World Journal of Pharmaceutical and Life Sciences
WJPLS
www.wjpls.org
SJIF Impact Factor: 7.409

MOGAD-ASSOCIATED OPTIC NEURITIS IN TWO PEDIATRIC PATIENTS

Manira Moussa Ahmed*, Omar Moussa Sougueh, Nadia Echcharii, Nabila Chekhlabi and Nezha Dini

Mohammed VI University of Health and Sciences, Casablanca, Morocco.

***Corresponding Author: Manira Moussa Ahmed**
Mohammed VI University of Health and Sciences, Casablanca, Morocco.

Article Received on 29//04/2025 **Article Revised on 19/05/2025** **Article Accepted on 08/06/2025**

INTRODUCTION
Optic neuritis (ON) in children represents a rare but serious inflammatory condition of the optic nerve, often presenting with acute visual loss, ocular pain, and variable neurological signs. It's a diagnostic emergency. Among the evolving spectrum of acquired demyelinating syndromes in pediatric populations, myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) has emerged as a distinct nosological entity, with important diagnostic, prognostic, and therapeutic implications. Characterized by the presence of serum immunoglobulin G (IgG) antibodies directed against the MOG protein. MOGAD is increasingly recognized as a major cause of recurrent or bilateral optic neuritis in the pediatric age group.

Unlike multiple sclerosis (MS) and aquaporin-4 antibody-positive neuromyelitis optica spectrum disorder (AQP4-NMOSD), MOGAD exhibits unique clinical, radiological, and immunological features. In children, optic neuritis associated with MOG antibodies is often bilateral, severe in presentation, but typically demonstrates a good response to corticosteroid therapy, with potential for significant visual recovery. Nevertheless, relapses are common, necessitating close monitoring and sometimes long-term immunomodulatory treatment.

Given the diagnostic challenges and therapeutic nuances of MOGAD in pediatric patients, particularly in distinguishing it from other demyelinating disorders, this study aims to describe the clinical presentation, radiologic findings, immunologic profiles, therapeutic strategies, and outcomes of children diagnosed with MOG-IgG–associated optic neuritis.

We report two pediatric cases of acute bilateral optic neuritis with similar clinical presentations but differing backgrounds, suggestive of immune-mediated etiologies such as MOG antibody-associated disease (MOGAD).

MATERIELS AND METHODS

Retrospective and descriptive study of MOGAD associated optic neuritis in two pediatric patients.

RESULTS

Observation 1

An 11-year-old girl, presented with a 10-day history of a sudden bilateral visual acuity loss associated with painful white eyes and frontal headaches. The symptoms had been evolving without fever or deterioration of general condition. She was referred to our facility after an ophthalmological evaluation and imaging. The patient had no history of autoimmune, ophthalmologic, or neurological disease. She was born from a non-consanguineous marriage, and demonstrated normal psychomotor development. There were no known viral contacts.

On admission, she was conscious, afebrile, and hemodynamically and respiratory stable she was overweight with a BMI at 29.5 .Neurological assessment revealed a normal gait without ataxia or meningeal signs. There were no disturbances in balance or coordination. Sensorimotor function was intact, muscle strength was preserved and symmetrical, and deep tendon reflexes were present and symmetrical.

Observation 2

One month prior admission, the patient presented with the onset of moderate headaches. Two weeks later, he patient experienced a sudden bilateral loss of visual acuity. The symptoms had been evolving without fever or deterioration of general condition.He had no medical history of autoimmune, ophthalmologic, or neurological disease. he was born from a non-consanguineous marriage, and demonstrated normal psychomotor development. There were no known viral contacts.

On admission, he was conscious, afebrile, and hemodynamically and respiratory stable. Neurological assessment revealed a normal gait without ataxia or

meningeal signs. There were no disturbances in balance or coordination. Sensorimotor function was intact, muscle strength was preserved and symmetrical, and deep tendon reflexes were present and symmetrical.

On both patients

Cranial Nerve Examination: There was no evidence of peripheral facial nerve palsy. The patient reported pain during extraocular movements. Ophthalmologic evaluation showed no signs of cataract, subconjunctival hemorrhage, dacryocystitis, or conjunctivitis. No nystagmus or divergent strabismus was noted. Pupils were equally dilated and reactive to light, with no abnormalities in size or symmetry.

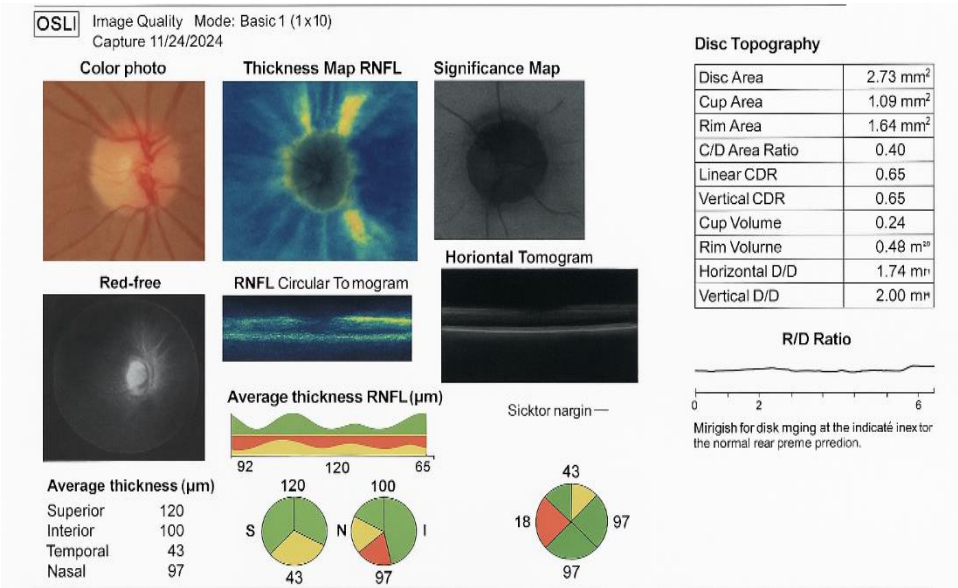
Biological and Imaging Findings

There was no evidence of an inflammatory syndrome. Anti-MOG antibodies were positive in the blood samples, while no other autoantibodies were detected.

The lumbar puncture did not reveal any oligoclonal bands and showed no signs of meningitis.

Ophthalmologic Evaluation

Ophthalmoscopic examination, complemented by optical coherence tomography (OCT), revealed grade 2 papilledema in the right eye and grade 1 in the left eye in the first patient. Both patients demonstrated signs consistent with bilateral optic neuritis.



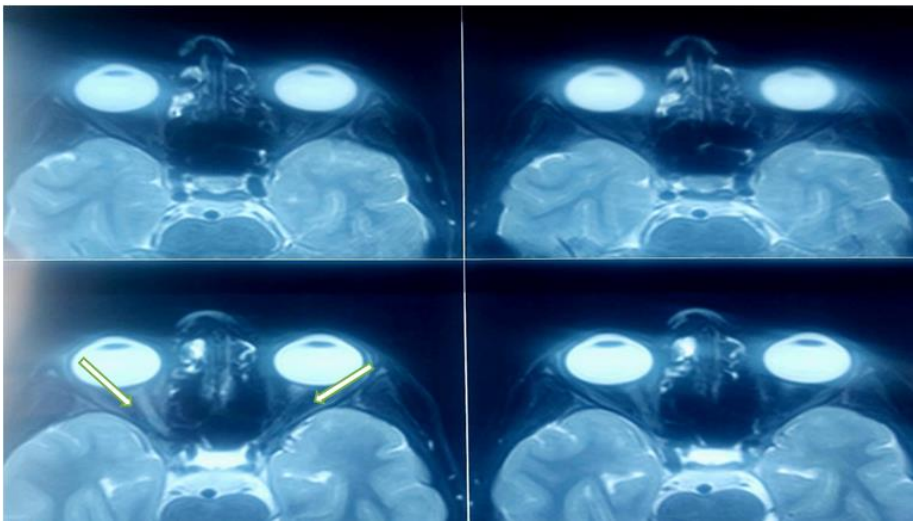
MRI: Findings suggestive of bilateral optic neuritis on both patients (more pronounced on the right side for the girl) No other detectable cerebral abnormalities.

Retinal Angiography

Retinal angiography confirmed bilateral optic neuritis in both cases.

In Case 1, there was a marked decrease in visual acuity in the right eye, with late-phase fluorescein retention at the level of the optic discs, more pronounced on the right side.

In the male patient, ocular involvement was bilateral and symmetrical.



Therapeutic decision and clinical evolution

Patients received a three-day course of intravenous Methyl prednisolone bolus therapy at a dose of 500 mg/m² CS/day. This was followed by a transition to oral corticosteroids with a gradual tapering regimen. Adjunctive supplementation with vitamin D and calcium was also administered.

The clinical course was favorable, marked by resolution of headaches and progressive improvement in visual acuity. No relapse was noted in both patients.

DISCUSSION

MOGAD is an emerging entity in pediatric neuroimmunology, with an estimated incidence of 0.16 per 100,000 children per year.[1] It accounts for up to 30% of acquired demyelinating syndromes in children.[2] The pathogenic target, MOG, is a surface protein of oligodendrocytes believed to function as a cell adhesion molecule, making it particularly vulnerable to autoimmune attack.[4]

Unlike MS or AQP4-NMOSD, MOGAD exhibits a more monophasic initial course, although relapses may occur, especially within the first two years.[5,6] The clinical and radiological presentation in children often includes bilateral optic neuritis, with severe optic disc swelling and longitudinal optic nerve enhancement on MRI. Children with MOGAD typically demonstrate good steroid responsiveness and excellent visual recovery, particularly when diagnosed and treated promptly.[5,6]

Diagnosis requires the detection of anti-MOG IgG using a live cell-based assay, along with clinical features and exclusion of other causes, including MS and AQP4-NMOSD.[3] CSF findings are often nonspecific, and oligoclonal bands—commonly seen in MS—are typically absent in MOGAD. MRI findings can help differentiate MOGAD from other demyelinating diseases, as brain lesions are less frequent and spinal cord involvement is less extensive in children with isolated ON.

Our two cases are consistent with the known profile of pediatric MOGAD: young age of onset, bilateral involvement, absence of systemic inflammation, and favorable response to corticosteroids. In line with findings from larger pediatric cohorts, the risk of relapse appears to be lower in children compared to adults, particularly in cases of isolated optic neuritis with transient antibody titers.[6,7]

Nevertheless, close follow-up is essential. Persistent MOG antibody positivity or early relapses may warrant the initiation of immunosuppressive maintenance therapy to prevent cumulative neurological damage.[8]

CONCLUSIONS

These cases highlight the importance of early recognition of MOG-IgG–associated optic neuritis in children. The combination of clinical presentation, MRI, and serologic testing facilitates timely diagnosis and effective corticosteroid therapy. Although outcomes are generally favorable in pediatric patients, particularly with isolated optic neuritis, long-term monitoring remains crucial due to the potential for relapses.

REFERENCES

1. De Mol CL, Wong Y, van Pelt ED, et al. Incidence and outcomes of acquired demyelinating syndromes in Dutch children: update of a nationwide and prospective study. *Mult Scler*, 2020; 26(7): 806–814.
2. Hennes EM, Baumann M, Schanda K, et al. Prognostic relevance of MOG antibodies in children with an acquired demyelinating syndrome. *Neuropediatrics*, 2017; 49(1): 3–11.
3. Banwell B, Bar-Or A, Arnold DL, et al. Diagnosing multiple sclerosis in children and adolescents. *Lancet Neurol*, 2023; 22(3): 268–282.
4. Johns TG, Bernard CC. The structure and function of myelin oligodendrocyte glycoprotein. *J Neurochem*, 1999; 72(1): 1–9.
5. Stiebel-Kalish H, Hellmann MA, Mimouni M, et al. Anti-MOG antibodies and visual outcomes in optic neuritis: a cohort study. *Neurol Neuroimmunol Neuroinflamm*, 2019; 6(3): e572.
6. Wendel EM, Oehmichen M, Ringelstein M, et al. Isolated optic neuritis in children with MOG antibodies. *Eur J Paediatr Neurol*, 2020; 27: 86–93.
7. Cobo-Calvo A, Ruiz A, Maillart E, et al. Clinical features and prognostic factors in MOGAD adults. *Ann Neurol*, 2021; 89(1): 30–41.
8. Bruijstens AL, Wokke JH, van Pelt ED, et al. Pediatric MOGAD: Treatment approaches and outcomes. *Eur J Paediatr Neurol*, 2020; 29: 41–53.