Rotavirus-infected children with clinically mild encephalopathy with a reversible splenial lesion (MERS)

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DESCRIPTION

A previously healthy 2-year-old boy presented to our hospital with seizure. He had fever, diarrhoea and vomiting for 2 days before admission. His seizure continued for 2 min and was accompanied by loss of consciousness and tonic seizure. His seizure spontaneously stopped and he fully recovered. He did not have Kernig's sign or neck stiffness. Blood test and cerebrospinal fluid (CSF) analysis were normal. We made a diagnosis of simple febrile seizure.

However, 20 h after admission, 2 min of tonic seizure occurred when he was crying. At the same time, rotavirus (RV) antigen was detected in his stool.

These were some atypical findings which were different from typical simple febrile seizure that not tonic-clonic seizures but tonic seizure, occurrence of the seizure 2 days after the onset of the illness, and occurrence of the second seizure 20 h after the first seizure. So, he was diagnosed as possibly having encephalitis and we performed neuroimaging evaluation.

Diffusion-weighted MRI of the brain showed an intensified signal in the splenium of the corpus callosum (figure 1A). Apparent diffusion coefficient (ADC) mapping showed decreased ADC values at the abnormality (figure 1B).

We confirmed a diagnosis of RV infection-associated clinically mild encephalopathy with a

Figure 1 (A) Diffusion-weighted MRI of the brain was performed on day 3 after onset of illness showed an intensified signal in the splenium of the corpus callosum. (B) Apparent diffusion coefficient (ADC) mapping (day 3) showed decreased ADC values at the abnormality. (C) Diffusion-weighted MRI (day 9) and (D) ADC mapping (day 9) revealed complete disappearance of the original lesion.

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reversible splenial (MERS) lesion.¹ He was treated with oral phenobarbital and seizure did not recur. EEG performed on day 5 was normal. A follow-up brain MRI revealed completely normal findings (figure 1C, D) and phenobarbital was discontinued on day 9.

Tada *et al*¹ identified MERS as a new type of acute encephalopathy which was characterised by transient splenial lesions with high-signal intensity in diffusion-weighted MRI, a mild course and a good outcome in 2004. MERS has been reported in encephalitis or encephalopathy induced by not only RV, but also influenza virus, adenovirus and mumps virus.¹ Although MERS usually transiently recovers, MERS with RV infection should be paid attention to because of progression to cerebellitis.¹ There have been some reports of hypotheses about RV-associated MERS.^{2–4} Some reports suggested that RV-associated MERS was caused by direct viral invasion of the central nervous system.² However, the other reports denied this hypothesis.³ Moreover, MERS could occur by other viruses. So, it is natural to conclude that the pathogenic mechanism of MERS is not RV-specific. In our case, RV was not detected in CSF by nested-reverse transcription PCR.

On the other hand, it has been proposed that MERS may be caused by intramyelinic axonal oedema (related from hyponatraemia) or local inflammatory cell infiltration (elevation of inflammatory cytokines such as interleukin-6 (IL-6)).⁴

However, hyponatraemia and elevation of IL-6 levels in the CSF are not always found in MERS.

Taken together, the mechanisms of RV-associated MERS remain unclear.

Learning points

- ▶ Neuroimaging should not be performed in the routine evaluation of the children with simple febrile seizures and the work-up of children with complex febrile seizures needs to be individualised.^{5 6}
- ▶ When the patient with retrovirus gastroenteritis has 'atypical' febrile seizure, it is important to observe the patient carefully and think a possibility of encephalitis including mild encephalopathy with a reversible splenial lesion.

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