LETTERS TO THE EDITOR

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Clinically mild encephalitis/encephalopathy with a reversible splenial lesion associated with aseptic meningoencephalitis

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A 27-year-old woman was admitted to our hospital with fever, headache, and gait and speech disturbances. Seven days before admission to our hospital, she was admitted to another hospital with the same symptoms; magnetic resonance imaging (MRI) conducted there on the third day of admission revealed high-intensity signals in the splenium of the corpus callosum on T2-weighted and diffusion-weighted images (Fig. 1a). Except for the splenial lesion, there were no other abnormal lesions in cerebrum and cerebellum. On admission to our hospital, she had difficulty in walking straight and in articulation. She had no family history of neurological disorders and did not report any exposure to toxins such as alcohol or drugs. Physical examination revealed body temperature of 37.0 ºC, blood pressure of 92/59 mm Hg, and pulse rate of 109 per minute. There were neither skin rashes nor superficial lymph node swellings. There was no stiffness in the neck; Kernig's sign was also negative. She had no nystagmus. Ataxia of bilateral lower extremities was observed. Motor power and sensory examinations were normal. Her deep-tendon reflexes were normoactive. Routine blood tests including complete blood count, biochemical analysis, estimation of C-reactive protein, electrolytes, prothrombin time, and partial thromboplastin time were within normal limits, similar to the reports from the previous hospital. The anti-nuclear antibody test and other tests for autoimmune antibodies were negative. Myeloperoxidase and proteinase 3-antineutrophil cytoplasmic antibodies could not be detected. Antibodies against herpes simplex virus, measles virus, cytomegalovirus, enterovirus 71, human herpes virus 6, and Epstein-Barr virus were also not detected. Lumbar puncture revealed pleocytosis of 260 cells/μL (mononuclear cells, 238 cells/μL; polymorphonuclear cells, 22 cells/μL), an increase in protein level (121 mg/dL), and a slight decrease in glucose level (34 mg/dL). Antibodies against herpes simplex virus, mumps virus, and enterovirus 71 were absent in the cerebrospinal fluid. Culture of cerebrospinal fluid sample was also negative for fungi, tubercle bacilli, and other bacteria. Fluorine-18 2-deoxy-2-fluoro-D-glucose positron emission tomography (^{18}F-FDG PET) carried out on the first day of admission to our hospital revealed increased glucose metabolism in the cerebellum (Fig. 2a) and the upper spinal cord (Fig. 2b). However, there were no abnormal lesions in the splenium (Fig. 2a). Finally, the patient was diagnosed clinically with mild encephalitis/encephalopathy with a reversible splenial lesion (MERS) associated with aseptic meningoencephalitis, the neurological lesions of which were suspected to be in the cerebellum and the upper spinal cord. Fever and headache improved considerably with the intake of non-steroidal anti-inflammatory drugs; however, gait and speech disturbances did not improve. Therefore, on the second day of admission to our hospital, she was treated with methylprednisolone at 1 g/day for 3 days and subsequently prednisolone (PSL) was prescribed at 20 mg/day for 7 days. MRI diffusion-weighted images obtained 8 days after admission to our hospital revealed the disappearance of the splenial lesion, indicating successful treatment (Fig. 1b).

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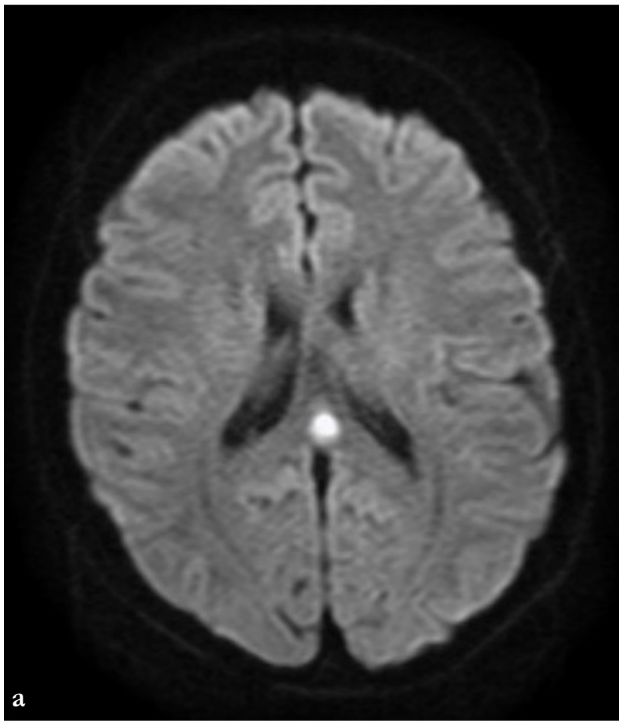

Fig. 1a. Magnetic resonance imaging (MRI) reveals the high-intensity signals in the splenium of the corpus callosum on diffusion-weighted images.

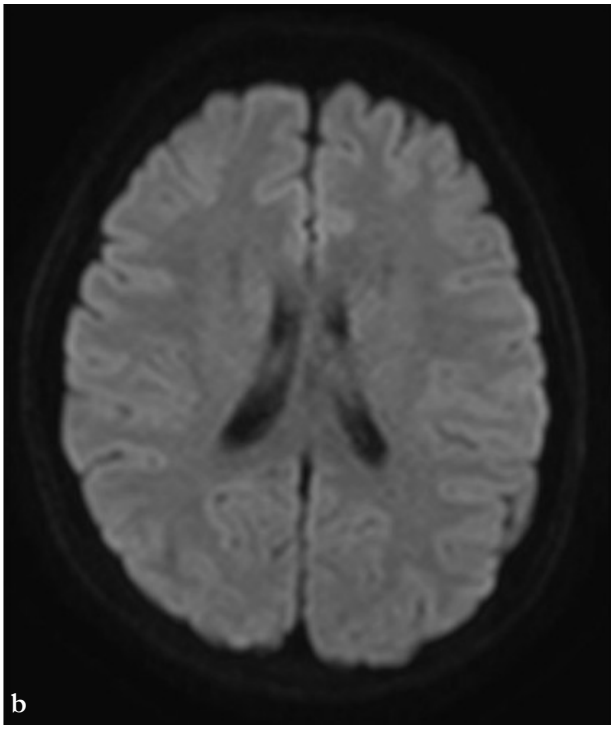

Fig. 1b. MRI obtained two weeks after admission to our hospital reveals the disappearance of the splenial lesion on diffusion-weighted image.

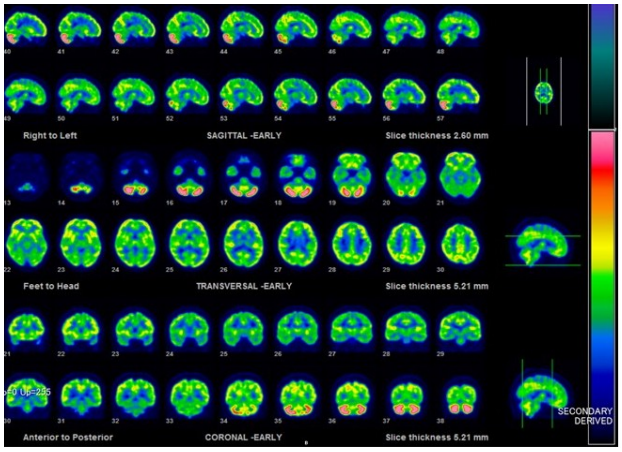

Fig. 2a. Fluorine-18 2-deoxy-2-fluoro-D-glucose positron emission tomography (^{18}F-FDG PET) reveals increased glucose metabolism in the cerebellum.

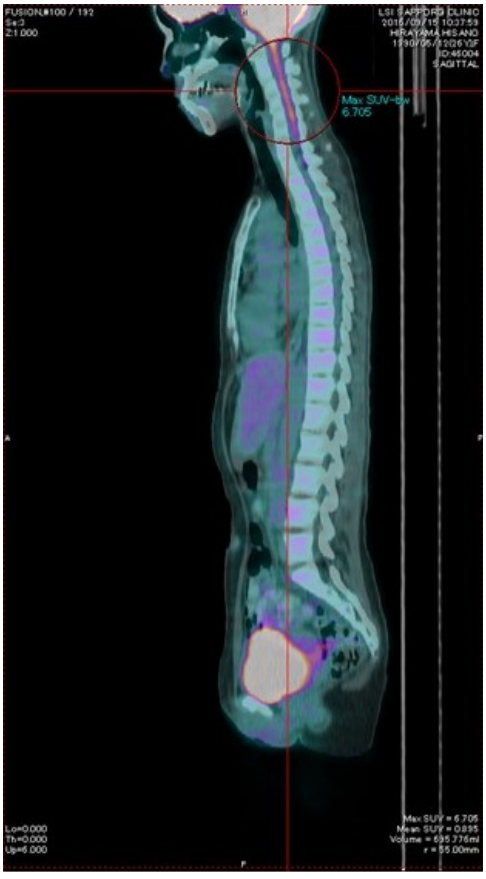

Fig. 2b. ^{18}F-FDG PET reveals increased glucose metabolism in the upper spinal cord.

Some cases of MERS are triggered by infections, either viral (such as influenza virus, rotavirus, mumps virus, and varicella-zoster virus) or bacterial (such as *Salmonella enteritidis, Escherichia coli* O157, *Mycoplasma pneumonia,* and *Legionella pneumophila*) (1). On the other hand, MERS has also been reported to be associated with hypoglycemia, antiepileptic drug toxicity or withdrawal, trauma, and high-altitude disease (2, 3). According to previously published reports, the clinical symptoms of MERS include non-specific

symptoms such as fever, headache, seizure, drowsiness, and delirium (4). With regard to the clinical symptoms other than the abovementioned ones, Hibino et al. reported a case of adult-onset adenovirus-associated MERS accompanied by transient hemiparesis and hemianesthesia (1). They mentioned that there were no symptoms or signs associated with the splenial lesion (1). Tomizawa et al. also reported a case of Legionella-associated MERS accompanied with gait disturbances, the cause of which was speculated to be a cerebellar lesion, and not a splenial one (5). Imai et al. demonstrated cerebellar hypoperfusion on single-photon emission computed tomography in a case of Legionella-associated MERS accompanied with cerebellar dysfunction (6). In the present report, MRI-detected splenial lesion could not be detected by ^{18}F-FDG-PET, although the cerebellar and the upper spinal cord lesions were detected by ^{18}F-FDG-PET. Based on these findings, an MRI-detected splenial lesion does not necessarily represent the cause of neurological symptoms and signs. Hence, we should not adhere only to the MRI-detected splenial lesions while searching for the cause of neurological symptoms and signs in MERS.

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