IMAGEM EM NEUROLOGIA/IMAGE IN NEUROLOGY

Bilateral Internuclear Ophthalmoplegia: Multiple Sclerosis rather Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease (MOGAD)?

Oftalmoparésia Internuclear Bilateral: Esclerose Múltipla ou Doença Associada aos Anticorpos Anti-MOG?

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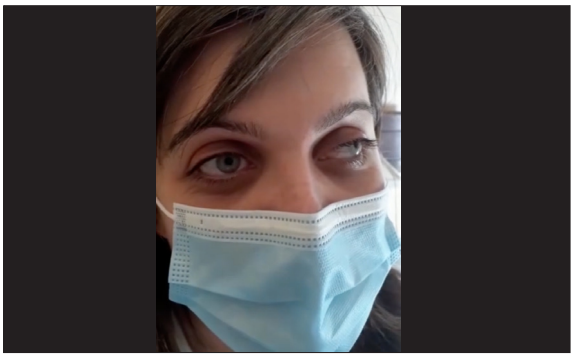
Abstract

Increasing knowledge about neuro-inflammatory conditions as lead to new challenges in establishing a diagnosis. Clinical distinctive features might help. We present a 34-year-old patient with bilateral internuclear ophthalmoplegia (bINO). Investigation was compatible with demyelinating central nervous system (CNS) disease. Low-positive IgG myelin oligodendrocyte glycoprotein (MOG) antibodies were found. Rituximab was started and the patient has remained without further clinical or magnetic resonance imaging (MRI) activity. Acknowledging that bINO is a well-known manifestation of multiple sclerosis (MS) but rarely reported among MOGAD patients its occurrence might help distinguish MS from MOGAD in an apparent overlap setting, favoring MS.

Resumo

O aumento do conhecimento sobre as doenças neuro-inflamatórias conduziu a novos desafios no estabelecimento de diagnóstico. Certas caraterísticas clínicas distintivas podem ajudar. Apresentamos uma doente de 34 anos com uma oftalmoplegia internuclear bilateral (OINb). A investigação foi compatível com doença desmielinizante do SNC. Foram detetados anticorpos IgG da glicoproteína da mielina oligodendrócitos (MOG) fracamente positivos. Foi iniciado rituximab e a doente manteve-se sem mais atividade clínica ou imagiológica. Reconhecendo que a OINb é uma manifestação bem conhecida da esclerose múltipla (EM), mas raramente relatada em doentes com MOGAD, a sua existência pode ajudar a distinguir a EM da MOGAD num contexto de aparente sobreposição, favorecendo a EM.

A healthy 34-year-old woman presented with isolated diplopia for the last two days. She mentioned an episode that occurred eight years earlier, which she characterized as "blurred vision over a week", fully recovered after steroids. She also stated that, at that time, had an ophthalmology consultation and underwent a brain magnetic resonance imaging (MRI), with "normal" results (images not available), no further workup was done, and she was discharged. The patient remained asymptomatic since then. At present, neurological examination (see **Video**) disclosed isolated bilateral internuclear ophthalmoplegia (bINO). Neuro-axis MRI showed T2/FLAIR, non-enhancing multiple lesions involving the right middle cerebellar peduncle, supratentorial periventricular white matter and a cervical spinal cord-enhancing lesion



Video 1. Neurological examination disclosing bilateral internuclear ophtalmoplegia. (see the video)

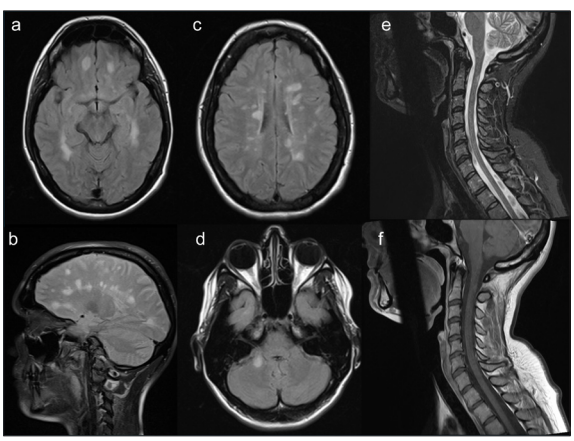


Figure 1. Brain MRI (a-d) showing multiple T2/FLAIR hyperintense lesions: periventricular, subcortical (a,c) and pericalosal (b) as well as in the right middle cerebellar peduncle (d). None contrast enhancing. Spinal cord MRI (e-f) disclosed posterior hyperintense T2/STIR lesion at C2 level (arrow) with discret gadolium enhancement (*). .

(**Fig. 1**). Cerebrospinal fluid (CSF) showed mildly elevated WBCs at 23 (normal <5/μL; 100% mononuclear), normal protein and glucose and negative bacteriological culture. Low-positive serum IgG myelin oligodendrocyte glycoprotein (MOG) antibodies were found by fixed cell-based assay (and confirmed 6 weeks later), serum AQP4-IgG turned out to be negative. Positive CSF-oligoclonal bands were identified. The patient recovered after methylprednisolone (1 g/5 days). No better explanation was found for this case´s diagnosis other than relapsing-remitting multiple sclerosis (MS), despite the identification of weak positivity for anti-MOG antibodies. Notwithstanding, safeguarding an eventual dismal evolution demanding future reconsideration of MOGAD's differential diagnosis, the patient started on rituximab (1000 mg 6/6 months). She remains asymptomatic, adding no further T2/FLAIR or enhancing brain or spinal cord lesions after 2 years of follow-up. Acknowledging that bINO is a well-known manifestation of MS[1] but rarely reported among MOGAD patients,[2] herein we highlight that its occurrence might help distinguish MS from MOGAD in an "apparent" overlap setting, favoring MS. ■

Contributorship Statement / Declaração de Contribuição
AJM: Design, conception and writing of the article.
AM: Critical review.
JPG: Review and final approval.
All authors have read and approved the manuscript.

AJM: Conceção e redação do artigo.
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JPG: Revisão e aprovação finais.
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