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Devic's Neuromyelitis Optica in Children: A Report of 2 Cases

S. Hida[1]*, N. Ousman[1], FZ. Mebrouki[1], A. Maadane[1], R. Sekhsoukh[1]

[1]Mohammed VI University Hospital, Oujda; Mohammed Premier University, Oujda; Faculty of Medicine and Pharmacy, Oujda

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***Corresponding author:** S. Hida
Mohammed VI University Hospital, Oujda; Mohammed Premier University, Oujda; Faculty of Medicine and Pharmacy, Oujda

Abstract

Neuromyelitis optica (NMO) is a rare autoimmune inflammatory demyelinating disorder of the central nervous system, predominantly affecting the optic nerves and spinal cord. Pediatric cases are uncommon and often underdiagnosed due to heterogeneous clinical and radiological presentations. We report two pediatric cases of NMO with distinct clinical and immunological features. ***Case 1*:** A 10-year-old girl presented with a 5-day history of progressive bilateral visual loss and impaired color vision. Ophthalmological examination revealed severe bilateral visual acuity reduction and grade II papilledema. Brain and spinal MRI showed T2 hyperintensity of the optic nerves and chiasm with cervical spinal cord involvement (C2–C7). Cerebrospinal fluid analysis showed mild hyperproteinorrachia. Anti-MOG antibodies were positive. The patient was treated with intravenous methylprednisolone followed by oral corticosteroids and intravenous immunoglobulins, with significant clinical improvement within 8 days. ***Case 2*:** An 8-year-old girl presented with a preceding flu-like illness followed by acute paraplegia, sphincter dysfunction, and bilateral visual impairment. Neurological examination revealed severe motor and sensory deficits consistent with longitudinal extensive transverse myelitis. MRI demonstrated diffuse spinal cord involvement with gadolinium enhancement and normal brain imaging. Laboratory tests showed elevated inflammatory markers and positive antinuclear antibodies. She received high-dose intravenous corticosteroids followed by oral prednisone and azathioprine, with favorable neurological recovery. Pediatric NMO is a rare but severe neuroinflammatory disease with variable clinical and immunological profiles. Early recognition through MRI and antibody testing is essential for prompt treatment. High-dose corticosteroids combined with immunotherapy can lead to favorable visual and neurological outcomes.

Keywords: Neuromyelitis optica, pediatric, optic neuritis, transverse myelitis, anti-MOG antibodies.

INTRODUCTION

Neuromyelitis optica (NMOD) is a rare inflammatory and demyelinating disease characterized by significant phenotypic and serological variability, and by the association of severe spinal cord involvement of subacute onset, and acute optic neuropathy, which can be unilateral or bilateral.

This disease is frequently reported in adults and rarely in children (constituting 3 to 5% of all NMO cases), where it is often underdiagnosed.

We report 2 cases of neuromyelitis optica (NMO).

CLINICAL CASE 1

A 10-year-old female patient with no particular medical history was brought to the ophthalmological emergency department for a bilateral decrease in visual acuity and an alteration in color vision that had been progressing over the past 5 days.

The ophthalmological examination revealed a bilateral visual acuity limited to counting fingers. The examination of the anterior segment was unremarkable. The fundus examination revealed bilateral grade II papilledema according to the Frisén scale. The neurological examination revealed only mild gait instability.

An emergency brain and spinal cord MRI revealed a T2 hyperintensity in the optic nerves and the chiasm, as well as a spinal cord hyperintensity extending from C2 to C7 without contrast enhancement. The lumbar puncture demonstrated the presence of mild hyperalbuminorrhachia. The biological assessment revealed the positivity of anti-MOG antibodies.

The retained diagnosis was therefore NMO associated with anti-MOG antibodies. Intravenous methylprednisolone boluses were initiated emergently for 5 days, followed by oral prednisone (20 mg/day) and polyvalent immunoglobulins administered over 2 days.

The evolution was favorable after 8 days of treatment, marked by a reduction in the papilledema and an improvement in visual acuity.

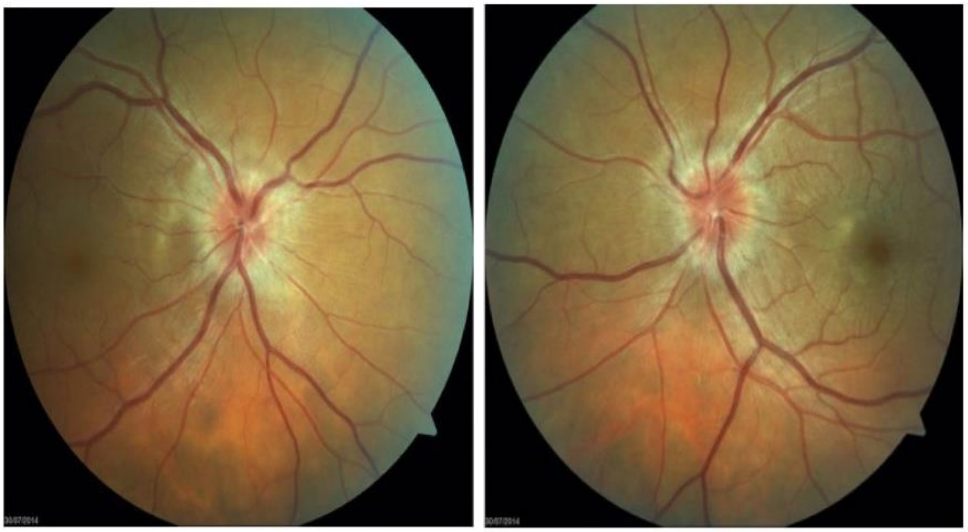

Figure 1: Fundus examination showing grade II papilledema according to the Frisén scale

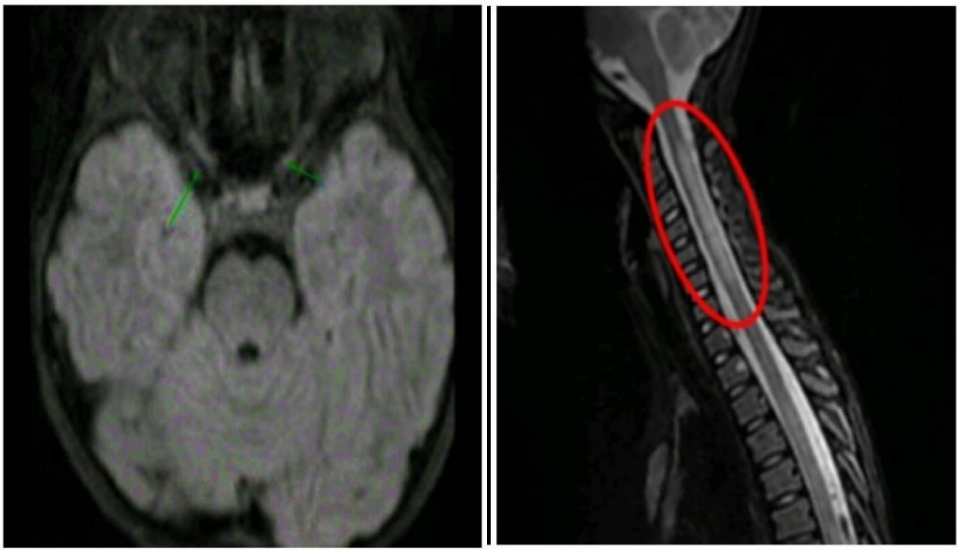

Figure 2: Brain and spinal cord MRI revealing a T2 hyperintensity in the optic nerves and chiasm, as well as a spinal cord hyperintensity from C2 to C7 without contrast enhancement

CLINICAL CASE 2

An 8-year-old female patient with no previous medical history presented one week prior to her admission to the pediatric emergency department with a flu-like syndrome characterized by fever, myalgia, cough, rhinorrhea, and vomiting. One day before her admission, she developed paraplegia and sphincter disorders, specifically urinary retention, followed by a bilateral decrease in visual acuity.

The ophthalmological examination showed a visual acuity reduced to counting fingers at 2meters, with mild bilateral optic disc pallor observed on the fundus examination.

The neurological examination demonstrated a motor deficit in the lower limbs with significant amyotrophy, alongside a superficial sensory deficit with a sensory level between T6 and T9. The deep tendon reflexes of both lower limbs, as well as the superficial abdominal reflexes, were abolished.

The MRI revealed non-specific spinal cord signal abnormalities, presenting as hypo-or isointensityon Tl-weighted imaging and hyperintensity

on T2-weighted imaging, affecting the entire length of the spinal cord with multifocal contrast enhancement after gadolinium injection. Furthermore, the cerebral level was normal.

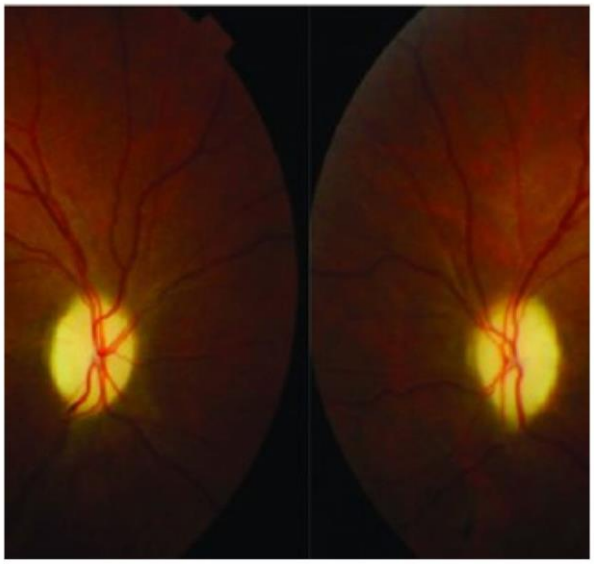

Figure 3: Fundus examination showing bilateral optic disc pallor

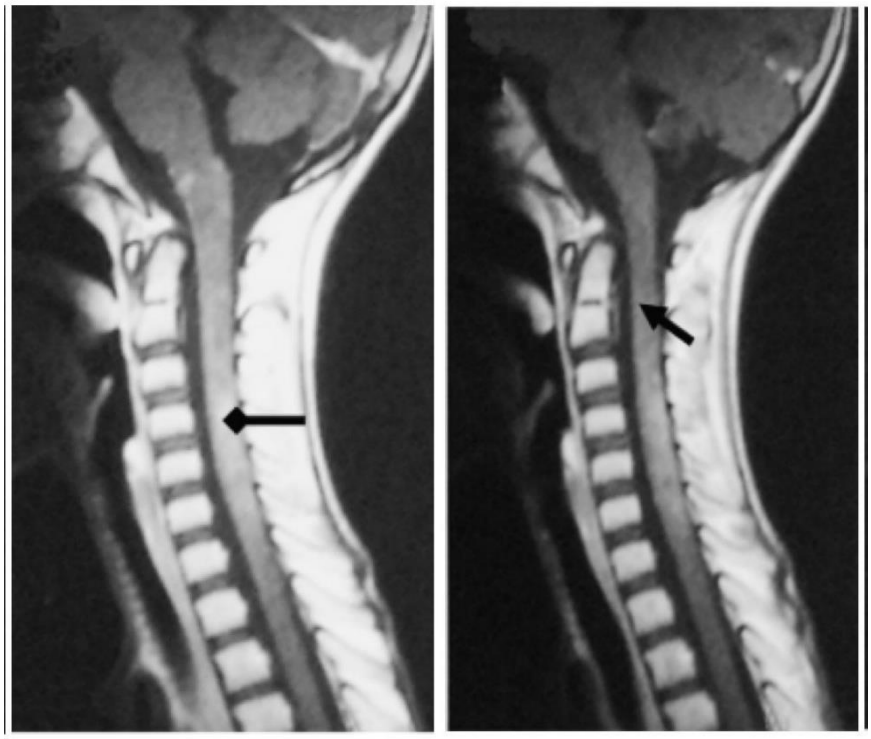

Figure 4: Sagittal section of the spinal MRI showing non-specific spinal cord signal abnormalities, appearing as hypo-or isointensity on T1 and hyperintensity on T2, involving the entire length of the spinal cord with multifocal contrast enhancement following gadolinium injection

The biological assessment showed an erythrocyte sedimentation rate (ESR) of 50 mm at the first hour and 70 mm at the second hour. The lumbar puncture revealed a normal opening pressure with moderate hyperproteinorrhachia at 0.54 g/L. Antinuclear antibodies (ANA)were positive. The diagnosis of NMO was retained.

The patient received corticosteroid therapy consisting of intravenous methylprednisolone for 5 days, which was then relayed by oral prednisone at a dose of 2 mg/kg per day. Starting from the third week, an immunosuppressant, azathioprine, was associated at a dose of 2 mg/kg per day. Additionally, the child underwent motor physiotherapy. The evolution was favorable, with the resolution of neurological symptoms

and an improvement in visual acuity after 1month. (The full-dose corticosteroid therapy was maintained for 2 months, followed by a tapering of 10 mg every 21 days until reaching a maintenance dose of 10 mg/day. This corticosteroid treatment combined with azathioprine was continued for 18 months).

DISCUSSION

Devic's neuromyelitis optica (NMO) is a rare, demyelinating, and inflammatory pathology of the central nervous system that primarily affects the optic nerves and the spinal cord.

It is defined by the association of transverse myelitis and optic neuropathy, which is most often bilateral in 91% of cases and unilateral in 9% of cases, occurring within an 8-week interval. It has been frequently reported in adults and rarely in children, and it appears as a distinct clinical entity.

The mechanism of this selective localization is unknown. It primarily affects young women aged between 18 and 45 years, although cases of Devic's neuromyelitis occurring in children and young adults have been reported.

The typical symptomatology of NMO begins with viral prodromes, followed by transverse myelitis or optic neuritis; the second disorder occurs days or weeks later. In most cases, the decrease in visual acuity is sudden and bilateral, as was the case in our patient, although it is frequently asymmetrical. Papilledema is often moderate.

The mean age of onset is 11 years, with a slight female predominance. The majority of patients had a history of a viral prodrome. The symptomatology is marked by optic neuritis in 66% of cases, including transverse myelitis in 28% of cases.

Optic neuritis is bilateral in 88% of cases, presenting with an acute and severe visual loss. Most patients present with papilledema. The severity of transverse myelitis is similar in most patients, characterized by lower limb paraplegia and hyperreflexia. In our observation, paraparesis was noted upon the young patient's admission.

An infectious etiology was suggested in the work of Scott, whereas current studies have not been able to pinpoint the exact etiology of NMO.

In children, several authors have suggested an autoimmune demyelination initiated by a viral illness to explain the pathogenesis of NMO). In the cerebrospinal fluid, there is a hyperproteinorrhachia greater than 1 g/L, hypercellularity exceeding 50 cells, and an absence of oligoclonal bands.

Brain MRI is most often normal during the acute phase, but during the state phase, T2-weighted sequences frequently reveal small foci included in the white matter of the brain, cerebellum, and spinal cords. Typically, the lesions involve the central part of the spinal cord more than the peripheral part, contrary to multiple sclerosis.

The differential diagnosis of NMO involves a first episode of multiple sclerosis, infectious encephalomyelitis, and acute myelo-optic neuropathy (AMON/SMON) in which gastrointestinal symptoms precede neuro-ophthalmological signs, along with a history of exposure to halogenated hydroxyquinolines and quinolones.

The treatment of Devic's neuromyelitis optica relies on the prescription of high-dose systemic corticosteroids for a short duration during the acute phase, possibly associated with plasma exchange or immunoglobulins. For relapsing forms, immunosuppressive therapy (azathioprine, cyclophosphamide) has been proposed. Preventive treatment with interferon beta is ineffective according to a recent French study.

CONCLUSION

NMO in children is rare. It is a distinct clinical entity with an excellent visual and neurological prognosis. Recurrence is rare and seems to occur without long-term sequelae. In contrast, in adults, the disease can be fatal and often leaves significant ophthalmological and neurological sequelae.

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