

Multiple sclerosis and Neuromyelitis Optica Spectrum Disorders and the Role of Spinal Cord

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Abstract

Spinal cord involvement is a crucial explanation for disability in patients with MS or neuromyelitis optica spectrum disorders (NMOSDs). Multiple sclerosis and NMOSDs are often distinguished from other disorders that cause myelopathy by results from laboratory and radiological investigations. However, limitations within the sensitivity and specificity of medulla spinalis imaging and poor correlation with disability measures have impeded the understanding of the connection between medulla spinalis involvement and clinical manifestations. Nevertheless, studies of the pathological features of MS and NMOSDs have shown that quantitatively different mechanisms cause differences in clinical course and pattern of accrual of permanent disability in the two disorders. Better understanding of those mechanisms is important to develop more informative clinical measures, electrophysiological methods, fluid biomarkers, and imaging techniques to detect and monitor medulla spinalis involvement within the diagnosis and management of patients with MS or NMOSDs, and as outcome measures in clinical trials.

Keywords: Neuromyelitis optica, Spinal cord, Medulla spinalis.

Introduction

Neuromyelitis optica (NMO) is a severe autoimmune inflammatory demyelinating disease of the central nervous system (CNS). The role of autoimmunity within the etiopathogenesis of NMO was elucidated in 2004 after the invention of aquaporin-4 (AQP4)-immunoglobulin G (IgG), an antibody against the astrocyte water channel. NMO was hence recognized as an autoimmune water channelopathy [1].

The high specificity of AQP4-IgG has permitted recognition of a wider spectrum of clinical and radiologic features associated with NMO. Other sites of CNS involvement not restricted to the optic nerves and medulla spinalis are described in AQP4-IgG-seropositive patients as diencephalic, brainstem, and cerebral lesions. In this seropositive group, coexisting autoimmune disorders have also been recognized, and use of the term NMO spectrum disorder (NMOSD) has been adopted.

This pictorial review summarizes the currently recognized wide spectrum of imaging patterns in NMOSD, which could help radiologists distinguish this disorder from a huge list of differential diagnoses. This distinction, particularly within the early stages, guides proper treatment and possibly avoids lasting disability. Moreover, we describe the history of NMOSD and therefore the evolution of its diagnostic criteria, also because the basis of its pathophysiology and clinical manifestations [2].

The core clinical characteristics of NMOSD are distinguished by the locations of the CNS lesions: optic nerves, medulla spinalis, area postrema, brainstem, diencephalon, and cerebrum. Optic nerve involvement typically manifests as bilateral optic neuritis involving the optic chiasma with severe vision loss. Complete acute medulla spinalis syndrome may be a classic clinical manifestation of medulla spinalis lesions. Intractable nausea, hiccups, and vomiting are associated with area postrema syndrome. Patients with diencephalic involvement may have narcolepsy, anorexia, inappropriate diuresis, hypothermia, and hypersomnia. In brainstem involvement, oculomotor dysfunctions, long tract signs, and ataxia are often seen [3].

The clinical manifestations and prognoses are distinct in seropositive and seronegative NMOSD. AQP4-IgG-seropositive patients usually have more severe clinical attacks, worse outcome, more relapses (81%–91%), higher female-to-male ratio, and more frequent coexisting autoimmune disorders compared with AQP4-IgG-seronegative patients.

References

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