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Devic's Neuromyelitis Optica with Positive Anti-Aquaporin-4 Antibody about Three Cases at the National Hospital of Niamey

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Abstract

Introduction: Devic's neuromyelitis optica (NMO), or Devic's disease, is a rare autoimmune disorder that belongs to the inflammatory demyelinating diseases of the central nervous system (CNS). It is a rare syndrome in Western countries, accounting for around 1% of demyelinating diseases of the central nervous system. However, its prevalence is rare in Niger. **Objectives**: To study the epidemiological, clinical, paraclinical, therapeutic and evolutionary aspects of NMO at National Hospital of Niamey. **Methods**: This is a case-report study conducted over a 34-month period from December 2019 to October 2022 involving patients with a diagnosis of NMO according to the latest 2015 Devic Neuromyelitis Optica Spectrum (NMOSD) criteria. **Results**: All cases in our study were girls aged between 12 and 23 years and met the 2015 diagnostic criteria. Initial clinical manifestations were 66% neuritis and 33% myelitis; acute transverse longitudinal myelitis was found in 66% of cases. Anti-Aquaporin-4 (anti-AQP4) antibodies were positive in 100% of cases. Management was based on corticosteroids alone in 66% of cases, and a combination of corticosteroids and prolonged immunosuppression in 33%. The Expanded Disability Status Scale (EDSS) score was 3 after 2 years of treatment and 8 after three weeks of treatment. **Conclusion**: NMO is an inflammatory demyelinating disease of the CNS, preferentially affecting the spinal cord and optic nerves, with a female predominance. Early diagnosis and management can limit neurological disability. Treatment in the acute phase is based on high-dose corticosteroid therapy, or plasma exchange therapy in the event of failure to respond.

Keywords

Anti-Aquaporin-4 Antibody, Devic's Neuromyelitis Optica, National Hospital of Niamey, Niger

1. Introduction

Devic's neuromyelitis optica (NMO), also known as Devic's disease, is a rare autoimmune disorder and one of the inflammatory demyelinating diseases of the central nervous system (CNS) [1]. The condition is rare in Western countries, accounting for around 1% of CNS demyelinating diseases [2]. It is also predominantly female, with a female-to-male sex ratio of 7 in adults and 3 in children. The average age of onset is around 40, but there are pediatric forms and very late-onset forms (after 80 years) [3]. NMO may be under-diagnosed in the West, due to diagnostic difficulties compared to multiple sclerosis (MS). Clinically, NMO is characterized by the occurrence of optic neuritis associated with episodes of extensive longitudinal myelitis. The combination of these neurological disorders can also be seen in MS, ADEM (acute demyelinating encephalomyelitis), systemic lupus erythematosus, Sjögren's syndrome, and more rarely in infectious (syphilis, Lyme disease, measles, herpes viruses, etc.), para-infectious or toxic (cadmium, lead) disorders. These similarities in the clinical presentation of these potentially demyelinating CNS diseases have long led to NMO being regarded as an atypical MS. The pathogenesis of NMO is characterized by an essentially humoral mechanism (B lymphocytes, presence of possibly pathogenic antibodies and complement activation). Recently, the theory of an essentially humoral pathophysiology has been reinforced by the identification of highly specific serum antibodies to NMO, localized at the blood-brain barrier, and directed against a transmembrane water channel called aquaporin-4 (AQP4) detectable in the serum of 75% of surrounding NMO patients [2]. Anti-AQP4 is a diagnostic and prognostic biomarker. Their discovery has broadened the spectrum of NMO to include isolated forms of myelitis or retrobulbar optic neuritis, as well as forms with encephalic or brainstem involvement, and has also highlighted differences in epidemiology, clinical course, pathophysiology, therapy and prognosis [4] [5]. However, a small number of patients (20% - 30%) have no antibodies. For these patients, the diagnostic criteria are stricter and more complex. Some patients meeting the criteria for NMO have additional autoantibodies to a myelin protein called Myelin Oligodendrocyte Glycoprotein (MOG). Although it shares certain similarities with NMO, it is a different disease in terms of mechanisms, treatment and clinical course [3]. The rarity of NMO, its relative lack of awareness, the complexity of diagnostic criteria in seronegative forms, and the specific features of paediatric forms, all contribute to diagnostic erraticism and thus a risk of extremely severe disability, due to delayed and/or inappropriate management [3]. These recent advances now allow us to see NMO as a distinct CNS demyelinating disease, leading to new diagnostic and ther-

apeutic perspectives [2]. Few studies of NMO have been carried out in Africa, even fewer in children, and most have been carried out in the Maghreb, focusing on small series or isolated cases [6]. Our study will focus on the epidemiological, clinical, paraclinical, therapeutic and evolutionary aspects of NMO.

2. Patients and Methods

2.1. Type of Study

This was a prospective, descriptive, cross-sectional study conducted over a 36-month period, from December 2019 to October 2022, in patients managed at the National Hospital of Niamey.

2.2. Inclusion and Non-Inclusion Criteria

All patients with optic neuritis, myelitis and anti-aquaporin-4 positive was include in the study.

Those who did not meet this criterion were not included.

2.3. Ethical and Administrative Considerations

The necessary authorizations were obtained from the Dean of the Faculty of Health Sciences of the Abdou Moumouni University and the Director General of the National Hospital of Niamey. Patient anonymity was strictly respected throughout the study.

3. Results

3.1. Case 1

A 12-year-old female living in Niamey, with no known pathological history, was referred from the ophthalmology department of National Hospital of Niamey for bilateral papilledema. The history of the disease dates back to December 2020, with a sudden drop in visual acuity in both eyes and no other accompanying signs. She reported a malaria attack prior to the illness. On ophthalmological examination, visual acuity was 1/10 in the right eye and 2/10 in the left. Slit-lamp examination showed a clear cornea and anterior chamber; the pupils were semi-mydriatic with little areflexia. Fundus examination showed bilateral papilledema (**Figure 1**). She received corticosteroid therapy with Prednisone 40 mg/24 h, and was then transferred to the neurology department for further management. On admission, neurological examination revealed a conscious patient with good orientation in time and space, Glasgow Coma Scale (GCS) 15/15, muscle strength rated 5/5 in all limbs; tone, osteotendinous and cutaneous-plantar reflexes, and superficial and deep sensitivity were normal. Limited visual field and EDSS score 3. Brain magnetic resonance imaging (MRI) was also normal (**Figure 2**). The biochemical and cytological study of the cerebrospinal fluid (CSF) was normal (normal levels of proteinorrachia, cellulorrachia and glucorrachia), with electrophoresis showing the absence of a gamma globulin oligoclonal profile. Anti-AQP4 antibodies were

weakly positive, while anti-MOG and ANCA antibodies were negative. These results led to the diagnosis of neuromyelitis. He received prednisone 20 mg/24 h for 14 days, then prednisone 15 mg/24 h for 14 days, then 10 mg/24 h for 14 days, and finally 5 mg/24 h for 14 days. He is maintained at 5 mg/24 h Prednisone with adjuvant Calcium and Sodium supplementation. Clinical examination in July 2022 revealed a conscious patient with good orientation in time and space, GCS 15/15, good hemodynamic and ventilatory status, blood pressure (BP) 120/70 mmHg, normal examination of cranial nerves, motricity, tone and sensitivity. Visual acuity was 9/10 in the right eye and 10/10 in the left. Oculomotricity examination showed poor eye convergence, but pupillary reflex was diminished. Pupil reflex was lazy. Visual field examination was normal, with SCORE EDSS (Expanded Disability Status Scale) at 0.

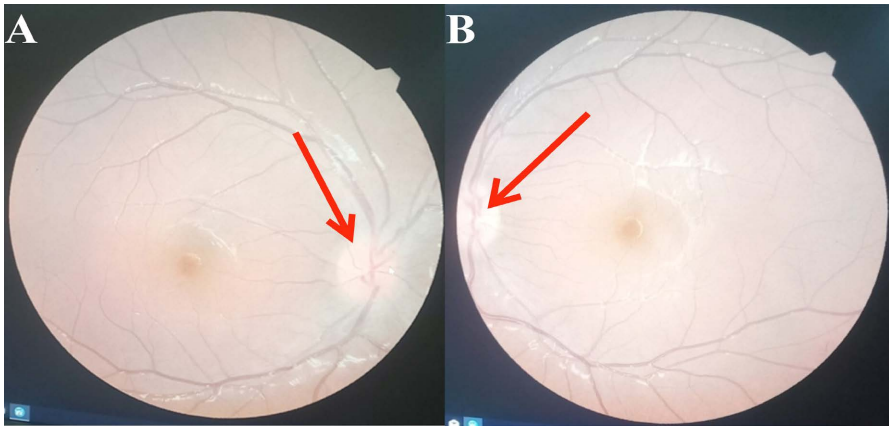

Figure 1. Fundus photograph showing papilledema (red arrows).

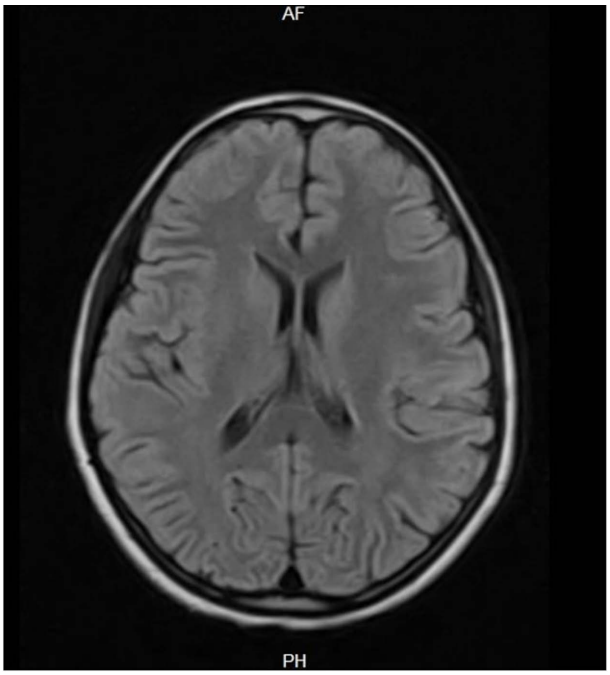

Figure 2. Cerebral MRI image with no abnormalities of Case 1.

3.2. Case 2

A 15-year-old female with no known pathological history was admitted for bilateral blindness with paraplegia, followed by urinary retention. The history began in 2019 with a visual blur, followed by a sudden and reversible drop in visual acuity in the left eye. This prompted admission to a nearby health center, where ophthalmological examination revealed anisocoria and retrobulbar optic neuropathy of the right eye on fundus. She was treated with Prednisone 20 mg/24 h corticosteroids. In view of the recurrence in the left eye, she consulted a neurologist and underwent a cerebral MRI scan, which showed no particularities. Treatment with prednisone 20 mg/24 h was maintained for several months. However, a follow-up cerebral MRI was carried out in view of the persistent drop in acuity in the left eye, which was without abnormality, and the initial treatment was maintained. In August 2021, the patient progressively presented with lower-limb tremors, intense low-back pain, paraparesis that evolved into paraplegia and urine retention, as well as thermo-algesic dysesthesia. Following this clinical picture, she was admitted to the neurology department of National Hospital of Niamey for better management. On admission, she was conscious and well oriented in time and space, with a GCS of 15/15. She presented with bilateral blindness, paraplegia with a motor strength of 0/5, sensory disorders, abolished osteotendinous reflexes, the presence of Babinski's sign, and an EDSS score of 8. Spinal cord MRI revealed a T1 hypo signal, T2 hyper signal and Ir signal abnormality of the spinal cord over a length of 143 mm from the first to the eighth dorsal vertebrae (D1 to D8), with contrast after injection (**Figure 3**). The diagnosis of transverse myelitis was accepted; the CSF showed normal levels of glucose, proteins and cells; protein electrophoresis showed a discrete band in the gamma globulins. Anti-AQP4 antibodies were positive, but IgG and IgM anti-cardiolipin autoantibodies were normal. Sedimentation rate and C-reactive protein were elevated. In view of these results, the diagnosis of Neuromyelitis optica was accepted. The patient received Prednisone 20 mg/24 h and methyl-prednisolone 1 g during the attack. Clinical examination in July 2022 revealed a conscious patient with good orientation in time and space, bilateral blindness and good hemodynamic and ventilatory status. Standing and walking were possible, and proprioceptive tactile and thermal sensitivity were preserved. The rest of the examination was unremarkable. However, episodes were becoming more frequent, and the current EDSS SCORE was 3.

3.3. Case 3

This 23-year-old married woman with no pathological history was admitted with tetraparesis. The history of the disease dates back to two weeks before her admission, with the sudden onset of motor deficits in the lower limbs, followed within a few hours by those in the upper limbs, all associated with intense, diffuse neuropathic pain. She underwent conventional treatment without success, and was then referred to the neurology department for further treatment. On admission,

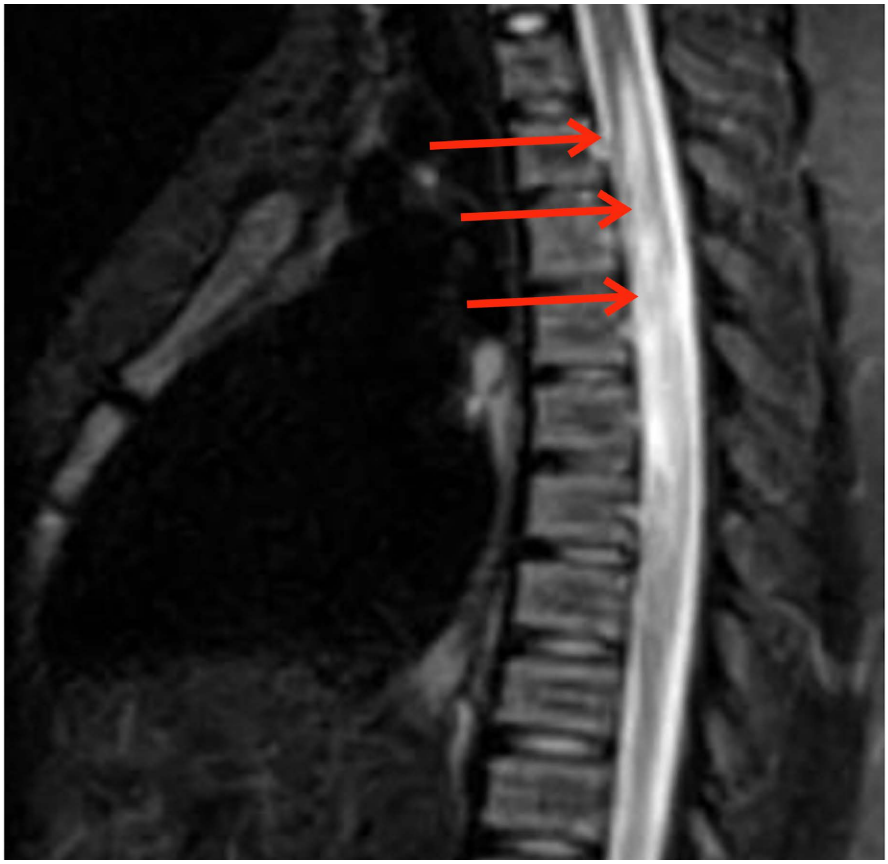

Figure 3. Spinal cord MRI of Case 2 showing extensive T2 hyper signal over more than 3 vertebrae (red arrows).

neurological examination revealed a conscious patient with good orientation in time and space, GCS 15/15, tetraparesis rated 0/5 in the lower limbs and 2/5 in the upper limbs, global hypotonia, sharp osteotendinous reflexes and indifferent cutaneous-plantar reflexes. There was also diffuse hyperesthesia, but more marked in the territories of the fifth and sixth cervical spinal nerves (C5-C6). Examination of the cranial pairs was unremarkable, and the EDSS score was 8.5. Spinal cord MRI showed a chronic spinal cord lesion (**Figure 4**), which led to the diagnosis of myelitis. LCS protein electrophoresis revealed a discrete gamma globulin band; anti-DNA, ANCA and anti-MOG were negative, while anti-AQP4 antibodies were weakly positive. Fundus examination revealed no anterior or posterior uveitis, and non-glaucomatous optic neuritis. Sedimentation rate and C-reactive protein were elevated. She received corticosteroid therapy with methylprednisolone 500 mg/24 h for 5 days, followed by prednisone 40 mg/24 h, and adjuvant treatment with potassium and calcium supplements. Persistent neuropathic pain led to the prescription of gabapentin 75 mg/24 h then 150 mg/24 h, combined with amitriptyline 10 mg/24 h drops. This was followed by a week of azothioprine 50 mg/24 h, then 100 mg/24 h. The evolution was marked by a clearer regression of the neuropathic pain and a slight recovery of the tetraparesis. The EDSS SCORE was estimated at 8.

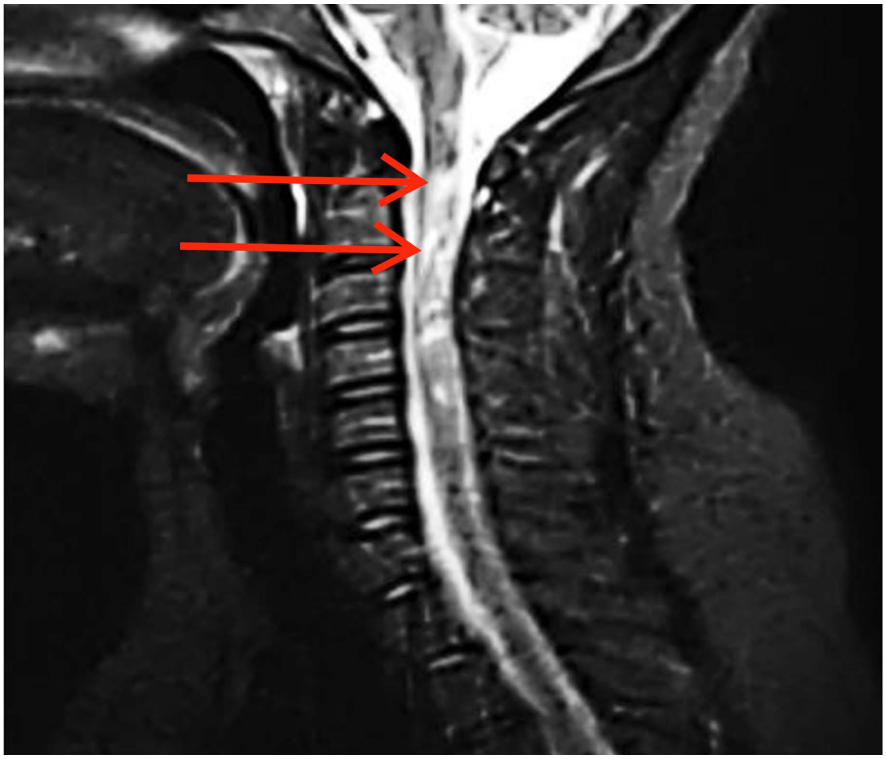

Figure 4. Spinal cord MRI of Case 2 showing extensive T2 hyper signal over more than 3 vertebrae (red arrows).

4. Discussion

4.1. Sociodemographic Aspects

Neuromyelitis optica is a rare, severe autoimmune disease caused by autoantibodies directed against aquaporin 4 (AQP4), with a high morbidity and mortality rate [7]. Indeed, the estimated prevalence is around 5 cases per 100,000 people, although this is higher in populations of Asian and African origin [8]. It is considered a rather rare syndrome in Western countries, accounting for around 1% of demyelinating diseases of the central nervous system (CNS) [2]. In Africa, few studies have been carried out on NMO, even fewer in children, and most have been carried out in the Maghreb, focusing on small series or isolated cases [6]. In our study, 3 cases were reported, fewer than those of Ben Aoun *et al.* [9], and Jeffery *et al.* [10]. This can be explained by the fact that our study concerned only anti AQP4-positive cases.

4.1.1. Sex

In our study, 100% of cases were women, with no male cases recorded. Our results concur with those of Ben Aoun *et al.* [9]. According to PNDS, they affect women much more frequently than men, with a female-to-male sex ratio of 7 in adults and 3 in children [3].

4.1.2. Age

The age of onset of our patients ranged from 11 to 23 years, with an average age

of 23. The average age of onset is around 40 years, but there are pediatric forms and very late-onset forms (after 80 years) [3]. Our results are lower than those of Cabre *et al.* (30.9 years) [11]. This could be explained by the very young population of our study and the coexistence of pediatric and adult cases.

4.2. Clinical Description

Devic's neuromyelitis optica is an inflammatory demyelinating disease of the central nervous system, preferentially affecting the spinal cord (myelitis) and optic nerves (optic neuritis) [12]. The discovery of anti-AQP4 antibodies and their specificity has led to the description of new clinical forms involving other structures of the central nervous system, including the brainstem, whose area postrema causes hiccups and incoercible vomiting, the diencephalon, which can lead to narcolepsy, and the encephalon. This antibody has also been used to identify monofocal forms (optic neuritis or extensive myelitis) with the same prognosis as NMO [13] [14]. In our study, we had 2 out of 3 cases of monofocal involvement and 1 out of 3 cases of neuritis. There were no cases of involvement of the brain stem, encephalon or diencephalon. Initial clinical manifestations were optic neuritis in 2 out of 3 cases and myelitis in 1 out of 3 cases. In a series of 18 pediatric cases of NMOSD. Jeffrey *et al.* found that the first manifestation of NMOSD was optic neuritis in 66% of cases, myelitis in 28% and concomitant damage in 6% [10]. Our results concur with those of Jeffrey *et al.* [10]. This is explained by the predominance of pediatric cases in our study. The clinical manifestations at the time of diagnosis were neuritis in 1 out of 3 cases, myelitis in 1 out of 3 cases and neuritis associated with myelitis in 1 out of 3 cases. These figures differ from the initial percentages, which can be explained by the fact that optic involvement may precede spinal cord involvement, resulting in retrobulbar optic neuritis recurring several years before spinal cord involvement, as was the case in our second patient (**Case 2**).

4.3. Paraclinical Description

4.3.1. Biology

1. CSF Study

The CSF study was carried out in all our patients and came back normal, in contrast to the literature, which speaks of an abnormal study with an often elevated level, exceptionally exceeding 1 g/l [15] [16]. Pleocytosis is observed in 1 in 3 cases if the CSF is collected at the time of a relapse [15]. Our results differ from those of De Seze *et al.* 2002 [16].

2. CSF Electrophoresis

The presence of oligoclonal bands was observed in 2/3 of our patients. The presence of oligoclonal bands in CSF varies from study to study, with 23% of cases in the series by De Seze *et al.* in 2002 [16]. Our results are much higher, which may be explained by the fact that the test was performed during myelitis attacks.

3. Anti-AQP4

In our study, all patients tested positive for anti-AQP4 antibodies. Anti-AQP4

antibodies detectable in serum in 75% of surrounding NMO patients (Bernard *et al.* 2015) [4].

Aquaporin 4 antibodies may be negative, but this does not invalidate the diagnosis of optic neuritis [4].

4.3.2. Imaging

In our study, MRI of the spinal cord and/or brain was performed in all patients, with no cases of cerebral involvement. Acute transverse longitudinal myelitis (MALT) was observed in 2 out of 3 cases (66%) of our patients. The most characteristic feature on spinal cord MRI is the presence of a lesion extending longitudinally over 3 or more vertebral segments (**Figure 3**, **Figure 4**), with T1 hyposignal and T2 hypersignal, and possible gadolinium uptake in the acute phase. As already indicated, encephalic imaging is usually normal at the outset, but non-specific white matter hypersignals are not uncommon. Much more rarely, diencephalic or periaqueductal lesions have been described and may be quite specific for Devic's disease [12].

In a study carried out in the neurology department of CHU Habib Bourguiba Sfax by Amal H. K. *et al.* during a 6-year period (2014-2020) including all patients followed for NMO, according to the 2015 International Panel for NMO Diagnosis (IPND) criteria and having positive anti-aquaporin-4 or anti-MOG to show that on brain MRI, the majority of lesions were typical, such as involvement of the periventricular ependymal regions (33% of patients), area postrema (20%), optic nerve (13%), corticospinal bundle (13%) and cloudy enhancement (6%), 13% of patients had atypia (cortical hyper signal, or adjacent and perpendicular to the lateral ventricles) [17]. Acute transverse longitudinal myelitis was observed in 60% of cases [17]. Our results concur with those of Amal *et al.*, Pittock *et al.*, Wingerchuck *et al* for spinal cord imaging and differ for brain MRI [17]-[19]. This may be explained by the fact that the brain MRI of our patients was performed in the initial phase of the disease, and the spinal cord MRI during the spinal cord crisis.

4.3.3. Fundus

In our study, all our patients had a fundus examination, including 1 in 3 cases of papilledema, 1 in 3 cases of NORB, and 1 in 3 cases of optic neuritis. In the acute stage, fundus examination is usually normal, with optic nerve inflammation preferentially located in the posterior part of the optic nerve or at the level of the optic chiasm [18]. This may be explained by the predominance of ocular manifestations in our study.

4.4. Therapy

In our study, all patients received corticosteroid therapy in the acute phase, including 2 cases with methylprednisolone combined with prednisone, and 1 case with prednisone. Despite their AQP4 positivity, only 1 of 3 cases received an immunosuppressant (azothioprine) as background treatment, while 66% received oral corticosteroids. Neuropathic pain was treated with gabapentin, with a favor-

able outcome. Treatment in the acute phase is based on high-dose corticosteroids, or plasma exchange in case of failure to respond. Prevention of recurrence is based on prolonged immunosuppression, with encouraging results [12]. In addition to its diagnostic value, AQP4 also has prognostic utility. Thus, the serological status of anti-AQP4 antibodies is important in therapeutic management. In fact, patients with a positive anti-AQP4 antibody assay should benefit from immunosuppressant-based disease-modifying therapy to prevent relapses. Tricyclic antidepressants and antiepileptics such as carbamazepine or gabapentin are generally highly effective [19]. The use of immunosuppressants in our study was insignificant in relation to the AQP4 positivity of our patients. The predominant use of corticosteroids in our study may be explained by the accessibility and cost of these products.

4.5. Prognosis

4.5.1. Visual Impairment/Motor Deficit

In our study, the first patient (**Case 1**) with optic neuropathy recovered completely, the second patient (**Case 2**) developed blindness with no motor sequelae, and the third patient (**Case 3**) had no significant immediate evolution. In children, the functional prognosis is better than in adults: Jeffrey *et al.* [10] reported that recovery from neurological and ophthalmic signs was rapid and complete in 17 of 18 children with NMOSD [8]. After a mean follow-up of 3.9 years, from 3 months to 12 years, only one recurrence was observed [10]. However, the publication by Hor JY *et al.* [20], states that there is a higher risk of developing blindness in children with optic neuropathy than in adults, but observes on the contrary a better functional prognosis in children with myelitis, often with no permanent motor sequelae [20]. Our results are in line with those of Hor JY *et al.* [20], which may be explained by the predominance of paediatric cases in our study.

4.5.2. EDSS Score

The EDSS score was 3 in our second patient (**Case 2**) after 2 years of treatment, and 8 in the third patient (**Case 3**) after two weeks of treatment. Disability is currently assessed using the EDSS scale, although this is not well adapted to NMO, particularly with regard to visual functions. In a French cohort, 56% of patients had reached EDSS 6, corresponding to the need to use a cane to walk about 100 meters, with an average delay of 10 years, and 7 years in an Italian cohort [21] [22]. Our results differ from those reported in the literature (Ghezzi *et al.*, and Collongues N. *et al.*) [21] [22]. This may be explained by the youth of our study population and the fact that the adult case is not in the initial phase of treatment.

5. Conclusion

Devic's neuromyelitis optica (DNMO) is an inflammatory demyelinating disorder of the central nervous system, preferentially affecting the spinal cord and optic nerves. The predominance of females was clear, with coexistence of pediatric and adult cases. Diagnosis is based on NMOSD diagnostic criteria. Treatment in the

acute phase is based on high-dose corticosteroid therapy, or plasma exchange in case of escape. Prevention of recurrence is based on prolonged immunosuppression, with encouraging results. Early diagnosis and prompt initiation of treatment to prevent relapses can limit neurological disability.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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