CLINICAL IMAGE

Longitudinal Extensive Transverse Myelitis as a Neuroradiological Clue to Neuromyelitis Optica Spectrum Disorder

Mariane Zampol[1], Thainá Louise Rodrigues[2], Eduardo de Almeida Guimarães Nogueira[3], Andréa de Carvalho Anacleto Ferrari de Castro[4], Fábio César Prosdócimi[5], Joseph Bruno Bidin Brooks[6]

[1]Medical Student, UNIMES-Universidade Metropolitana de Santos, Brazil, [2]Medical Student, UNIMES-Universidade Metropolitana de Santos, Brazil, [3]Department of General Clinics, Hospital Beneficência Portuguesa de Santos, Brazil, [4]Department of Neurology, Universidade Metropolitana de Santos, Brazil, [5]Department of Structure and Function, UNIMES-Universidade Metropolitana de Santos, Brazil, [6]Department of Structure and Function, UNIMES-Universidade Metropolitana de Santos, Brazil

Neuromyelitis Optica Spectum Disorder (NMOSD) is a rare autoimmune and inflammatory disease of the Central Nervous System (CNS) caused by aquaporin-4 (AQP4) water channel autoantibody. The clinical manifestation of this astrocyte channelopathy includes optic neuritis, longitudinally extensive myelitis, and dysfunction of other CNS structures that express AQP4 such as, area postrema, the brainstem, the hypothalamus, the periventricular area, although a wider range of manifestations are recognized as part of NMOSD. More clinical and neuroimaging additional evidence are required for diagnosis when AQP4 serologic testing is unavailable.[1-4] The report of this case was submitted and approved by the ethics committee of Universidade Metropolitana de Santos.

The present case refers to a 44-year-old Caucasian female patient with a 3-week history of progressive weakness of the upper and lower limbs associated with tingling, neck pain and urinary incontinence. Neurological examination revealed proportional tetraparesis (grade 4) associated with

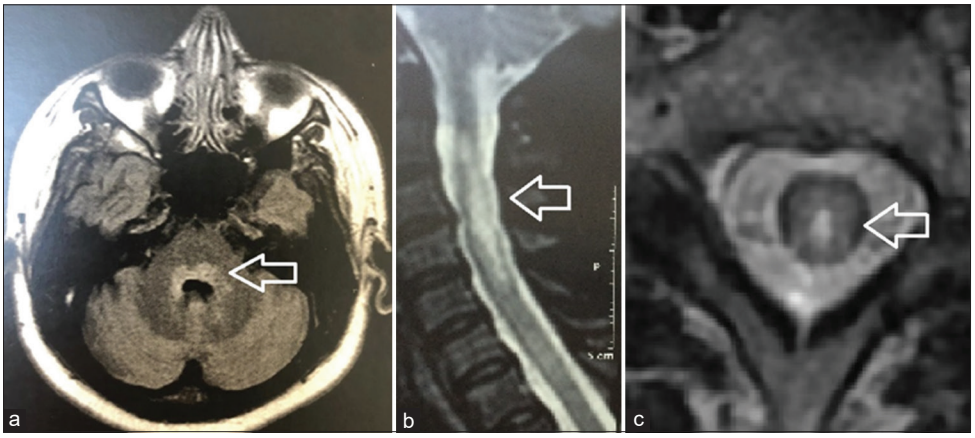

Figure 1: Case report image. (a)- FLAIR Axial Brain magnetic resonance imaging: Hyperintense lesion around the 4th ventricule (white arrow); (b)-T2 Sagittal cervical spinal cord; and (c)-T2 Axial cervical spinal cord: C2, C3, C4, and C5 central hyperintense lesion-longitudinal extensive transverse myelitis (white arrow)

Address for correspondence:
Joseph Bruno Bidin Brooks, Universidade Metropolitana de Santos-UNIMES Avenida Gen. Francisco Glicério 8 Santos, São Paulo, 11045-002, Brazil. Phone/Fax: +55-13-3228-3400.

upper motor neuron dysfunction, superficial, and deep hypoaesthesia with a sensitive level in C3 and Lhermitte's sign. Cranial and cervical spinal cord magnetic resonance imaging showed the presence of demyelinating lesion around the 4th ventricule and a central and longitudinal extensive lesion in the cervical spinal cord [Figure 1]. Laboratory examination showed AQP4 antibody positivity by cell block assay method. Cerebrospinal fluid examination showed 10 cells, all of them lymphomonocytes, and protein of 46 mg/dL (lumbar puncture). All other tests on spinal fluid were normal. Meanwhile, the possible clinical diagnosis of NMOSD were postulated and started treatment with 1 gram of methylprednisolone intravenously and for a period of 5 days with satisfactory results. Therapeutic follow-up was instituted with immunosuppressive therapy, including azathioprine and prednisone and paraesthesia treatment with carbamazepine with partial improvement of symptoms. This case report alerts to the possibility of NMOSD in the differential diagnosis of patients with longitudinal extensive transverse myelitis. NMOSD clinical-radiologic correlation attention may help physicians make early and correct diagnoses and appropriate treatment.

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