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Review Article

Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease: A Diverse Demyelinating Entity in Pediatric Patients

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Abstract

Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD) is an autoimmune demyelinating disorder characterized by antibodies targeting the myelin oligodendrocyte glycoprotein, a protein of the myelin sheath in the central nervous system. Increasingly recognized as a distinct clinical entity, MOGAD requires tailored treatment strategies and careful long-term monitoring, rather than being considered a variant of multiple sclerosis and neuromyelitis optica spectrum disorder. While acute episodes respond well to immunotherapy, relapses are common and require long-term management. Progress in immune-based and remyelination therapies offers hope for improving outcomes.

Keywords: Autoimmune disease, Multiple sclerosis, MOGAD, Treatment.

مرض مرتبط بالأجسام المضادة للبروتين السكري قليل التغصن المايلين: كيان متنوع مزيل للمايلين لدى المرضى الأطفال

الخلاصة

المرض المرتبط بالأجسام المضادة للبروتين السكري قليل التغصن (MOG) هو اضطراب مزيل للمايلين في المناعة الذاتية يتميز بالأجسام المضادة التي تستهدف بروتين سكري قليل التغصن المايلين، وهو بروتين من غمد المايلين في الجهاز العصبي المركزي. ويتطلب المرض استراتيجيات علاجية مخصصة ومراقبة دقيقة على المدى الطويل، بدلا من اعتباره نوعا مختلفا من التصلب المتعدد واضطراب طيف التصلب المتعدد والتهاب النخاع البصري. في حين أن النوبات الحادة تستجيب بشكل جيد للعلاج المناعي، فإن الانتكاسات شائعة وتتطلب إدارة طويلة الأمد. يوفر التقدم في العلاجات المناعية والعلاجات المايلينية الأمل في تحسين النتائج.

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INTRODUCTION

Myelin oligodendrocyte glycoprotein (MOG) antibody-associated disease (MOGAD) is an autoimmune demyelinating disorder characterized by antibodies targeting the myelin oligodendrocyte glycoprotein, a protein located on the outer surface of the myelin sheath in the central nervous system [1,2]. This immune-mediated clinical syndrome, characterized by specific clinical, immunological, and radiological findings, differs from other demyelinating diseases, namely multiple sclerosis (MS) and aquaporin-4 antibody-positive neuromyelitis optica spectrum disorder (AQP4+ NMOSD) [3–5]. Increasingly recognized as a distinct clinical entity, MOGAD warrants tailored treatment strategies and meticulous long-term monitoring, rather than being considered a variant of MS or NMOSD [6]. In the pediatric population, the clinical spectrum of MOGAD is being identified more often in clinical practice as serological testing for MOG antibodies has become more widespread, revealing some variations in the clinical spectrum compared to those in adults [7]. The acute disseminated encephalomyelitis (ADEM)-like phenotypes are highly prevalent in pediatric MOGAD [8,9]. In contrast to adolescents and adults, optic neuritis (ON) and transverse myelitis (TM) are more common MOGAD presentations [10]. Once considered a monophasic disorder, MOGAD is now recognized as a relapsing disease, yet children usually have a lower risk of relapses and a better prognosis than adults [11,12].

Epidemiology

The prevalence of MOGAD is predicted to be 2.5 per 100,000 people, with an annual incidence of 3 per one million [13]. MOGAD displays a characteristic

bimodal distribution, primarily affecting children between 6 and 8 years old, who account for roughly 30% to 50% of all diagnosed patients, with no ostensible sex predominance, and adults [14,15]. Between the ages of 30 and 40, with a higher prevalence observed in females, suggesting a potential influence of sex-related factors on disease susceptibility [16]. Recent publications have suggested a reduced relapse risk for white patients when compared to non-white patients [17].

Immunopathogenesis

Myelin oligodendrocyte glycoprotein (MOG) is a transmembrane protein representing about 0.05% of total myelin; its external location renders it highly immunogenic [1,18]. MOG is involved in cell adhesion, stabilization of microtubules, and neurological receptor functions [19]. Immunopathogenesis involves the activation of peripheral T cells and the production of MOG-IgG, leading to subsequent CNS infiltration. Mechanisms include antibody-dependent cellular cytotoxicity (ADCC), complement activation, and microglial phagocytosis [20,21]. Histology typically reveals white matter lesions that exhibit diffuse, coalescent perivenous lesions with intracortical demyelination around small veins, accompanied by relative preservation of astrocytes in MOGAD [22]. In comparison to MS lesions, they are usually discrete and found around larger veins, with subpial demyelination [23]. In addition, iron-rimmed chronic active lesions are absent in MOGAD, while they are usually detected in MS [24]. In contrast, NMOSD shows neuronal loss in cortical layers II–IV, with no cortical demyelination [25]. Another difference is that CD4+ T cells predominate in MOGAD, while CD8+ cells are more prevalent in MS [26]. Genetic predisposition (e.g., *HLA-DRB1*15:01*) and viral triggers can induce the disease [27]. Viral infections, such as Epstein–Barr virus, may contribute to the pathology through molecular mimicry and epitope spreading [28]. Cytokines such as IL-6 and TNF-α are also found to be elevated in the CSF of MOGAD patients [29].

Clinical Phenotypes of Pediatric Presentations

The clinical phenotype and course of MOGAD may differ according to age, and the grade of myelin maturation can explain that [30]. In young children, the median age at onset is between 6 and 7 years [31]. The International MOGAD Panel proposed criteria for clinical presentation, including ADEM, ON, and myelopathy, as well as cerebral, brainstem, and cerebellar deficits, and cortical encephalitis [32]. A leukodystrophy-like phenotype may be seen in younger children [33]. In about half of the cases, MOGAD presents with a clinical picture similar to acute disseminated encephalomyelitis (ADEM), where encephalopathy is reported in nearly all cases, often accompanied by multifocal neurological deficits such as ataxia and cranial nerve palsies [8,9,34]. Brain MRI typically shows large, ill-defined bilateral cortical, subcortical, and deep gray matter lesions, with simultaneous involvement of the optic nerve and spinal cord [35]. Any child with recurrent ADEM or ADEM associated with recurrent optic neuritis should raise suspicion for MOGAD [36]. Optic neuritis is often seen in pediatric patients, although it is less common than in adults and teenagers. It usually affects both eyes, with MRI showing extensive signal enhancement along the optic nerves, favoring the front part of the nerves rather than the optic chiasma and tracts [37, 38]. Notably, optic neuritis has a higher chance of relapses compared to ADEM or myelitis presentation. However, despite severe vision loss, during the acute phase most children tend to recover well, with many achieving visual acuity of 20/25 or better at 6 months [39]. MOGAD-related optic neuritis can be distinguished from MS or NMOSD by its usual bilaterality, with papilledema present in 86% of patients [40]. In MRI, involvement of the optic sheath and surrounding structures occurs in up to 50% of MOGAD-ON cases [41]. MOGAD can also present as transverse myelitis in children. In some cases, it is preceded by infection or vaccination, but in most patients, no such history is available [42]. It typically affects the conus medullaris and is characterized by significant swelling of the spinal cord. While MOG-TM is generally responsive to steroids with a good long-term recovery, about 9% of patients experience poor recovery [43]. Brainstem involvement occurs in about one-third of cases and can happen alongside cerebrum, optic neuritis, and spinal cord distributions, or sometimes in isolation [44]. It is usually linked with very disabling symptoms like cranial nerve palsies, weakness, ataxia, and hypoventilation syndrome. The medulla oblongata is the most frequently affected part of the brainstem [45]. Although Area Postrema Syndrome (APS) is a key feature of NMOSD, it has also been seen in MOGAD [46]. Additionally, a group of patients may be present with fever, headaches, seizures, and cerebrospinal fluid pleocytosis. They typically respond poorly to antiviral therapy while showing rapid improvement on immunotherapy. They have MOG antibody-positive encephalitis, a form that is frequently misdiagnosed as viral meningoencephalitis [47-49].

Disease Course

MOGAD often tends to relapse, particularly in patients who remain MOG-IgG seropositive or undergo premature tapering of corticosteroids [50,51]. Within the first two years, approximately 47% of patients experience a relapse; this rate reaches 72% within five years of onset [52]. Children usually relapse earlier,

often within six months of diagnosis. Generally, children have better outcomes than adults; however, some pediatric patients may still face cognitive or motor impairments [53]. These findings highlight the importance of careful follow-up and early therapy to help lessen the long-term disease impact [54].

Diagnostic Considerations

Antibody Detection

A definitive diagnosis of MOG antibody-associated disease (MOGAD) depends on detecting MOG-IgG with live cell-based assays that include full-length human myelin oligodendrocyte glycoprotein [55]. These tests have shown excellent diagnostic accuracy, with sensitivity above 90% and specificity over 99% [56]. Testing is best done on serum rather than cerebrospinal fluid (CSF), since serum titers provide greater diagnostic value. Notably, titers above 1:160 in adults and above 1:32 in children are generally seen as markers of active disease [57].

Cerebrospinal Fluid Laboratory Findings

Cerebrospinal fluid analysis in MOGAD typically shows a mild to moderate increase in white blood cells, ranging from 50 to 200 cells/mm³, accompanied by elevated protein levels, indicating an inflammatory process [58]. There is nonexistence or only transient presence of oligoclonal bands (OCBs), a feature found in more than 85% of MS cases [59]. A fundamental difference in immunopathology exists between MOGAD and MS [60]. The role of CSF MOG-IgG antibody testing has become clearer, with current evidence favoring testing CSF mainly in seronegative patients with suggestive phenotypes of MOGAD [61].

Neuroimaging

Magnetic resonance imaging (MRI) of the brain and spinal cord is crucial for supporting a diagnosis of MOGAD and differentiating it from other demyelinating disorders [62]. In children, MRI often shows bilateral, poorly defined lesions with a “fluffy” appearance, usually associated with acute disseminated encephalomyelitis (ADEM)-like features [63,64].

Differential Diagnosis

Distinguishing MOG antibody-associated disease (MOGAD) from other demyelinating disorders is crucial for correct diagnosis and proper treatment. Multiple sclerosis (MS) can typically be excluded without hallmark features like Dawson’s fingers on MRI and the consistent presence of oligoclonal bands (OCBs) in the CSF [65]. Conversely, neuromyelitis optica spectrum disorder (NMOSD) is identified by the presence of aquaporin-4 antibodies (AQP4-IgG) and distinctive peri-ependymal lesions in the brainstem [66,67]. It is also important to note that MOGAD may occasionally coexist with other autoimmune markers, such as antibodies against myelin-associated glycoprotein (anti-MAG) or N-methyl-D-aspartate receptors (anti-NMDAR), creating complex clinical presentations [68,69].

Treatment Strategies

Acute Management

Intravenous methylprednisolone is the first-line treatment in the acute stage, usually administered at a dose of 30 mg/kg/day, with a maximum of 1 g per day for 3 to 7 days, depending on response [70]. For patients who do not respond to corticosteroids, alternative treatments such as plasma exchange or intravenous immunoglobulin (IVIG) may be necessary [71]. Prompt diagnosis and timely treatment are essential, as delaying therapy beyond 7 days after onset is linked to significantly worse outcomes and a higher risk of severe complications [72].

Maintenance Therapy

In pediatric patients, clinicians often recommend a gradual and prolonged tapering of steroids over 3 to 6 months to reduce side effects while managing inflammation [73]. Before starting immunosuppressive therapy, it is important to review and update the child’s vaccination status. In patients with relapsing disease or incomplete recovery, or those who develop steroid side effects, maintenance immunotherapy is essential. Commonly used agents include rituximab, mycophenolate mofetil, and azathioprine [74,75].

Emerging Therapies

Several innovative therapies are currently under investigation and may expand future treatment options. These include rozanolixizumab, satralizumab, tocilizumab, evobrutinib (a BTK inhibitor), and remyelination agents such as PIPE-307 [76-78].

Prognosis and Outcomes

The risk of relapses is moderate and can be reduced with extended immunosuppression [51,79]. Prognosis is influenced by type of onset, age, gender, ethnicity, and recovery from initial attack [80]. Prompt treatment after optic neuritis leads to better visual outcomes, with over 90% recovery if treated early versus 50% if treatment is delayed [81]. After ADEM, up to 30% of children may experience cognitive issues, especially with attention and executive function [82]. Although MRI abnormalities resolve in two-thirds of patients,

persistent lesions may signal risk for long-term disability [83]. Predictors of recurrence include high MOG-IgG titers, shorter relapse intervals, and optic nerve involvement [84]. Pediatric autoimmune neurological disorders, such as MOGAD and type 1 diabetes mellitus (T1DM), are increasingly recognized for their impact beyond organ-specific damage, extending to cognitive and neuropsychological functions. Recognizing these broader neurocognitive impacts is essential for early intervention, multidisciplinary care, and long-term support for affected children. Endocrine dysfunctions are frequently observed in pediatric neurodevelopmental and neuroimmunological disorders, highlighting the close interaction between the immune and endocrine systems during early growth and brain maturation [85-89]. Despite advances, challenges remain, including uncertainty about optimal immunotherapy duration, limited pediatric data on biologics, and the need for multicenter registries to improve treatment guidelines across populations [90,91]. International collaboration is the key to refining diagnostics and advancing individualized care [92,93].

Conclusion

MOGAD is now recognized as a distinct autoimmune demyelinating disorder with clinical, radiological, and serological features that differentiate it from MS and AQP4+ NMOSD. Pediatric MOGAD presents unique clinical and prognostic characteristics. While acute episodes respond well to immunotherapy, relapses are common and require long-term management. Progress in immune-based and remyelination therapies offers hope for improving outcomes.

Conflict of interests

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Data sharing statement

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