Patient is a 44-year-old woman with a 28-year history of epilepsy, mainly focal, with monthly generalisations and a pseudotumoral lesion of the right hemisphere shown by MRI scans (Fig. 1). A review of her medical history revealed a HSP, positive for HIV, HCV, and syphilis, no history of malignancy, and positive serological tests for VZV and OMA, positive for VZV, and negative for HIV, HCV, and syphilis. She was also positive for QuantiFERON-OMA and negative for PPD. The patient showed increased ESR, CRP, and ANA antibodies, including anti-neuromyelitis oligoclonal antibodies, and had a negative oligoclonal banding test in cerebrospinal fluid. Haematology tests showed normal counts, while her platelet count was reduced.

We present the case of a 44-year-old woman with pseudotumoral lesions simulating central nervous system demyelinating disease.

To follow remain contactatal:
Inflammatory pseudotumour is a rare condition (0.3 cases/100 000 person-years\textsuperscript{5,6}). Differential diagnosis includes infection, neoplasia, and vascular disorders.\textsuperscript{1,3} Certain clinical and radiological characteristics support diagnosis,\textsuperscript{1,2,6} whereas biopsy is used only for atypical or refractory lesions.\textsuperscript{6} Several cases have been reported of patients who showed no response to corticosteroids but were responsive to other immunosuppressive treatments,\textsuperscript{5–7} with IVIg, plasmapheresis,\textsuperscript{8} rituximab,\textsuperscript{7,9–12} or cyclophosphamide.\textsuperscript{7} Ours is the first case in the literature of rituximab being used after IVIg 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,\textsuperscript{1,4} 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,\textsuperscript{1,3,13} 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.

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References


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