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Case Report

Pediatric myelin oligodendrocyte glycoprotein antibody associated disease following incomplete Kawasaki – a case report with varied association

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ABSTRACT

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) has emerged as a distinct inflammatory demyelinating disorder of the central nervous system. Although relatively rare, growing recognition of this entity has highlighted important differences from multiple sclerosis and neuromyelitis optica spectrum disorder, particularly with respect to clinical phenotype, neuroimaging features, and long-term prognosis. We report a 5-year-old girl who presented with acute, painful, and progressive diminution of vision in the right eye, accompanied by impaired color perception for 3 days. Cerebrospinal fluid analysis was unremarkable, with antibody testing negative for neuromyelitis optica spectrum disorder (NMOSD) and positive for myelin oligodendrocyte glycoprotein (MOG) antibodies. She had a recent history of incomplete Kawasaki disease treated with intravenous immunoglobulin one month prior. While systemic corticosteroids led to partial visual improvement, complete recovery was achieved following intravenous immunoglobulin therapy. This case highlights the importance of assessing MOG-IgG antibodies in pediatric patients presenting with optic neuritis. Precise differentiation from other demyelinating disorders is essential for accurate diagnosis and appropriate management. The overall prognosis is generally favorable.

Keywords: MOGAD, Kawasaki disease, Optic neuritis, Demyelination

INTRODUCTION

Since the identification of myelin oligodendrocyte glycoprotein (MOG) as a target antigen in acute disseminated encephalomyelitis (ADEM), MOG antibody-associated disease (MOGAD) has been increasingly recognized as an important cause of inflammatory demyelinating disorders in both pediatric and adult populations.[1,2]

MOGMOGAD represents a demyelinating disorder with a clinical course and therapeutic approach distinct from multiple sclerosis and neuromyelitis optica spectrum disorder (NMOSD). Early recognition based on characteristic clinical and imaging features permits targeted antibody testing and timely initiation of appropriate treatment.

The clinical spectrum of MOGAD is varied, including recurrent ON, transverse myelitis, and acute disseminated myelitis (ADEM).[3] The hallmark of the disease is the presence of antibodies against MOG, the detection of which has been facilitated by the development of specific cell-based assays that identify a distinct subset of affected patients. Here we report a case of a 5-year-old female child diagnosed with MOGAD, with a preceding history of incomplete Kawasaki disease, suggesting a possible underlying immune-mediated association.

CASE REPORT

A 5-year-old female child, first born to third-degree consanguineous parents, presented with sudden onset pain in the right eye followed by progressive diminution of vision over four days, associated with impaired color

perception. There was no history of seizures, sensory loss, focal limb weakness, altered sensorium, behavioral changes, cognitive decline, bowel or bladder dysfunction, or symptoms suggestive of meningeal irritation.

The child had a significant past history one month prior, when she was hospitalized for unexplained fever and bilateral non-purulent conjunctivitis of six days' duration. Laboratory evaluation revealed elevated inflammatory markers, including erythrocyte sedimentation rate (62 mm/hr), C-reactive protein (321 mg/dl), and D-dimer (1466 ng/dl), with neutrophilic leukocytosis on complete blood count. Two-dimensional echocardiography demonstrated coronary artery dilatation with Z-scores of the left main coronary artery (2.07), left anterior descending artery (3.34), right coronary artery (3.26), and left circumflex artery (-0.19), consistent with American heart association risk level III for coronary artery aneurysms. A diagnosis of incomplete Kawasaki disease was made, and she was treated with intravenous immunoglobulin, aspirin, and systemic corticosteroids, followed by regular follow-up. Repeat echocardiography after two weeks showed normalization of coronary artery dimensions.

One month later, she presented with the aforementioned visual complaints. She was developmentally appropriate for age with normal anthropometric parameters. On examination, vital signs were stable. Neurological evaluation revealed normal higher mental functions. Cranial nerve examination showed a right relative afferent pupillary defect, with normal pupillary response on the left. Visual acuity assessment revealed perception of light in the right eye and 6/6 vision in the left eye. Other cranial nerves were intact. Motor, sensory, cerebellar, and autonomic examinations were unremarkable, with no signs of meningeal irritation. Fundoscopic examination demonstrated right optic disc edema (Figure 1).

Laboratory investigations showed hemoglobin 13 g/dl, total leukocyte count 10,400/mm³, platelet count 232,000/mm³, ESR 16 mm/hour, and CRP 3.9 mg/dl. Cerebrospinal fluid analysis revealed normal glucose (65 mg/dl) and protein (13 mg/dl) levels, with negative oligoclonal bands and sterile cultures. Magnetic resonance imaging of the brain and orbits demonstrated acute right optic neuritis without associated intracranial abnormalities (Figure 2). Optical coherence tomography revealed peripapillary fluid accumulation and retinal nerve fiber layer edema in the right eye.

Given the suspicion of a demyelinating disorder, an autoimmune antibody panel was performed, which was negative for antinuclear antibodies and anti-aquaporin-4 antibodies, but positive for anti-myelin oligodendrocyte glycoprotein antibodies. A diagnosis of MOG antibody-associated optic neuritis was therefore established, and treatment was initiated.

The patient received intravenous methylprednisolone for five days, followed by a tapering course of oral corticosteroids, resulting in partial visual improvement to finger counting at one foot with grade 1 relative afferent pupillary defect. In view of the incomplete response, intravenous immunoglobulin was administered at a dose of 2 g/kg over 24 hours, leading to further improvement in visual acuity. She is currently under follow-up and remains relapse-free.

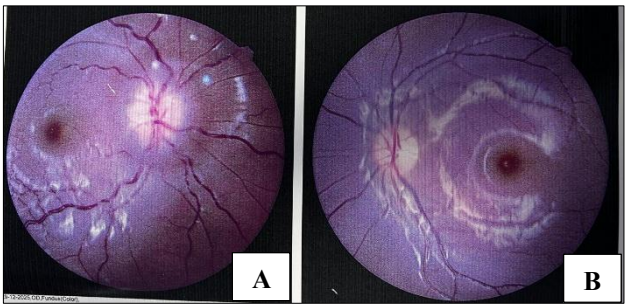

Figure 1: Fundoscopic examination of the right eye demonstrating optic disc edema.

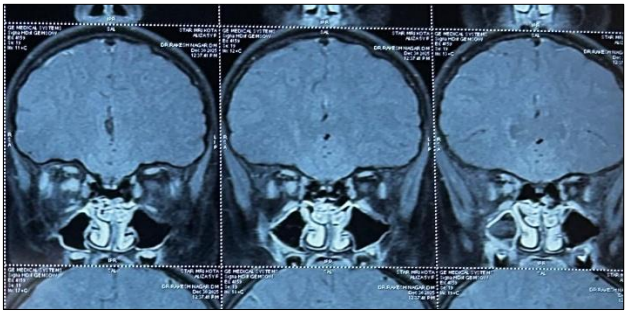

Figure 2: MRI brain and orbit (axial T2/FLAIR sequence) demonstrating signal hyperintensity of the right optic nerve consistent with acute optic neuritis.

DISCUSSION

MOGAD is a recently identified central nervous system demyelinating disorder with the presence of conformation-sensitive antibodies against MOG detected by a cell-based assay.[2] The prevalence is approximately 1.3-2.5/100,000, and the annual incidence is approximately 3.4-4.8 per million.[4] In addition to its unique clinical features, MOGAD demonstrates characteristic immunopathology, including perivenous demyelination, MOG-predominant myelin loss, absence of astrocytic injury, and Th17-related cytokine upregulation.[5]

Recently published international criteria define MOGAD based on the presence of a core demyelinating clinical event (optic neuritis as in our case, myelitis, ADEM, cerebral monofocal or polyfocal deficits, brainstem or cerebellar deficits, and cerebral cortical encephalitis often with seizures), positive serum MOG-IgG, and exclusion of alternative diagnoses, particularly multiple sclerosis.[6] In patients with low-positive MOG-IgG titers, at least one

supportive clinical or MRI feature is required, as low-level positivity may occur in other demyelinating disorders. Diagnostic challenges may persist in certain clinical presentations, such as cerebral mono- or polyfocal deficits, where clinical and imaging features are less well defined, especially in the setting of low-positive antibody titers.

An important clinical aspect of MOGAD is that approximately 20-40% of the cases had infectious prodromes or precedent infections which included the common cold, pharyngolaryngitis, bronchitis, pneumonia, acute gastroenteritis, and infections related to influenza, mycoplasma, streptococcus, and chlamydia. The Japanese study resulted that the precedent infection was more prevalent in the pediatric group (<10 years) than in the adult group.[7] Association of autoimmune diseases are observed in up to 7-10% of MOGAD patients, with Sjogren syndrome, rheumatoid arthritis, ulcerative colitis, thyroid disorder, psoriasis, and NMDAR encephalitis being commonly reported, but the frequency appears to be lower than that in AQP4+NMOSD.[8]

To date, there are no published reports clearly describing an association between Kawasaki disease and MOG antibody-associated disease in the pediatric population. However, the occurrence of MOGAD following Kawasaki disease in our patient raises the possibility of a shared immune-mediated mechanism. Kawasaki disease is characterized by systemic immune activation and cytokine dysregulation, which may predispose susceptible individuals to secondary autoimmune phenomena.

Given that MOGAD is increasingly recognized as a post-inflammatory, antibody-mediated demyelinating disorder, it is plausible that antecedent immune activation in Kawasaki disease may act as a trigger rather than a direct cause. This observation expands the spectrum of potential inflammatory antecedents associated with pediatric MOGAD and warrants further investigation.

This case describes pediatric MOG antibody-associated optic neuritis occurring after incomplete Kawasaki disease, a temporal association not previously reported. While causality cannot be established, antecedent immune activation may represent a potential trigger for MOGAD. Recognition of this possible link underscores the importance of MOG-IgG testing in children presenting with optic neuritis and supports further investigation into post-inflammatory mechanisms in paediatric demyelinating disorders.

CONCLUSION

General paediatricians, as primary care providers for children, are often the first to encounter such presentations and should maintain a high index of suspicion for MOG antibody- associated disease in children presenting with acute visual loss, particularly following a recent inflammatory illness.

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