A CASE OF TUMEFACTIVE MULTIPLE SCLEROSIS MIMICKING NMOSD WITH OPTIMAL RESPONSE TO OCRELIZUMAB: MRI PITFALLS

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Objectives: We describe a case of aggressive tumefactive multiple sclerosis (**MS**) with some MRI features **resembling** neuromyelitis optica spectrum disorder (**NMOSD**), to highlight the importance of MRI pitfalls in the differential diagnosis of demyelinating central nervous system diseases to start early high-efficacy treatment.

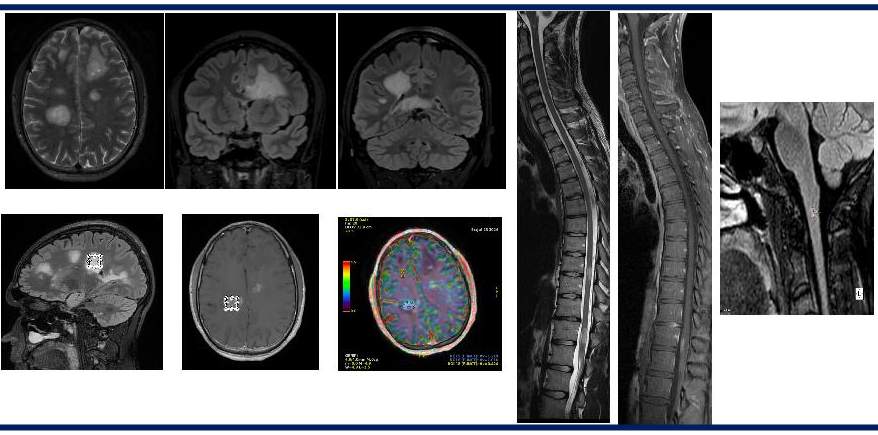
Materials: Clinical, MRI and laboratory data were collected from a previously healthy **24-year-old** man who presented with acute multifocal neurological symptoms (diplopia, ataxia, tetraparesis).

Methods: Brain and spinal MRI on a 3 Tesla scanner was performed at admission and during follow-up, with typical **MRI** MS protocol. Cerebrospinal fluid (**CSF**) oligoclonal bands (**OCB**) and anti-aquaporin4 (**AQP4**) and myelinoligodendrocyteglycoprotein (**MOG**) antibodies were analyzed.

Results: Baseline MRI revealed **≥ 20 supra and infratentorial lesions,** some of them with tumefactive features (the largest measuring 30 mm). Key findings included a **splenial lesion** of the corpus callosum with open-ring enhancement, **concentric Baló-like rings**, and several periventricular lesions. Additional plaques involved the midbrain and the **periependymal white** matter of the fourth ventricle. Cervical and thoracic MRI scans identified three short, non- contiguous cord lesions at C4, D9 and D12. CSF showed **type3 OCB** with normal protein; serum and **CSF anti-AQP4 and anti-MOG antibodies were negative**.

Despite five days of high dose pulse therapy with steroids, the patient developed new sensory–motor deficits (EDSS raised from 2.5 up to 3.5) and new enhancing lesions. Rescue therapy with five PEX sessions was ineffective.

Escalation to **ocrelizumab, preceded by supplementation with IVIg**, prevented new lesion formation and led to substantial neurological recovery (EDSS 1.5).



Discussion: Lesion distribution involved **some typical NMOSD location (splenium and periependyma**), while open-ring enhancement, Baló-like concentric rings, confluent periventricular plaques and short spinal cords lesions are characteristic of MS.

The presence of **OCB with negative anti- AQP4/MOG antibodies** also **suggested MS diagnosis**. In our case, anti-CD20 therapy allowed a rapid disease control and clinical improvement despite poor response to steroids and PEX, as reported in several case-reports in literature.

Conclusions: Comprehensive brain and spinal **MRI combined with laboratory analysis** is essential for **distinguishing tumefactive MS from NMOSD** in young adults with rapidly progressive deficits, allowing early high-efficacy treatment.

Anti-CD20 therapy should be started **early** as rescue therapy **in case of poor response to steroids or plasma-exchange.**

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