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Recurrent Central Nervous System Demyelination with Longitudinally Extensive Transverse Myelitis in a Young Female: A Case of Neuromyelitis Optica Spectrum Disorder

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Key Message

- Recurrent LETM in young females may indicate neuromyelitis optica spectrum disorder (NMOSD).
- Anti–aquaporin-4 antibody testing is essential for accurate NMOSD diagnosis.
- Early immunosuppressive therapy prevents relapses and long-term neurological disability

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KEYWORDS

Neuromyelitis optica spectrum disorder; Longitudinally extensive transverse myelitis; Aquaporin-4 antibody; Recurrent NMOSD

ABSTRACT:

Neuromyelitis optica spectrum disorder (NMOSD) is an immune-mediated demyelinating disease of the central nervous system that commonly presents with recurrent neurological deficits and longitudinally extensive transverse myelitis (LETM).[1 2] We report a 33-year-old female with recurrent episodes of left-sided limb weakness. Magnetic resonance imaging revealed a small demyelinating lesion in the brain and a longitudinally extensive cervical spinal cord lesion extending from C3 to C7 with contrast enhancement, a characteristic finding in NMOSD.[1] Cerebrospinal fluid analysis showed lymphocytic pleocytosis and strong positivity for anti–aquaporin-4 antibodies, with negative oligoclonal bands, supporting the diagnosis.[2] [3]The patient showed significant clinical improvement following high-dose intravenous methylprednisolone. Early recognition of NMOSD is crucial to prevent relapses and long-term neurological disability.[3 4]

Introduction

Inflammatory demyelinating disorders of the central nervous system (CNS) represent a heterogeneous group of immune-mediated diseases, commonly affecting young adults and often following a relapsing course. These disorders include multiple sclerosis (MS), neuromyelitis optica spectrum disorder (NMOSD), and myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD), each with distinct pathological mechanisms and treatment strategies.

Longitudinally extensive transverse myelitis (LETM), defined as a spinal cord lesion extending over three or more vertebral segments, is a hallmark of NMOSD and helps differentiate it from classical MS. Detection of anti–aquaporin-4 (AQP4) antibodies plays a pivotal role in establishing the diagnosis and guiding long-term immunosuppressive therapy.

We describe a young female with recurrent neurological deficits, characteristic MRI findings, and positive AQP4 antibodies, highlighting the diagnostic challenges and clinical significance of early identification of NMOSD-spectrum disorders.

Case Presentation

A 33-year-old female presented with weakness in the left upper and lower limbs of three weeks duration. The weakness was insidious in onset, progressive and not associated with sensory symptoms initially. She had similar episodes in the past that resolved with medical intervention but had lost follow-up.

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There was no history of fever, headache, vomiting, diplopia, visual disturbances, facial numbness, speech difficulty, bowel or bladder dysfunction, recent infections, trauma, or systemic illness. The patient had a documented history of midbrain and cervical spine demyelination from her medical records and she was diagnosed to have NMOSD. There was no significant family history of neurological or autoimmune disorders. The patient was not a known case of diabetes mellitus, hypertension, or thyroid disease.

On clinical examination, she was conscious and oriented. Her higher mental functions and cranial nerve examination was unremarkable. Tone was mildly increased in both lower limbs, her motor power was 5/5 in both upper limbs and 4/5 in both lower limbs. Her plantar reflex was extensor bilaterally. Her gait was spastic. There was sensory impairment below the level of C2 bilaterally and patchy involvement in the lower limbs. Vitals parameters were stable

Magnetic resonance imaging (MRI) of the brain with contrast revealed a small FLAIR hyperintense lesion in the right fronto-mesial region without contrast enhancement, suggestive of a demyelinating plaque. MRI of the cervical spine showed patchy, longitudinally extensive T2 hyperintensity with cord expansion and post-contrast enhancement extending from C3 to C7 vertebral levels, consistent with active demyelination. Neurophysiological evaluation demonstrated sensory neuropathy involving both upper limbs and a mixed axonal and demyelinating sensory-motor radiculoneuropathy affecting both lower limbs. VEP was within normal limits.

Cerebrospinal fluid analysis revealed a total cell count of 15 cells/mm³ with lymphocytic predominance, normal protein levels (35 mg/dL), and normal glucose concentration (132 mg/dL), with no growth on culture. CSF testing showed strong positivity for anti–aquaporin-4 antibodies, while myelin oligodendrocyte glycoprotein (MOG-IgG) antibodies and oligoclonal bands were absent. Based on the clinical presentation, radiological findings, and immunological profile, a diagnosis of longitudinally extensive transverse myelitis with a recurrent inflammatory demyelinating disorder, suggestive of neuromyelitis optica spectrum disorder, was established. She fulfilled the criteria for NMOSD

Table: 1 CSF Analysis

Parameter	**CSF Finding**
Total cell count	15 cells/cu mm
Lymphocytes	97%
Neutrophils	3%
Protein	35 mg/dL
Glucose	112 mg/dL
Culture and sensitivity	no growth seen
Anti-Aquaporin-4 (NMO-IgG)	Strong positive
Anti-MOG antibody	Negative
Oligoclonal bands	Not detected
	Serum
Oligoclonal bands	Not detected

Treatment

She was treated with intravenous methylprednisolone 1g for 5 days followed by supportive medications and neuropathic pain medications. Gradual clinical improvement was observed during hospitalization. The patient showed significant neurological improvement and was discharged in a stable condition. She was discharged with tapering dose of oral steroids. On follow-up her symptoms improved well and was advised steroid-sparing immunosupressants.

Discussion

This case illustrates a relapsing demyelinating disorder in a young female with clinical and radiological features suggestive of NMOSD. The presence of LETM involving multiple cervical segments, minimal brain involvement, absence of oligoclonal bands, and strong positivity for anti–AQP4 antibodies favors NMOSD over multiple sclerosis.[1,3]

AQP4 antibodies are highly specific for NMOSD and play a pathogenic role by targeting astrocytic water channels, leading to secondary demyelination. Early initiation of high-dose corticosteroids is effective in reducing acute inflammation and improving neurological outcomes, as observed in this patient.[3]

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Accurate differentiation between NMOSD and other demyelinating disorders is essential, as disease-modifying therapies used for MS may be ineffective or harmful in NMOSD.[1],[5] Early diagnosis allows timely initiation of appropriate immunosuppressive therapy to prevent recurrent attacks and long-term disability.[3],[6]

Conclusion

Recurrent neurological deficits associated with longitudinally extensive spinal cord lesions should prompt evaluation for NMOSD. MRI findings and CSF AQP4 antibody testing are critical for early diagnosis. Prompt treatment with high-dose corticosteroids can lead to favorable outcomes and reduce long-term neurological morbidity.

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Conflict of Interest

The authors declare that there are no conflicts of interest related to this work. The authors have no financial or personal relationships that could have inappropriately influenced this study.

References

[1] Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. Neurology. 2015;85(2):177-189.

[2] Lennon VA, Kryzer TJ, Pittock SJ, Verkman AS, Hinson SR. IgG marker of optic-spinal multiple sclerosis binds to the aquaporin-4 water channel. J Exp Med. 2005;202(4):473-477.

[3] Weinshenker BG, Wingerchuk DM. Neuromyelitis spectrum disorders. Mayo Clin Proc. 2017;92(4):663-679.

[4 Jarius S, Wildemann B. The history of neuromyelitis optica. J Neuroinflammation. 2013;10:8.

[5] Pittock SJ, Lucchinetti CF. Neuromyelitis optica and the evolving spectrum of autoimmune aquaporin-4 channelopathies: a decade later. Ann N Y Acad Sci. 2016;1366(1):20-39.

[6] Palace J, Lin DY, Zeng D, Majed M, Elsone L, Hamid S, et al. Outcome prediction models in AQP4-IgG positive neuromyelitis optica spectrum disorders. Mayo Clin Proc. 2019;94(7):1310-1323.