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Response relating to the article *Primary progressive multiple sclerosis overlapping with anti-GAD and anti-Hu antibodies positive neurological syndromes*

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In the comment on our article from Dr. Rzepiński, the relevant demonstration of intrathecal antibody IgG synthesis in our patients for an MS diagnosis proved by at least two oligoclonal bands unique to CSF is questioned [1, 2].

In the position paper *Diagnosis of multiple sclerosis: 2017 revisions of the Mc Donald criteria,* there is a statement that two or more CSF specific oligoclonal bands more reliably indicate intrathecal antibody synthesis than do other tests, citing as the reference the article by Freedman et al. published in 2005 [3]. In their review article, Deisenhammer et al.mention that increased production of intrathecal immunoglobulins is the hallmark of typical CSF changes in MS [4]. Such intrathecal IgG synthesis can only be assessed if compared to serum. Oligoclonal bands in CSF can only be intrathecally synthesised if bands selectively occur in CSF, or if there are more bands in CSF than in serum, instances referred to as patterns 2 and 3 by Freedman et al.

Depending on the IgG separation method, serum bands should be outnumbered by 1–3 bands in the CSF. In our cases, the serum bands were outnumbered by five IgG bands in the CSF, clearly demonstrating specific intrathecal IgG synthesis as required for an MS diagnosis. In the 2020 article by Lopes et al., the presence of OCB in both biological fluids, but with higher numbers in the CSF than in the serum, indicates intrathecal IgG synthesis [5].

Based on these statements, we postulate that intrathecal IgG synthesis was proved in both our patients. Additionally, MRZ reaction and kFLC index were positive in the first patient and there was mild pleocytosis in the second patient, emphasising the presence of inflammation in the CNS. MRI scans of the spinal cord were carried out in the patients but without any significant focal hyperintense lesions. As there is no specific MRI pattern for both anti GAD and anti Hu syndromes, and MRI brain scans indicated MS, it is unlikely that such findings could be explained solely by antibody-associated syndromes. As far as the possibility of the occurrence of unspecific white matter lesions in the second patient is concerned, in the elderly we cannot exclude that some of them could be of this origin, but the second patient also fullfilled MS MRI criteria.

We are aware that there is no specific test for an MS diagnosis despite continuously improving MS diagnostic criteria, and we wish to add some new aspects into this differential diagnostic field.

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