Received: 28 February 2024 | Revised: 24 April 2024 | Accepted: 1 June 2024

DOI: 10.1002/ccr3.9063

CASE REPORT

Clinical Case Reports Open Access WILEY

A rare case of systemic lupus erythematosus-associated neuromyelitis optica spectrum disorder with cystic lesions and dual seropositivity for anti-AQP4 and anti-MOG antibodies

Omar Al Jassem[1] | **Rami Rifi**[1] | **Karim Kheir**[1] | **Alaa Masri**[1] | **Hassan Eid**[2]

[1]Faculty of Medical Sciences, Lebanese University, Beirut, Lebanon

[2]Department of Neurology, New Mazloum Hospital, Tripoli, Lebanon

Correspondence
Omar Al Jassem, Faculty of Medical Sciences, Lebanese University, Beirut, Lebanon.
Email: omaror75@gmail.com

Key Clinical Message

In patients with SLE, concurrent NMOSD can manifest with optic neuritis and transverse myelitis. AQP-4 antibody positivity confirms the diagnosis. Prompt treatment is critical to manage the acute symptoms and prevent relapses, as highlighted by a young patient's case with optic neuritis and extensive spinal cord lesions.

Abstract

Neuromyelitis optica spectrum disorder (NMOSD) is a rare autoimmune disorder of the central nervous system that affects the optic nerve and spinal cord. It is associated with autoantibodies against aquaporin-4 (AQP-4) and/or myelin oligodendrocytes glycoproteins. It is diagnosed based on clinical, radiological, and serological criteria, and treated with immunosuppressants in the acute phase. Long-term immunosuppression is essential to prevent potential relapses. In this case report, we present the case of a 19-year-old female patient with systemic lupus erythematosus (SLE), who presented with blurriness and loss of vision in her left eye. Optical coherence tomography was normal, but a gadolinium-enhanced cervico-dorsal MRI showed multiple lesions extending from the brainstem to the C7-T1 junction suggestive of longitudinally extensive transverse myelitis (LETM), the largest of which was a cystic lesion at the cervico-spinal junction. A contrast injection also revealed left optic neuritis. Cerebrospinal fluid analysis showed elevated IgG and red blood cell count, but no oligoclonal bands. The patient tested positive for AQP-4 autoantibodies, confirming the diagnosis of NMOSD. Treatment with intravenous methylprednisolone led to partial improvement, but the patient experienced a relapse with severe neurological symptoms, including tetraplegia and bladder and bowel dysfunction. This case illustrates the importance of considering NMOSD in the differential diagnosis of patients with SLE who present with optic neuritis and/or myelitis, especially when MRI findings are suggestive of LETM. Early diagnosis and adherence to treatment are crucial to prevent further relapses and deleterious sequelae.

KEYWORDS
aquaporin-4 antibodies, autoimmune diseases, neuromyelitis optica spectrum disorder, optic neuritis, transverse myelitis

1 | INTRODUCTION

Neuromyelitis optica spectrum disorder (NMOSD) is a rare autoimmune demyelinating inflammatory disorder of the central nervous system (CNS) that predominantly affects the optic nerve and spinal cord, often leading to severe disability and poor prognosis. NMOSD is associated with autoantibodies against aquaporin-4 (AQP-4), a water channel protein expressed in astrocytic foot processes, and/or autoantibodies against myelin oligodendrocytes glycoproteins (MOG).[1,2]

The diagnosis of NMOSD is based on clinical, radiological, and serological criteria. The appropriate treatment consists of managing the acute phase with high-dose corticosteroids and/or plasma exchange, in addition to long-term immunosuppression to prevent relapses. Nevertheless, some patients may be noncompliant with the treatment, or have contraindications or adverse reactions to the prescribed medications, leading to additional complications.[3]

The discovery of NMOSD can be traced back to 1894, when Dr Eugène Devic and his doctoral student Fernand Gault first delineated the condition, leading to its subsequent recognition as Devic's disease.[4] While initially categorized as a subtype of multiple sclerosis (MS), NMOSD is now universally recognized as an independent disorder.[5] In fact, NMOSD has a prevalence of 0.3 to 4.4 cases per 100,000 individuals, and is more commonly found in individuals of Asian or African descent. It is, however, less prevalent among Europeans.[6]

2 | CASE REPORT

2.1 | Case history and methods

In the following case report, we present the case of a 19-year-old female, who is known to have systemic lupus erythematosus (SLE) for 6 years, treated with hydroxychloroquine.

Our patient presented to an ophthalmologist with a 1-week history of blurriness and loss of vision in her left eye. Her visual acuity was 1/200 in the left eye and 100% in the right eye. The patient could only notice hand motion in the central view, but was able to count fingers in the temporal view. She had no pain, redness, or discharge from her eyes. Her intro-ocular pressure, slit-lamp examination, and funduscopy were all normal.

Optical coherence tomography (OCT) was performed, after cessation of hydroxychloroquine, to exclude any visual toxicity due to secondary effects of this medication. OCT showed normal results, excluding retinopathy, corneal deposits, glaucoma, macular edema, and optic neuropathy.

For further evaluation, our patient was referred to a neurologist, where she denied any headache, fever, seizures, weakness, or bladder or bowel problems. Numbness and neck stiffness were reported by the patient. Otherwise, her neurological examination was normal.

A gadolinium-enhanced cervico-dorsal MRI of the spine was performed, showing several hyperintense lesions in the spinal cord, the biggest of which was seen in the cervical spine, presenting as a cystic spinal lesion, and causing an increase in the thickness of the cervical spinal cord. This lesion extended from the brainstem to the level of the C7-T1 intervertebral disc, suggesting a diagnosis of longitudinally extensive transverse myelitis (LETM). Furthermore, three noncystic lesions were detected at the level of the dorsal spine: a 15 mm lesion at the level of T3, as well as two lesions located between T8 and T10, measuring 15 mm and 30 mm, respectively. Moreover, a contrast injection at the cerebral level showed a small contrast enhancement of the left optic nerve, suggesting left optic neuritis (Figure 1A).

In addition to these radiological findings, a cerebrospinal fluid (CSF) analysis showed normal protein, glucose, and LDH levels, as well as an elevated IgG level and red blood cell count, but no oligoclonal bands, as shown in Table 1.

To rule out conditions like neurosarcoidosis and subacute combined neurodegeneration, a chest X-ray and vitamin B12 level were ordered, respectively, but turned out to be normal. Moreover, acid-fast and Gram stains showed no bacterial growth in the CSF after 4 days of incubation, excluding any potentially concomitant bacterial infections.

Interestingly, our patient tested positive for both AQP-4 and MOG autoantibodies, which are specific serological markers for NMOSD.

3 | RESULTS

Based on the previous data, our patient was diagnosed with NMOSD, and intravenous (IV) methylprednisolone

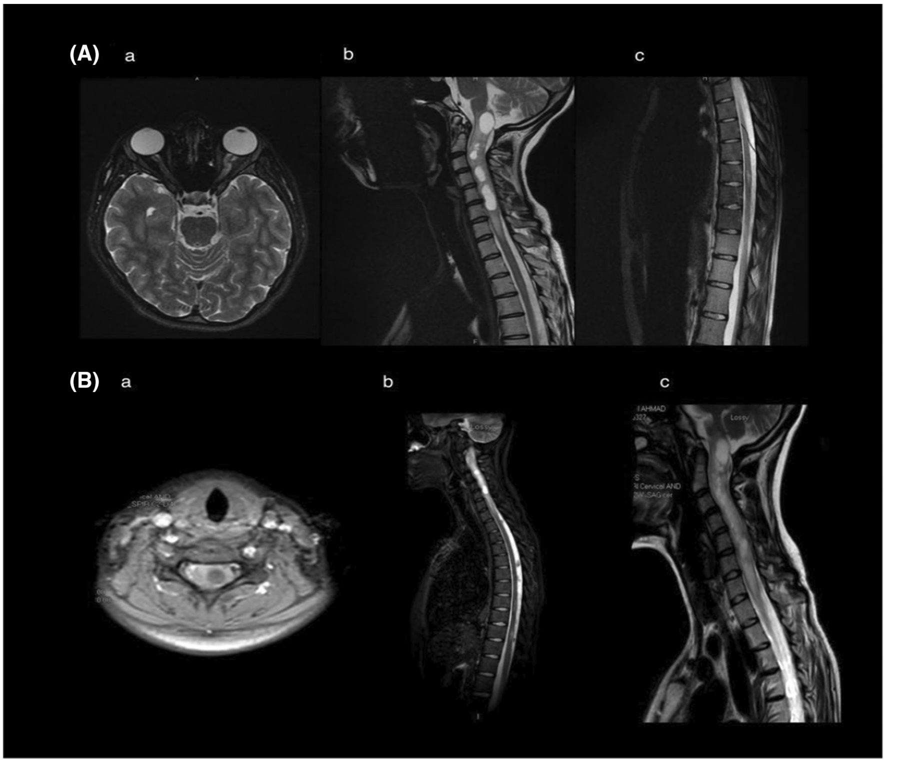

FIGURE 1 MRI of the brain and spine. (A.a) Brain MRI at presentation (T2-weighted axial sections). (A.b and A.c) show sagittal T2-weighted MRI images of the cervical spine and thoracic spine, respectively. (B.a) shows an axial T1-weighted MRI image of the spine. (B.b and B.c) show sagittal T1-weighted MRI images of the spine.

TABLE 1 CSF analysis and oligoclonal banding of our patient.

	Measured variables	Reference range	Results
Cerebrospinal fluid chemistry	Glucose levels (mg/dL)	40–80	66
	LDH (U/L)	<70	<41
	Protein levels (g/L)	0.12–0.6	0.49
Oligoclonal banding (CSF/Serum) by electrophoresis	IgG-CSF (mg/dL)	0–3.4	8.89
	IgG-Serum (g/L)	7–16	15.2
Cerebrospinal fluid cell count	Leukocytes (cells/uL)	0–5	5
	Erythrocytes (cells/uL)	0–5	1000
	Lymphocytes (%)	60–70	100%
	Neutrophils (%)	0–6	0%
	Monocytes (%)	15–30%	0%
	Eosinophils (%)	0	0
	Basophils (%)	0	0

was initiated for 5 days, after which complete visual recovery was achieved. The patient was then discharged with a plan to taper oral prednisone. Nevertheless, she did not adhere to her medication regimen, and was readmitted 15 days later with a relapse of NMOSD, manifesting as tetraplegia and loss of proprioception and sensation in both arms, in addition to bladder and bowel dysfunction.

Upon readmission, a new MRI revealed a diffuse, extensive infiltrating-like process, involving the cervical cord from the middle of the medulla oblongata down to T1 vertebra, with a swollen cervical cord showing a heterogeneous signal, compatible with multifocal cystic zones. The cervical cord's thickness was estimated to be 17 mm at the cranio-cervical junction, where the largest cyst was identified (Figure 1B).

The patient was treated again with solumedrol/methylprednisolone sodium succinate, 1-g IV bolus in 250 cc normal saline solution for 5 days, omeprazole, 40-mg IV, enoxaparin sodium, 20-mg subcutaneously, paracetamol, 1-g IV every 6 h, glycerin suppositories, and rituximab 1000 mg twice 14 days apart.

Due to the development of tetraplegia, our patient was transferred to a rehabilitation center for physiotherapy. All her laboratory results, upon the first and second admissions, are shown in Table 2.

4 | DISCUSSION

The following case report illustrates the clinical features, diagnosis, and treatment of NMOSD, a rare and potentially devastating disorder that affects the optic nerve and spinal cord. NMOSD is associated with serum autoantibodies against AQP4 and/or MOG, which are markers of disease activity and prognosis. The resultant CNS damage is caused by the presence of serum autoantibodies that bind to the astrocytic water channel protein AQP4 at the foot processes of astrocytes.

AQP4 is the most common type of aquaporin in mammals' brains, where it normally maintains water

TABLE 2 The blood tests' results at the New Mazloum Hospital, upon the first and second admissions.

	Measured variables	Reference range	First admission (2/1/2024)	Second admission (22/1/2024)
Blood chemistry	Glucose (mg/dl)	74–106	110	100
	Creatinine (mg/dl)	0.52–1.04	0.6	0.4
	eGFR (mL/min/1.73m^2)	>90	128.79	205.63
	Sodium (mmol/L)	137–145	140	137
	Potassium (mmol/L)	3.5–5.1	4	4.1
	Chloride (mmol/L)	98–107	106	104
	Carbon dioxide (mmol/L)	22–30	26	21
	Calcium (mg/dl)	8.4–10.2	9.9	9.2
	Protein, Total (g/L)	63–82	71	–
	ALT (SGPT) (U/L)	5–35	15	32
	Lactate dehydrogenase (U/L)	120–246	135	–
	C-reactive protein (mg/L)	0–10	6.3	6.1
Endocrinology	TSH (mU/L)	0.35–4.94	1.2930	–
Hematology	Leukocytes ($\times 10^3/\mu L$)	3.39–8.86	12.35	4.24
	Erythrocytes ($\times 10^6/\mu L$)	3.91–5.31	4.27	4.19
	Hemoglobin (g/dL)	10.6–14.8	12.3	11.6
	Hematocrit (%)	32.9–41.2	34.1	32.5
	MCV (fL)	77.7–93.7	79.9	77.6
	Platelets ($\times 10^3/\mu L$)	186–353	352	240
	Neutrophils (%)	42.5–73.2	79.2	67.7
	Lymphocytes (%)	18.2–47.4	14	23.6
	Monocytes (%)	4.3–11	6.6	8.0
	Eosinophils (%)	1–4	0.1	0.5
	Basophis (%)	0–0.7	0.1	0.2
Vitamins	Vitamin B12 (pg/mL)	400–883	730	–
Immuno-serology	Anti-aquaporin-4 titer		1/32	–
	Anti-MOG titer		1/8	–

homeostasis and helps mediate waste protein clearance.[7] Within the CNS, AQP4 is most abundant in the optic nerve, the hypothalamus, the cerebellum, the para-ventricles regions, and the spinal cord. Moreover, it is also present in other organs, including the kidneys, and the digestive and respiratory systems.[8]

It has been shown that AQP4 antibodies, also known as NMO IgG, are very specific (94%), and moderately sensitive (76%) for NMOSD.[9] The pathophysiology of NMOSD is mediated by these AQP4 immunoglobulins (IgGs) that enter the CNS via the blood–brain barrier (BBB) and selectively bind to the AQP4 channels at the foot processes of astrocytes.[10] While NMO IgG cannot cross the BBB in healthy individuals to cause CNS disorders, the antibodies that are produced extrathecally disrupt the BBB to provoke NMOSD in the affected patients.[11] In the latter, these autoantibodies mainly belong to the IgG1 subtype (98%), which can strongly activate the complement system.[12] The subsequent complement activation can increase the permeability of the BBB, with additional recruitment of pro-inflammatory leukocytes (neutrophils, eosinophils, macrophages, and natural killer cells), promoted by C5a, causing astrocyte and neuronal damage, and potentially death due to the deposition of C5b-C9 complexes. Moreover, the AQP4 antibody-complement-mediated cytotoxicity is a major cause of damage to AQP4-expressing astrocytes.[10]

Another mechanism that explains the astrocytic damage in NMOSD patients is mediated by antibody-dependent cellular cytotoxicity. In fact, mature B cells that produce autoantibodies against AQP-4 proteins can also trigger the production of interleukin-6 (IL-6), which helps break down the BBB and enhances B cells survival. Plasmablasts that are supported by IL-6 increase the release of AQP4-IgG, with the help of AQP4-reactive T cells, and Th17-related inflammatory cytokines.[10]

Furthermore, AQP4-binding antibodies can provoke glutamate impairment by downregulation of the excitatory amino acid transporter 2, which is responsible for the extracellular glutamate clearance to prevent neuronal excitotoxicity and hyperexcitability.[13]

Additionally, the death of astrocytes results in the loss of support for the surrounding neurons and oligodendrocytes. Damage to the latter appears to follow the immune-mediated astrocyte injury. Consequently, demyelination is a secondary event occurring in NMOSD patients due to significant losses in glial astrocytes and oligodendrocytes.[10]

NMOSD may be idiopathic, or can occur in association with other autoimmune diseases such as SLE, as seen in the case of our patient.[14] While patients of any age can be affected, this disorder is more common in women, and usually starts around the age of 39. It includes several clinical syndromes: optic neuritis (ON), LETM, area postrema syndrome, acute brain stem syndrome, diencephalic syndrome, and symptomatic cerebral syndrome.[6]

According to the 2015 International Panel for NMOSD Diagnostic Criteria for Adult Patients, our patient met all the criteria for "NMOSD with positive AQP4-IgG", as evidenced by the presence of ON and LETM, positive AQP4-IgG, and exclusion of alternative diagnoses as shown in Table 3.[15] In fact, vitamin B12 and chest X-ray were ordered to rule out subacute combined neurodegeneration and neurosarcoidosis, respectively, but turned out to be normal. Moreover, while short spinal segment lesions are a common finding seen in MS, the spinal MRI of our patient showed extensive lesions, involving more than three segments, which makes the diagnosis of MS very unlikely.[6]

While the pathophysiological link between SLE and NMO remains unclear, some studies have estimated that the probability of having both SLE and NMO in the same individual is about one in 5,000,000.[14]

Differentiating between lupus myelitis and NMO or MS in patients with SLE is challenging: myelitis in SLE usually occurs 1–2 years after the onset of SLE and is associated with SLE flares, whereas in NMO and MS, myelitis occurs several years later and is not related to flares of SLE. Moreover, while uncommon in MS, SLE myelitis and NMO can both cause ON. In addition, MRI findings are crucial to distinguish between these entities: in SLE myelitis and MS, the lesions involve one to two spinal segments, or rarely present as LETM, affecting three or more vertebral segments. However, in NMO, LETM is very common. Furthermore, brain involvement is rare in NMO, but is commonly detected in the MRI of patients with SLE myelitis and MS.

TABLE 3 The 2015 international panel for NMOSD diagnostic criteria for adult patients.

Diagnostic criteria for NMOSD with AQP4-IgG
1. At least one core clinical characteristic
2. Positive test for AQP4-IgG using best available detection method (cell-based assay strongly recommended)
3. Exclusion of alternative diagnoses
Diagnostic criteria for NMOSD without AQP4-IgG or NMOSD with unknown AQP4-IgG status
1. At least two core clinical characteristics occurring as a result of one or more clinical attacks and meeting all of the following requirements:
(a) At least one core clinical characteristic must be optic neuritis, acute myelitis with LETM, or area postrema syndrome
(b) Dissemination in space (two or more different core clinical characteristics)
(c) Fulfillment of additional MRI requirements, as applicable
2. Negative tests for AQP4-IgG using best available detection method, or testing unavailable
3. Exclusion of alternative diagnoses
Core clinical characteristics
1. Optic neuritis
2. Acute myelitis
3. Area postrema syndrome: episode of otherwise unexplained hiccups or nausea and vomiting
4. Acute brainstem syndrome
5. Symptomatic narcolepsy or acute diencephalic clinical syndrome with NMOSD-typical diencephalic MRI lesions
6. Symptomatic cerebral syndrome with NMOSD-typical brain lesions
Additional MRI requirements for NMOSD without AQP4- IgG and NMOSD with unknown AQP4-IgG status
1.Acute optic neuritis: requires brain MRI showing (a) normal findings or only nonspecific white matter lesions OR (b) optic nerve MRI with T2-hyperintense lesion or T1-weighted gadolinium enhancing lesion extending over >1/2 optic nerve length or involving optic chiasm
2. Acute myelitis: requires associated intramedullary MRI lesion extending over >3 contiguous segments (LETM) OR >3 contiguous segments of focal spinal cord atrophy in patients with history compatible with acute myelitis
3. Area postrema syndrome: requires associated dorsal medulla/area postrema lesions
4. Acute brainstem syndrome: requires associated periependymal brainstem lesions

Additionally, the assessment of oligoclonal bands can be of great benefit, since these bands are mostly absent in both NMO and lupus myelitis, but are commonly present in patients with MS.[16,17]

In patients with NMOSD, the treatment aims to reduce the inflammation, and to prevent potential relapses that can cause irreversible neurological damage and disability. However, our patient did not comply with the recommended treatment due to depression, and developed total paraplegia with loss of proprioception and sensation in both arms upon the second admission. Therefore, it is crucial to highlight the necessity for persistent follow-up in the case of NMOSD patients, coupled with rigorous education, to prevent such disabilities and permanent damage, including blindness and paralysis.[18]

Furthermore, acute episodes of NMOSD are typically managed using corticosteroids, such as IV methylprednisolone, and therapeutic plasma exchange may be considered for cases with severe or refractory attacks. In fact, plasma exchange removes up to 99% of circulating autoantibodies after 5–7 cycles, and may also eliminate other inflammatory factors that contribute to the disability. If symptoms persist without improvement after both steroid administration and plasma exchange, the option of treatment with IV immunoglobulins or an escalation to cytoablative therapy, such as cyclophosphamide, may be offered to affected patients. For long-term prevention, immunosuppressive agents such as azathioprine, or treatment involving B cell-targeted therapies like intravenous rituximab, are used. Tocilizumab is considered to be a second-line treatment in NMOSD after rituximab, as it can inhibit the IL-6 receptors, and provoke a reduction in the AQP4-IgG production, in addition to decreasing the permeability of the BBB. Additional alternatives include mycophenolate mofetil, methotrexate, and mitoxantrone. However, the latter is often avoided as an initial treatment due to significant side effects, including cardiotoxicity and leukemia. Since some patients may be noncompliant with the treatment or have contraindications or adverse effects to the prescribed medications, poor outcomes and complications may be observed in such patients.[10,19] Therefore, rigorous education regarding the nature and management of NMOSD is highly encouraged, in addition to continuous monitoring of the adherence and response of patients to the treatment. Early diagnosis and treatment of NMOSD can improve the quality of life and prognosis of the disease.

5 | CONCLUSION

This paper elucidates the clinical aspects of neuromyelitis optica spectrum disorder in a patient that presented with recurrent episodes of optic neuritis and longitudinal extensive transverse myelitis, leading to potentially visual and motor impairments. The diagnosis of neuromyelitis optica spectrum disorder is based on clinical and serological criteria, particularly on the presence of aquaporin-4 autoantibodies. However, the diagnosis and treatment of neuromyelitis optica can be challenging, especially in developing countries, where limited access to specialized care and advanced imaging techniques can hinder the appropriate management of this condition. To prevent the deleterious sequelae of neuromyelitis optica, physicians should ensure appropriate management of the acute phase of the disease, and must maintain regular evaluation to prevent relapses and monitor potential complications.

AUTHOR CONTRIBUTIONS

Omar Al Jassem: Conceptualization; methodology; writing – original draft; writing – review and editing. **Rami Rifi:** Conceptualization; writing – original draft. **Karim Kheir:** Conceptualization; methodology; writing – original draft; writing – review and editing. **Alaa Masri:** Conceptualization; writing – original draft. **Hassan Eid:** Conceptualization; methodology; supervision; writing – original draft; writing – review and editing.

ACKNOWLEDGMENTS

There are no acknowledgments for this submission.

FUNDING INFORMATION

The authors did not receive any financial support during the conduction of this research.

CONFLICT OF INTEREST STATEMENT

The authors have no conflicts of interest to disclose.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Omar Al Jassem https://orcid.org/0009-0009-4691-846X

REFERENCES

1. Carnero Contentti E, Correale J. Neuromyelitis optica spectrum disorders: from pathophysiology to therapeutic strategies. *J Neuro-Oncol*. 2021;18(1):208. doi:10.1186/s12974-021-02249-1
2. Spiezia AL, Carotenuto A, Iovino A, et al. AQP4-MOG double-positive neuromyelitis optica Spectrum disorder: case report

with central and peripheral nervous system involvement and review of literature. *Int J Mol Sci*. 2022;23(23):14559. doi:10.3390/ijms232314559
3. Sato D, Callegaro D, Lana-Peixoto MA, Fujihara K. Treatment of neuromyelitis optica: an evidence based review. *Arq Neuropsiquiatr*. 2012;70:59-66. doi:10.1590/S0004-282X2012000100012
4. Shumway CL, Patel BC, Tripathy K, De Jesus O. *Neuromyelitis Optica Spectrum Disorder (NMOSD)*. StatPearls Publishing; 2024 Accessed: Feb. 03, 2024. [Online]. Available: http://www.ncbi.nlm.nih.gov/books/NBK572108/
5. Juryńczyk M, Craner M, Palace J. Overlapping CNS inflammatory diseases: differentiating features of NMO and MS. *J Neurol Neurosurg Psychiatry*. 2015;86(1):20-25. doi:10.1136/jnnp-2014-308984
6. Huda S, Whittam D, Bhojak M, et al. Neuromyelitis optica spectrum disorders. *Clin Med*. 2019;19(2):169-176. doi:10.7861/clinmedicine.19-2-169
7. Verkman AS. More than just water channels: unexpected cellular roles of aquaporins. *J Cell Sci*. 2005;118(15):3225-3232. doi:10.1242/jcs.02519
8. Nielsen S, Nagelhus EA, Amiry-Moghaddam M, Bourque C, Agre P, Ottersen OP. Specialized membrane domains for water transport in glial cells: high-resolution Immunogold Cytochemistry of Aquaporin-4 in rat brain. *J Neuro-Oncol*. 1997;17(1):171-180. doi:10.1523/JNEUROSCI.17-01-00171.1997
9. Wingerchuk DM, Lennon VA, Pittock SJ, Lucchinetti CF, Weinshenker BG. Revised diagnostic criteria for neuromyelitis optica. *Neurol Ther*. 2006;66(10):1485-1489. doi:10.1212/01.wnl.0000216139.44259.74
10. Abou Raya A, Raya SA. Neuromyelitis optica spectrum disorders (NMOSD) and systemic lupus erythematosus (SLE): dangerous duo. *Int J Rheum Dis*. 2024;27(1):e14973. doi:10.1111/1756-185X.14973
11. Bukhari W, Barnett MH, Prain K, Broadley SA. Molecular pathogenesis of Neuromyelitis Optica. *Int J Mol Sci*. 2012;13(10):12970-12993. doi:10.3390/ijms131012970
12. Kira J. Autoimmunity in neuromyelitis optica and opticospinal multiple sclerosis: Astrocytopathy as a common denominator in demyelinating disorders. *J Neurol Sci*. 2011;311(1):69-77. doi:10.1016/j.jns.2011.08.043
13. Hinson SR, Roemer SF, Lucchinetti CF, et al. Aquaporin-4-binding autoantibodies in patients with neuromyelitis optica impair glutamate transport by down-regulating EAAT2. *J Exp Med*. 2008;205(11):2473-2481. doi:10.1084/jem.20081241
14. Adawi M, Bisharat B, Bowirrat A. Systemic lupus erythematosus (SLE) complicated by Neuromyelitis Optica (NMO - Devic's disease): clinic-pathological report and review of the literature. *Clin Med Insights Case Rep*. 2014;7:41-47. doi:10.4137/CCRep.S15177
15. Wingerchuk DM, Banwell B, Bennett JL, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurol Ther*. 2015;85(2):177-189. doi:10.1212/WNL.0000000000001729
16. Piga M, Chessa E, Peltz MT, Floris A, Mathieu A, Cauli A. Demyelinating syndrome in SLE encompasses different subtypes: do we need new classification criteria? Pooled results from systematic literature review and monocentric cohort analysis. *Autoimmun Rev*. 2017;16(3):244-252. doi:10.1016/j.autrev.2017.01.011
17. Karathanasis DK, Rapti A, Nezos A, et al. Differentiating central nervous system demyelinating disorders: the role of clinical, laboratory, imaging characteristics and peripheral blood type I interferon activity. *Front Pharmacol*. 2022;13:898049. doi:10.3389/fphar.2022.898049
18. Wingerchuk DM, Weinshenker BG. Neuromyelitis optica: clinical predictors of a relapsing course and survival. *Neurol Ther*. 2003;60(5):848-853. doi:10.1212/01.wnl.0000049912.02954.2c
19. Jarius S, Wildemann B, Paul F. Neuromyelitis optica: clinical features, immunopathogenesis and treatment. *Clin Exp Immunol*. 2014;176(2):149-164. doi:10.1111/cei.12271

How to cite this article: Al Jassem O, Rifi R, Kheir K, Masri A, Eid H. A rare case of systemic lupus erythematosus-associated neuromyelitis optica spectrum disorder with cystic lesions and dual seropositivity for anti-AQP4 and anti-MOG antibodies. *Clin Case Rep*. 2024;12:e9063. doi:10.1002/ccr3.9063