We report serial MR imaging of an 11-year-old boy who had a recurrent episode of clinically mild encephalitis/encephalopathy with a reversible splenial lesion. During the first episode, brain lesions were limited to the corpus callosum. However, for the second episode, the lesions were distributed in the corpus callosum and bilateral deep white matter. No abnormality remained in the follow-up MR images obtained after full recovery.

**Index words**: Encephalitis  
Recurrence  
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Diffusion Magnetic Resonance Imaging

Recently, transient splenial lesion with restricted water diffusion has been reported in various conditions including clinically mild encephalitis/encephalopathy with a reversible splenial lesion (MERS), withdrawal of antiepileptic drugs, as well as metabolic or toxic insult such as immunoglobulin therapy (1–3). Despite the different clinical conditions, previously reported cases showed very similar clinical and MR imaging patterns. In almost all cases, the neurological symptoms were transient, and the brain lesions were usually limited to the corpus callosum (CC) and were reversible. We recently experienced a similar case, but the episode was recurrent. In this case, we describe the clinico-radiological course and discuss the possible pathogenesis.

**Case Report**

A previously healthy 11-year-old boy was admitted to our hospital presenting with abdominal pain over the course of the previous day. On admission, he was alert without any neurological abnormalities. During the evening of his admission, drowsiness associated with high fever developed and progressed to delirium on the second hospital day (HD). Blood examinations revealed mild leukocytosis (11,200/mm³). The blood culture was negative and antmycoplasma antibody titer was significantly elevated (1:1,280 positive) in the serum. The altered consciousness continued until diffusion-weighted MR imaging (DWI) was performed on the second HD (spin-echo EPI, TR/TE = 5,300/98, 4-mm section thickness, b factor = 2,000 sec/mm²) and it revealed the lesion within the corpus callosum (CC) (Fig. 1). The apparent diffusion coefficient (ADC) value was within the range of 330–390 × 10⁻⁶mm²/s. CSF examination and viral studies were not done. On the third HD, without any specific treatment except IV cefotaxime and oral erythromycin, his mental
status began to return towards normal. On the 11th HD, he recovered completely and was discharged.

About 3 months later, when an influenza alarm was issued, he was again admitted to our hospital due to a severe headache and high fever up to 40°C. Initially, he was alert and oriented. On the first HD, a CSF examination was performed under the impression of acute meningitis. His WBC count in the CSF was 465/\textmu m^3 (80% polymorphonuclear granulocyte). The concentrations of protein and glucose were 43 mg/dL and 82

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Fig. 1. MR imaging of the first episode. Diffusion-weighted imaging (DWI) and ADC map (A, B) indicated that the lesions in the genu and splenium of the CC (arrows) show a pattern of water diffusion restriction (high signal intensity on the DWI, low signal intensity on the ADC map).

Fig. 2. MR imaging of the second episode. DWI (A-C) and ADC map (D-F) demonstrate the lesion in the entire CC and deep periventricular white matter, which show a pattern of restricted water diffusion.
mg/dL, respectively. Blood examinations revealed leukocytosis (21,400/mm³) and his anti-mycoplasma antibody was positive at 1:1,280 dilution. Microbiologic tests including PCR of the blood and CSF were negative for bacteria and herpes simplex virus, enterovirus, and mycoplasma pneumoniae. On the first HD, he was diagnosed with aseptic meningitis and supportive treatment including 20% mannitol injection was started. Through the first night in hospital, he started to show delirious mental status. As he was considered to have encephalitis or encephalitis-associated condition, brain MRI and EEG were performed. EEG on the second HD showed diffuse irregular continuous slow waves and frequent bursts of high amplitude rhythmic delta waves with both frontal areas. DWI on the second HD revealed distinct lesions with high signal intensity in the deep periventricular white matter and the entire CC (Fig. 2). The ADC value in the CC was within the range of 240–346 × 10⁻⁶ mm²/s. Following brain MRI, the influenza virus was considered as the possible cause and Tamiflu was administered on the second HD. On the third HD, he began to show dramatically improved mental status. On the seventh day, he was discharged without any neurological abnormalities. Follow-up brain MR imaging after 20 days revealed no abnormalities. The ADC value of the CC was within the range of 430–450 × 10⁻⁶ mm²/s. Although he showed complete normal condition and no abnormal lesion remained in the MRI, there were still several intermittent rhythmic or arrhythmic slow waves in both frontal areas on follow-up EEG. The final follow-up, which included brain MRI and EEG, was performed 4 months after the second attack, and there were no abnormalities observed in the brain MRI (Fig. 3) or the EEG.

**Discussion**

Transient CC lesions with restricted water diffusion in DWI is a well-known phenomenon associated with various conditions including meningocerebralitis, withdrawal of antiepileptic drug, metabolic disorder [hypoglycemia, electrolyte imbalance] and high altitude cerebral edema [4]. The known pathogen associated with MERS include the influenza virus (A and B, 19%), mumps virus (7%), adeno virus (6%), Streptococcus (6%), and Escherichia coli (6%). However, the pathogen associated MERS is unknown in 41% of cases. One limitation in our report was that we did not evaluate all possible pathogens of MERS, and we could not determine the exact pathogen. The common imaging features were mildly increased signal intensity on T2WI, high signal on DWI, and decreased ADC values [1, 5, 6]. It is still not clear why CC is always involved and why ADC is decreased with the lesion site. Intramyelin edema due to separation of the myelin layer, transient inflammatory infiltration like in multiple sclerosis, hyponatremia resulting in water influx to brain and cerebral edema, and excitotoxic injury have been suggested as the reason of the reduced water diffusion [1, 3, 7].

There were no previous reports concerning the recurrence of the MERS associated with meningocerebralitis or other conditions. In our case, the lesion loads were
different between the two episodes. The different lesion loads may be due to different time course between the CC lesion and other white matter lesion (8), or the difference in severity between the two episodes. We could not clearly explain the reason for the recurrence and we consider the possibility of various cytokine (such as interleukin-6) mediated immune responses to be the causative agents [1, 9, 10]. In our case, anti-mycoplasma antibody titer was elevated in both episodes, and this infectious agent might be the possible pathogen in our case.

In conclusion, transient CC lesion with restricted water diffusion may recur under the circumstances of repeated exposure to the causative agents. In our case, the clinical course and imaging finding was more severe in the recurrent episodes.

References