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TO THE EDITORS

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'Primary progressive multiple sclerosis overlapping with anti-GAD and anti-Hu antibodies positive neurological syndromes' — clinical considerations

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To the Editors

I was interested to read the case reports published in the *Polish Journal of Neurology and Neurosurgery* entitled: 'Primary progressive multiple sclerosis overlapping with anti-GAD and anti-Hu antibodies positive neurological syndromes' by Štourač et al. [1]. The authors presented a description of two patients with overlapping progressive neurological deficits classified as primary progressive multiple sclerosis (PPMS), with the presence of anti-GAD antibodies in the first case, and anti-Hu antibodies in the second. In both patients, paraneoplastic aetiology was excluded based on additional diagnostic tests and several years of clinical observation. Interestingly, anti-neuronal antibodies persisted in serum and cerebrospinal fluid (CSF) despite the passage of time and the treatment used (steroids and/or immunomodulatory therapies). Importantly, oligoclonal IgG bands (OCBs) were also detected in serum and CSF. The authors stated that both patients fulfilled the 2017 McDonald criteria for PPMS by confirmed disability progression, T2-hyperintense lesions in typical locations in MRI brain scans, and positive OCBs in CSF [1, 2]. However, the 2017 McDonald criteria require demonstration of two or more CSF-specific OCBs for a PPMS diagnosis [2]. Therefore, both the presence of at least two OCBs in CSF and the absence of bands in serum are essential to reveal intrathecal antibody synthesis [3]. When OCBs are not unique to CSF, two or more T2-hyperintense lesions in the spinal cord should be demonstrated to establish the diagnosis of PPMS. Furthermore, the 2017 Panel membership recommends MS diagnosis when there is no better explanation for the clinical symptoms [2]. The above-mentioned issues raise some clinical points for consideration. Firstly, it should be clarified whether the presented patients have ever had MRI scans of the spinal cord, and if so what was found? Obtaining this data is particularly important to validate a PPMS diagnosis. Secondly, did the authors consider the presence of white matter hyperintensities as a consequence of neurological syndromes associated with anti-neuronal antibodies alone? Thirdly, from a clinical point of view I am curious whether, in Case 2, age-related factors could not have contributed to the formation of T2-hyperintense lesions in the brain, since no such lesions were found in the first MRI scans [4].

Nevertheless, the demonstration of antibodies potentially associated with paraneoplastic syndromes in PPMS patients in the era of growing availability of therapies that can increase the risk of neoplasms is highly important [4, 5]. Therefore, I would like to thank the authors for sharing their observations in the *Polish Journal of Neurology and Neurosurgery*.

Conflicts of interest: *None.*

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