Dear Editor:

Isolated demyelinating lesion and non-tumoral demyelinating aspects: a case of a 26-year-old woman with inflammatory of the central nervous system.


References


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Available online 10/16/2018 20:005

https://doi.org/10.1016/j.nrleng.2018.01.005

Fig. 1. (A, B) Hemorrhagic and non-hemorrhagic temporal lobe encephaloceles. (A) Sagittal T2-weighted MRI showing high-field isointense signal of the lesion compatible with an intracranial hypomagnesemia. (B) Axial T2-weighted MRI showing high-field isointense signal of the lesion compatible with an intracranial hypomagnesemia. (C) Axial T2-weighted MRI showing high-field isointense signal of the lesion compatible with an intracranial hypomagnesemia.
Inflammatory pseudotumour is a rare condition (0.3 cases/100,000 person-years). Differential diagnosis includes infection, neoplasia, and vascular disorders. Certain clinical and radiological characteristics support diagnosis, whereas biopsy is used only for atypical or refractory lesions. Several cases have been reported of patients who showed no response to corticosteroids but were responsive to other immunosuppressive treatments, with IV Ig, plasmapheresis, rituximab, or cyclophosphamide. Ours is the first case in the literature of rituximab being used after IV Ig to treat a corticosteroid-refractory pseudotumoural lesion, and the seventh case of rituximab being used after plasmapheresis.

The lack of treatment response is therefore considered to go beyond the absence of response to corticosteroids, in clinical practice, it is difficult to decide when to perform a brain biopsy in cases of refractory pseudotumoural lesions. Furthermore, biopsy may show inflammation and demyelination, although it rarely enables us to differentiate between onset of multiple sclerosis or neuromyelitis optica, and focal forms of monophasic encephalitis that will not progress to a chronic demyelinating disease. Close, prolonged clinical follow-up is therefore essential.

We established diagnosis of focal, monophasic autoimmune encephalitis, given the isolated pseudotumoural lesion of inflammatory aetiology (which was confirmed by an anatomical pathology study), with negative oligoclonal bands, of monophasic course (to date) and responsive to treatment with rituximab (supporting immune-mediated pathogenesis with B-cell involvement).

In conclusion, some autoimmune lesions cannot be identified with a known serological marker and represent a diagnostic challenge. Focal, monophasic autoimmune encephalitis may explain this case and other cases of iso-
lated inflammatory pseudotumoural lesions in patients with no previous diagnosis of chronic demyelinating disease, showing a monophasic course during follow-up.

Acknowledgements

We would like to thank Dr Laura Zaldumbide Dueñas (anatomical pathology department, Hospital Universitario de Cruces), who performed the anatomical pathology study of the brain biopsy sample and provided the corresponding images.

References


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https://doi.org/10.1016/j.nrleng.2018.01.006
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