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Anti-MOG syndrome: a road to be paved

Síndrome anti-MOG: um caminho a ser pavimentado

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Myelin oligodendrocyte glycoprotein (MOG) is a protein expressed only on the outermost lamellae of the myelin sheath and on the surface of oligodendrocytes in the central nervous system (CNS). MOG represents less than 0.05% of total myelin proteins but despite its meager concentration, it is believed to be involved in important functions, such as being a surface marker of the mature oligodendrocyte and participating in the interactions between myelin and its players[1].

The better structural characterization of MOG as well as its location rapidly made it an antigen used successfully in the induction of experimental models of demyelination such as experimental autoimmune encephalomyelitis. Thereafter, the presence of anti-MOG antibodies was investigated in those diseases that represented the clinical prototype of demyelinating conditions: acute disseminated encephalomyelitis (ADEM) in children and multiple sclerosis (MS) in adults[2]. Although anti-MOG antibodies have been found in such prototypical situations, probably for laboratory methodological reasons, the clinical utility of antibody detection as a reliable biomarker was abandoned[3].

The development of a more specific and sensitive laboratory methodology (cell based assay, CBA) allowed to revisit the anti-MOG antibody in demyelinating diseases and to establish it as a new biomarker. To date, once the presence of the antibody in the serum has been identified, it seems reasonable to include the following entities under the anti-MOG spectrum: recurrent or bilateral optic neuritis (ON), ADEM, neuromyelitis optica spectrum disorders anti-aquaporin-4 negative and longitudinal extensive transverse myelitis[4]. Recently, Ogawa et al. described four young men with unilateral encephalitis and epileptic seizures of benign evolution and response to steroids in which the anti-MOG antibody was the only one detected.[5] However, we must keep in mind that it is a knowledge still under construction, a road to be paved, particularly regarding the recognition of the whole spectrum, its long-term behavior and the best therapeutic approach.

In this issue of Arquivos de Neuropsiquiatria, Costa et al. describe six patients (four men) with optic neuritis (five monophasic, four bilateral) and serum anti-MOG antibody in very high titers except in one case[6]. Due to the rarity of the disease, the number of anti-MOG positive patients analyzed is quite reasonable. However, the authors included a positive anti-MOG patient (patient 2) whose clinical presentation was neuritis and myelitis, which is beyond the scope of the article. Regarding the data presented, Costa et al. could have added more information regarding MRI. For example, no mention was made of the lenght of optic neuritis, which is usually greater than that found in patients with MS and even with NMOSD aquaporin 4 positive, although it is not usual to reach the optic chiasm. Another data that could be investigated would be the presence of intraorbital fat tissue contrast enhancement, a characteristic recently described and apparently quite specific in the context of anti-MOG optic neuritis[7].

The work of Costa et al has its merit in being the first Brazilian publication of a series of cases of anti-MOG positive patients with optic neuritis. In addition, it reinforces our obligation to include in the differential diagnosis of idiopathic optic neuritis the possibility of anti-MOG spectrum. We should expect the authors to publish the prospective follow-up data so that we can better understand the natural history, prognosis and better long-term management.

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