TUMEFACTIVE BRAIN LESION WITH RAPID CAVITY FORMATION ASSOCIATED WITH ANTI-AQUAPORIN-4 ANTIBODY

A 54-year-old woman with no medical history presented with left hemiparesis and hemianopsia. Brain MRI showed an extensive lesion without enhancement in the right parietal lobe, followed by rapid cavity formation (figure 1). Brain biopsy performed 55 days after disease onset revealed decreased glial fibrillary acidic protein and aquaporin-4 (AQP4) immunoreactivity, though we could not show the activated complement due to the paucity of the sample (figure 2).

Brain biopsy results revealed that the patient was positive for anti-AQP4 antibody. She received methylprednisolone pulse therapy, followed by plasmapheresis. Progression was inhibited. Based on anti-AQP4 antibody-seropositive cases, the concept of neuromyelitis optica spectrum disorders is becoming broader.1 These disorders should be considered in differential diagnoses when tumefactive brain lesions are observed.2 Our case highlights the importance of examining AQP4 immunoglobulin G, because it could result in an early diagnosis without performing biopsy.3

Noncontrast fluid-attenuated inversion recovery images on days 2 (A), 8 (B), and 35 (C). Note extensive cavity formation on day 35. The lesion shows high intensity on an apparent diffusion coefficient map on day 2 (D). Enhanced T1-weighted images on days 3 (E) and 35 (F) show no enhancement.
(A) Double staining with anti-aquaporin-4 (AQP4) antibody (dark green) and Luxol fast blue (blue) is shown. Loss of AQP4 immunoreactivity with myelin pallor is shown (surrounded by arrows). (B) Staining with glial fibrillary acidic protein (GFAP) (brown) is shown. Loss of GFAP immunoreactivity is observed in the corresponding area shown in A (surrounded by arrows). Scale bar: 50 μm.
Tumefactive brain lesion with rapid cavity formation associated with anti-aquaporin-4 antibody
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Neurol Neuroimmunol Neuroinflamm 2016;3;
DOI 10.1212/NX1.0000000000000230

This information is current as of April 14, 2016

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