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ANTI-MOG IGG POSITIVE OPTIC NEURITIS IN A 10-YEAR-OLD CHILD: A CASE REPORT

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ABSTRACT

Optic neuritis in children is a significant cause of acute visual impairment. Myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD) is increasingly recognized as a distinct demyelinating disorder, differing from multiple sclerosis and anti-aquaporin-4 (AQP4) positive neuromyelitis optica spectrum disorder. We report a 10-year-old girl who presented with painful left eye movements and progressive diminution of vision for 4–5 days. Examination revealed visual acuity reduced to perception of hand movements in the left eye and disc edema on fundoscopy (LE > RE). MRI brain and orbit demonstrated T2 hyperintensity in the intraorbital and intracanalicular segments of the left optic nerve. Anti-AQP4 antibody was negative while Anti-MOG IgG antibody was positive. Visual evoked potential (VEP) showed absent waveforms in the left eye, confirming demyelinating optic neuropathy. The child was treated with intravenous methylprednisolone pulse therapy (30 mg/kg/day for 5 days) followed by oral prednisolone. Due to minimal visual recovery, referral for intravenous immunoglobulin (IVIG) therapy was planned. This case highlights the importance of Anti-MOG IgG antibody testing in pediatric optic neuritis and the need for prompt immunotherapy to improve visual outcomes.

INTRODUCTION

Optic neuritis is an inflammatory condition of the optic nerve and represents a significant cause of acute visual impairment in the paediatric population. The aetiology is diverse and includes multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD) associated with anti-aquaporin-4 (AQP4) antibodies, and myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD). MOGAD is an increasingly recognized clinically distinct demyelinating syndrome with a unique immunopathological profile. It is caused by antibodies targeting myelin oligodendrocyte glycoprotein (MOG), a protein expressed on the outer surface of myelin sheaths and oligodendrocyte cell bodies.

In children, MOGAD is a relatively common cause of optic neuritis and may present as an isolated episode or as part of a broader spectrum including acute disseminated encephalomyelitis (ADEM), transverse myelitis, or overlap syndromes. Anti-MOG IgG antibody positivity differentiates this entity from MS-associated and AQP4-positive optic neuritis — a distinction of clinical importance since treatment strategies and prognoses differ. Early recognition and prompt immunotherapy are critical for preventing long-term visual morbidity and reducing the risk of relapse.

CASE REPORT

A 10-year-old female child, third order by birth, born to non-consanguineous parents, was brought with complaints of diminished vision in the left eye for 4–5 days. As narrated by the mother, the child was apparently well until 4–5 days prior to presentation when she developed sudden onset pain in the left eye, which increased on eye movement and was not aggravated by bright light exposure. One day after the onset of eye pain, she developed progressive blurring of vision in the left eye and was unable to recognise or read clearly. There was no history of fever, trauma, headache, loss of consciousness, vomiting, myalgia, limb weakness, skin rashes, tingling, facial numbness, or bowel/bladder incontinence. No history of tuberculosis exposure, weight loss, or night sweats was noted.

Past history was unremarkable with no previous hospital admissions or similar episodes. Family history was not significant. Developmental history revealed age-appropriate milestones; the child was studying in the 5th standard and mingles well with peers.

On general examination, the child was haemodynamically stable with heart rate 90 bpm, respiratory rate 20 cpm, blood pressure 106/65 mmHg, SpO2 97% on room air, and temperature 98.4°F. Anthropometry revealed weight 29.3 kg (25th–50th centile), height 144 cm (75th centile), and BMI 14.15 kg/m^2. Systemic examination of the respiratory and cardiovascular systems was unremarkable. Per abdomen was soft with liver palpable 1 cm below the right costal margin and no splenomegaly.

Neurological examination revealed intact higher mental functions with orientation to time, place, and person. Cranial nerve examination was significant for visual acuity in the left eye reduced to perception of hand movements only (CN II), with normal range of movements and vision in the right eye. Fundoscopy revealed disc edema, more prominent in the left eye than the right (LE > RE). The remaining cranial nerves were intact. Motor system examination showed normal bulk, tone, and power (5/5 in all groups bilaterally). Deep tendon reflexes were 2+ bilaterally with bilateral flexor plantar responses. No signs of meningeal irritation or cerebellar dysfunction were observed.

Laboratory investigations including complete blood count (Hb 14.1 g/dL, WBC 10,190/mm^3, platelets 4,09,000/mm^3), liver function tests, renal function tests, and serum electrolytes were within normal limits. C-reactive protein was negative and blood culture showed no growth after 48 hours. Cerebrospinal fluid (CSF) analysis was largely unremarkable with protein 23.9 mg/dL, chloride 120.5 mEq/L, LDH 21 U/L, and a cell count of 1 lymphocyte/mm^3.

MRI brain and orbit with contrast revealed T2 hyperintense signal in the intracanalicular and intraorbital segments of the left optic nerve with hyperintensity in the left retro-orbital fat, with no abnormal optic nerve enhancement. MRI spine screening and MR venogram were normal. MR paranasal sinuses showed hyperintense polypoidal mucosal thickening in bilateral maxillary sinuses. Anti-aquaporin-4 (AQP4) IgG antibody was negative while Anti-MOG IgG antibody was positive, confirming the diagnosis of MOGAD-associated optic neuritis.

Following paediatric neurology consultation, the child was initiated on intravenous methylprednisolone pulse therapy at 30 mg/kg/day (880 mg in 100 mL normal saline over 1 hour)

for a total of 5 days. Lumbar puncture was performed under sedation. The child was subsequently converted to oral prednisolone (Wysolone) at 1.5 mg/kg/day with gastroprotection (Junior Lanzole 30 mg once daily) and calcium supplementation (Shelcal 500 mg thrice daily). Visual evoked potential (VEP) performed during the inpatient stay showed absent NPN waveforms and non-superimposable waveforms in the left eye, consistent with demyelinating optic neuropathy.

Despite completing methylprednisolone pulse therapy, only minimal improvement was noted in left eye vision. The child was discharged on oral prednisolone (Wysolone 45 mg once daily) for 3 months with calcium supplementation and was referred to a higher centre (Indira Gandhi Hospital) for intravenous immunoglobulin (IVIG) therapy and further neuro-ophthalmological evaluation.

DISCUSSION

MOGAD is a spectrum of inflammatory demyelinating disorders of the central nervous system caused by antibodies targeting myelin oligodendrocyte glycoprotein (MOG). Optic neuritis is the most common manifestation of MOGAD in children and may present as an isolated episode or in conjunction with other CNS demyelinating events. It is now well established that MOGAD is a distinct entity from MS and AQP4-positive NMOSD, with a different clinical course, pathogenesis, treatment response, and prognosis.

MOG-associated optic neuritis is characterised by painful visual loss, often with pronounced disc edema on fundoscopy, and involvement of the anterior optic nerve. While bilateral involvement is more typical, unilateral presentations such as in our case are not uncommon. The combination of painful eye movements, disc edema, and severe visual impairment — with MRI showing T2 hyperintensity in the intraorbital and intracanalicular segments of the optic nerve — is highly characteristic of MOGAD. The absence of optic nerve enhancement and normal MRI spine further supported isolated optic nerve involvement, distinguishing this from NMOSD or MS.

CSF analysis in MOGAD-associated optic neuritis is typically unremarkable or shows mild pleocytosis, as observed in our patient with a cell count of just 1 lymphocyte/mm^3 and normal protein — consistent with published literature. VEP findings of absent waveforms confirmed significant conduction delay indicative of demyelinating optic neuropathy. The negative anti-AQP4 antibody helped exclude NMOSD, and the positive anti-MOG IgG antibody established the diagnosis.

High-dose intravenous methylprednisolone is the mainstay of treatment for acute MOGAD-associated optic neuritis and is associated with faster visual recovery. A slow oral steroid taper following pulse therapy is recommended to reduce the risk of relapse. In cases with incomplete or inadequate response to corticosteroids, intravenous immunoglobulin (IVIG) is a well-established and effective second-line therapy in children with MOGAD. The need for IVIG in our case, despite completing a full course of methylprednisolone pulse therapy, underscores both the severity of the initial presentation and the importance of early escalation of therapy in non-responders. Long-term follow-up with serial ophthalmological assessments and repeat anti-MOG IgG titers is essential for monitoring disease activity and guiding decisions regarding maintenance immunotherapy.

CONCLUSION

Anti-MOG IgG antibody–associated optic neuritis is an important and potentially treatable cause of acute visual loss in children. Clinicians should maintain a high index of suspicion for MOGAD in paediatric patients presenting with acute painful progressive visual loss, particularly when disc edema is present on fundoscopy. Prompt serological testing for anti-MOG IgG antibodies, appropriate neuroimaging, and timely initiation of high-dose corticosteroid therapy are essential for optimising visual outcomes. Cases refractory to steroids should be promptly referred for IVIG therapy. Early diagnosis and aggressive immunotherapy remain the cornerstones of management in MOGAD-associated optic neuritis.

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