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C. Montejo, M. Vicente, A. Sánchez, A. Renú*

*Servicio de Neurología, Hospital Clínic de Barcelona, Barcelona, Spain*

* Corresponding author.
*E-mail address:* arenu@clinic.ub.es (A. Renú).

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# Progressive multifocal leukoencephalopathy in an immunocompetent patient☆

## Leucoencefalopatía multifocal progresiva en un paciente inmunocompetente

*Dear Editor:*

I read with great interest the study by Muiño et al.,[1] describing the case of a 72-year-old patient who presented progressive multifocal leukoencephalopathy (PML) with no underlying immune disorder. The authors report 7 cases of PML in immunocompetent individuals. I would like to comment on another case reported by myself and colleagues.[2] Our patient was a 45-year-old woman presenting weakness and numbness of the right hand, with onset in March 2000. The initial examination, 2 months after symptom onset, revealed postural tremor, rigidity, and hyperreflexia of the right arm. This motor deficit progressed over the following months, with myoclonic jerks developing. In September 2000, she presented right hemiparesis with continuous myoclonic jerks, mainly affecting the flexor and extensor muscles of the right hand. From October 2000, she presented the same symptoms on the left side. In the final stage, the patient developed decorticate posture, although she remained lucid until her death in June 2001. The patient did not respond to antiepileptic drugs. Analytical studies, including an immunological profile, tumour markers, and serology studies, yielded normal or negative results. Consecutive EEG and EMG recordings detected signs characteristic of epilepsia partialis continua (EPC) and progressive slowing of background activity. The baseline brain MRI study (May 2000), especially the FLAIR sequences, showed hyperintensities in the U-shaped fibres of the left precentral cortex. Consecutive follow-up MRI scans showed lesion progression and the appearance of similar contralateral alterations. A stereotactic brain biopsy was not diagnostic. A complete autopsy revealed that the patient died due to bronchopneumonia and massive gastrointestinal bleeding, with no signs of neoplasm. The brain showed microscopic lesions characteristic of PML, with demyelination, predominantly in the motor and parietal areas and corticospinal tracts. The cerebral cortex, basal ganglia, brainstem, and cerebellum were intact. In view of these findings, we suggested that a ''motor cortex isolation syndrome'' may be the cause of the EPC.

The title of our article was intended to emphasise the semiological aspects, that is, it made no reference to the state of the immune system; furthermore, we did not include ''immunocompetence'' as keyword. This explains why a literature search on Pubmed, combining the terms ''progressive multifocal leukoencephalopathy'' and ''immunocompetence,'' does not return our article. In any case, I do agree with the authors that the presence of PML in an immunocompetent individual is exceptional.

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J. Berciano

*Servicio de Neurología, Hospital Universitario Marqués de Valdecilla (IDIVAL), Universidad de Cantabria, Centro de Investigación Biomédica en Red de Enfermedades Neurodegenerativas (CIBERNED), Santander, Cantabria, Spain*

*E-mail address:* jaberciano@humv.es

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