on day 1. Her postictal sensorium was normal and she was managed with a provisional diagnosis of febrile seizures. She had persistant fever spikes and leucocytosis and was started on ceftriaxone. Her sensorium worsened from day 4 of fever with focal deficit involving left lower limb. Considering possibility of CNS infection/ ADEM, MRI, and Lumbar puncture were done and antimicrobial coverage was escalated to Meropenam, Acyclovir, and Doxycyline. MRI was suggestive of ADEM. CSF-22 lymphocytes/mm3. Glucose-47(90), Protein-60. Methylprednisolone pulse dose(30mg/kg/day planned for 5 days) was started. As child had a rapidly progressive course, IVIG (2g/kg divided into 3 days) was started after discussing with relatives. Antiraised ICP measures were initiated including 3% NS infusion. Child had transient seizures on day 4 and was loaded with leveteracetam and fosphenytoin. Child had refractory status epilepticus on day 6 and was intubated and started on midazolam infusion. Midazolam was escalated upto 20ug/kg/min along with intermittent phenobarbitone boluses as seizures were refractory. Leveteracetam and phenytoin were tapered and lacosamide was added. Seizures were controlled and midazolam was tapered after 24 hrs of seizure free period and she was extubated after her sensorium improved. She had transient left focal seizures while on oral AEDs and valproate dose was hiked. Child was started on physiotherapy and rehabilitation. Serum MOG antibody qualitative test was reported positive and titres are pending.

Child had low grade fever with increased secretions from day 3 of ventilation. Xray showed right upper lobe consolidation/ collapse. Possibility of aspiration/ventilator-associated pneumonia were considered. There was no worsening of ventilator settings. Minocycline was added and given for 5 days and stopped after ET aspirate and blood culture were reported to be negative.

Duration of ventilation: 6 days
Duration of Cental venous line (Right femoral): 7 days

DISCUSSION

The clinical phenotype of MOG antibody disease is diverse, with ON being the most common phenotype of MOG antibody disease accounting for more than 80%, followed by TM and ADEM. Recently, some doctors have identified rare cases of demyelinating disease associated with MOG Ab. One case is that a patient may have encephalitis with trigeminal afferent zone abnormalities [7]. Another case is a patient with aseptic meningitis and optic neuritis [8]. And the third case resembles a typical multiple sclerosis[9]. A UK study showed that the clinical symptoms of MOG antibody disease in adults are not the same as in children. In children with MOG antibody disease, the ADEM type is more common, approximately 40%, and relatively rare in adults, approximately 9% [10]. The clinical manifestations of MOG antibody disease in adults are closer to NMOSD. Patients with MOG antibody disease were found to have higher CSF white blood cells, protein concentration, CSF pressure, and larger

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nuclear magnetic lesions[11]. In this case, the subtle white matter changes in the encephalopathic child led us to suspect immune-mediated encephalopathy. Although consistent with a diagnosis of ADEM, some features were not typical, particularly the movement disorder seen more often in anti-NMDAR encephalitis[12]. The discovery of pathogenic autoantibodies binding to extracellular proteins in neurons and glial proteins led to a paradigm shift in the diagnosis and treatment of childhood encephalitis[13,14]. The most common autoimmune encephalitis in children is anti-NMDAR encephalitis. Children usually present with a polysymptomatic, recognizable disease syndrome with psychiatric features, agitation, movement disorders, mutism, seizures, and encephalopathy[15]. Interestingly, our patient did not have agitated insomnia, which often occurs in patients with anti-NMDAR encephalitis, and in fact, showed increased insomnia. drowsiness. In contrast to MOG-Ab-related disease, brain MRI is usually normal in anti-NMDAR encephalitis. Although patients with anti-NMDAR encephalitis often respond slowly to steroid therapy and may require second and third-line treatment options, first-line immunotherapy for MOG-Ab-related disease usually produces a rapid response. as observed in our case. Although basal ganglia are often described in patients with MOG-Ab-associated disease, movement disorders or tonic ocular deviation are not well-described features. Ocular tonic abnormality may result from symptomatic damage in the central nervous system (CNS) demyelination, but is more commonly described in pediatric patients with seizures, ischemic events, tumors, or vestibular, brainstem, or cerebellar diseases. Our patient had a long-term videoEEG, which confirmed that the ocular tonic abnormalities and stereotypic movements were not epilepsy; however, independent epileptiform discharges have occasionally been observed. Dyskinesia is a rare but reported side effect of phenytoin therapy. This usually occurs only when serum drug levels are within the toxic range. We believe that this is very unlikely to be the cause of our patient's abnormal movements, as they resolved immediately after intravenous methylprednisolone when he was continued on phenytoin.

CONCLUSION

Our cases suggest that although they present clinically and radiologically as an ADEM-like syndrome, demyelinating disorders associated with MOG-abs can pathologically resemble MS.MOG-abs are increasingly identified in adult patients with inflammatory CNS demyelination that has not yet occurred in a defined spectrum that includes NMOSD, ADEM, and unilateral or bilateral isolated optic neuritis (ON). They are considered highly sensitive and specific when tested with appropriate methods using live cell-based (non-fixed) assays. MOG-abs are more consistently detected in serum than in CSF, and intrathecal synthesis of MOG-abs is uncommon. Although CSF MOG-abs in children with ADEM are uncommon, a prospective systematic study of adults with multifocal demyelinating CNS syndrome and MOG is guaranteed abs will hopefully clarify whether our observation of intrathecal MOG-abs synthesis is coincidental or causal.

Our findings show that

I. MOG-abs-induced inflammatory demyelination, independent of clinical presentation, is histopathologically similar to MS
II. Should encourage clinicians to test MOG-abs in inflammatory CNS diseases suggestive of ADEM-ile, ON, or NMOSD. in serum. and CSF using appropriate test methods.

Although characterized for a long time, ADEM remains a moving target for both diagnostic and therapeutic intervention as more information is gathered about its etiology and pathophysiology. Growing knowledge of ADEM associated with MOG antibodies has meanwhile provided clinicians with additional diagnostic and therapeutic targets, although much more research is needed for targeted and individualized interventions in the future.

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