Tumefactive multiple sclerosis (MS) may pose a challenging differential diagnosis for its rather exuberant radiological presentation. However, there are some imaging clues to help us unveil this inflammatory entity.

We present a case of an 18-year-old girl with a depressive disorder presented with acute headache and sensorimotor complaints, and a subacute depressive disorder for two months. At examination, she had a slight attention deficit, an anosodiaphoria regarding her deficits, a grade 4 right hemiparesis with an unsteady gait, a generalized hyperreflexia with pathological pyramidal signs and a predominantly distal thermo-algic hypoesthesia, hypopalesthesia and hypokinesia of all limbs, with no sensory level. No history of recent infection nor vaccination.

Neuroimaging showed multiple tumefactive T2-FLAIR hyperintense brain lesions predominantly affecting the periventricular white matter, several nodular and ring-enhancing lesions and additional spinal cord lesions (Fig. 1). At first glance, this clinical and radiological picture compelled us to conduct a careful differential diagnosis, including inflammatory and demyelinating, infectious and neoplastic disease.

In this case, open-ring enhancing lesions were indicative of demyelinating disease. Importantly, different inflammatory demyelinating entities should be considered, such as multiple sclerosis (MS), acute disseminated encephalomyelitis (ADEM), neuromyelitis optica spectrum disorder (NMOSD), myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) and even Baló’s concentric sclerosis by the resemblance of its characteristic features with some heterogeneous lesions of this case. However, lesion distribution, particularly cortical/juxta-cortical and in the calloso-septal interface, and the SWI central-vein were very suggestive of multiple sclerosis.1,2 Moreover, initial neuroimaging met McDonald criteria

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**Figure 1.** Brain MRI
(A, B, C) Axial T2-FLAIR showing multiple hyperintense tumefactive lesions predominantly affecting the periventricular white matter with additional cortical/juxta-cortical lesions (arrows). (D) Sagittal T2-FLAIR showing lesions in the callosal-septal interface (arrow) and a tumefactive lesion in the cervical spinal cord (dotted arrow). (E) Axial T1 contrast-enhanced demonstrating open-ring enhancement. (F) Axial SWI demonstrating the central vein sign.

**Figure 2.** Post-immunotherapy brain MRI
Post-immunotherapy follow-up brain MRI performed 2 weeks after presentation. (A, B) Axial and (C) sagittal T2-FLAIR showing marked reduction of the perilesional edema with resolution of the tumefactive appearance and cavitation of some of the lesions (arrows). There is also near-complete resolution of the tumefactive appearance of the spinal cord lesion.
for dissemination in time and space, elucidating that a monophasic disease was less likely.

CSF oligoclonal bands were present. AQP4 and MOG antibodies were negative. Methylprednisolone caused unequivocal remission of both psychiatric symptomatology and neurological deficits. Imaging improvement was found on the follow-up brain magnetic resonance imaging (MRI) performed 2 weeks after presentation, with marked reduction of the perilesional edema, near-complete resolution of the tumefactive appearance of the lesions and cavitation of some of the lesions (Fig. 2).

These suggestive neuroimaging features proved to be important supportive findings for a prompt diagnosis within a broad spectrum of inflammatory diseases, as well as other etiologies to consider, thus allowing proper treatment in useful time.

Even under atypical presentations, T2-FLAIR and SWI sequences provide us accurate neuroimaging clues to diagnose a tumefactive MS.

**Contributorship Statement / Declaração de Contribuição**

MDS: Conception, writing and final approval.
BC: Images preparation, writing and final approval.
MD: Conception and final approval.
CC: Critical review with intellectual contribution and final approval.

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**References / Referências**