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【 PICTURES IN CLINICAL MEDICINE 】

Transient Diffusion-weighted Imaging Hyperintensity of the Cerebellar Cortex in Paraneoplastic Cerebellar Degeneration

Yasumasa Hashimoto[1-3], Kenichi Komatsu[1], Tomokazu Nakagawa[1] and Sadayuki Matsumoto[1]

Key words: MRI, DWI, FLAIR, Anti-Yo antibody-positive paraneoplastic cerebellar degeneration, cerebellar atrophy

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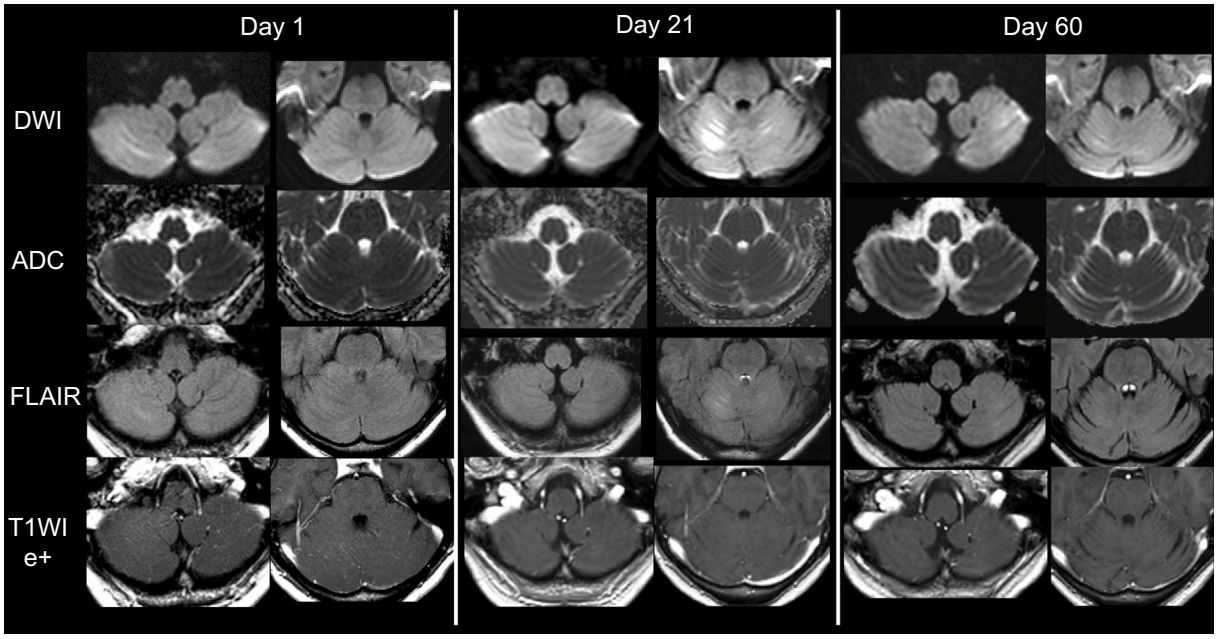

Picture.

A-53-year-old woman presented with a 2-week history of dysarthria and gait instability. On admission (day 1), a neurological examination revealed gaze-evoked nystagmus, mild dysarthria and severe truncal ataxia. Slight diffusion-weighted imaging (DWI) and fluid-attenuated inversion recovery (FLAIR) hyperintensity in the right cerebellar cortex were suspected on MRI (Picture). The cerebrospinal fluid (CSF) cell count was 20/μL, the protein level was 47 mg/dL, and the cytology was negative. Anti-Yo antibody-positive paraneoplastic cerebellar degeneration (PCD) with presumed ovarian cancer was diagnosed after a thorough systemic work-up. Cerebellar symptoms progressed despite steroid pulse therapy, and MRI signal change moved to the bilateral upper portions of the cerebellum on day 21. The apparent diffusion coefficient (ADC) of the lesion was decreased in this second MRI (Picture). Although CSF findings normalized by day 29, the cerebellar symptoms progressed until paraaortic lymphadenectomy and plasma ex-

[1]Department of Neurology, Kitano Hospital, The Tazuke Kofukai Medical Research Institute, Japan, [2]Department of Molecular Therapy, National Institute of Neuroscience, National Center of Neurology and Psychiatry, Japan and [3]Department of Neurology, Kansai Medical University, Japan

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Correspondence to Dr. Kenichi Komatsu, kkomatsu@kuhp.kyoto-u.ac.jp

change were performed on day 38 and from day 41 to day 55, respectively. On day 60, the MRI signal change resolved, but cerebellar atrophy was evident. No gadolinium enhancement was seen during the disease course (Picture).

Transient hyperintensity on DWI and FLAIR preceding cerebellar atrophy were noteworthy in this case. Most previous reports have described only atrophic changes, and few have described FLAIR hyperintensity. DWI hyperintensity of the cerebellar cortex has been reported in association with infectious cerebellitis (1) and heat stroke (2), but never with PCD. Increased cellularity due to inflammatory cell infiltration has been suggested to be the cause of DWI hyperintensity in infectious cerebellitis (1), and cytotoxic edema has been discussed in relation to heat stroke (2). Inflammatory cell infiltration has been described in the early phase of PCD, while Purkinje cell loss without inflammation is noted in the late phase of the disease (3). The MRI signal change in our case may represent cytotoxic edema of Purkinje cells or increased cellularity due to inflammatory cell infiltration in the acute phase of the disease.

The authors state that they have no Conflict of Interest (COI).

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