Case Report

A Case of Mild Encephalopathy with a Reversible Splenial Lesion Associated with G5P[6] Rotavirus Infection

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We report a case of mild encephalopathy with a reversible splenial lesion (MERS) associated with acute gastroenteritis caused by rotavirus (RV) infection. The patient (male, 4 years and 3 months old) was admitted to our hospital for diarrhea and febrile seizures. Head MRI revealed a hyperintense signal in the splenium of the corpus callosum on DWI and a hypointense signal on the ADC-map. After awakening from sedation, the patient’s disturbance of consciousness improved. On day 5 after admission of the illness, the patient was discharged from the hospital in a good condition. Electroencephalography on day 2 after admission was normal. On day 8 of admission, head MRI revealed that the splenial lesion had disappeared. RV antigen-positive stools suggested that RV had caused MERS. This RV genotype was considered to be G5P[6]; it may have spread to humans as a strain reassortment through substitution of porcine RV into human RV gene segments. This extremely rare genotype was detected first in Japan and is not covered by existing vaccines; this is the first sample isolated from encephalopathy patients. Few reports have investigated RV genotypes in encephalopathy; we believe that this case is valuable for studying the relationship between genotypes and clinical symptoms.

1. Introduction

Tada et al. (2004) reported a case involving clinically mild encephalopathy with a reversible splenial lesion (MERS) [1]. The clinical symptoms of this condition include delirium, disturbance of consciousness, seizures, and vomiting. The splenial lesions show hyperintense signals on diffusion-weighted imaging (DWI) and a low apparent diffusion coefficient (ADC). These lesions characteristically disappear within 1 week. The prognosis of MERS is generally good [2]. We report a case of MERS associated with rotavirus (RV) infection.

2. Case Presentation

A male patient aged 4 years and 3 months presented to the ER (emergency room) of our hospital with vomiting, diarrhea, and seizures. On day 1 of the illness, he had developed gastrointestinal symptoms, including vomiting and watery diarrhea that occurred 4-5 times per day. Four days later, bilateral generalized tonic-clonic seizures began, for which he was transported to our hospital. The seizures lasted up to 1 minute and subsided spontaneously. The patient was in a restless state of consciousness (level E3V4M5 according to the Glasgow Coma Scale and level II-10 according to the Japan Coma Scale). He stared, averted his gaze, continued to cry, and threw objects. This disturbance of consciousness persisted for 7 hours. The patient was sedated with midazolam, and MRI was performed. On awakening after 2 hours, the patient had regained a normal state of consciousness, after which there was no recurrence of disturbance of consciousness or seizures.

On admission, the patient’s vital signs were as follows: temperature, 37.7°C; heart rate, 130 beats/minute; blood pressure, 100/60 mmHg; and SpO2, 98% (room air). No
Figure 1: (a) Head MRI on day of admission (day 4 of illness). MRI was performed on the day the seizures and disturbance of consciousness appeared. DWI reveals a hyperintense signal in the splenium of the corpus callosum and a hypointense signal on the ADC-map. (b) Head MRI performed 8 days after admission (day 12 of the illness). Head MRI (DWI, ADC-map) shows that the abnormal signal detected in the splenium of the corpus callosum had disappeared.

3. Discussion

RV is a dsRNA virus belonging to the Reoviridae family. Variability in the genotypes of RV in humans affects the expression of the outer shell proteins VP4 P (proteolytic cleavage protein, “P”) and VP7 G (glycoprotein, “G”) [4]. To date, 27 G genotypes and 35 P genotypes have been confirmed. Of these, 5 GP genotypes account for approximately 90% of human RV infections worldwide: G1P[8], G2P[4], G3P[8], G4P[8], and G9P[8] [6].

In 2006, the administration of 2 types of live vaccines, namely, Rotarix and RotaTeq, was initiated worldwide. These vaccines are effective against the major genotypes of RV [7]. In our patient, the RV genotype isolated from the stool was G5P[6]. Because G5P[6] is extremely rare, the protective effects of existing vaccines have not been investigated.

According to a national survey by Hoshino et al. (2012), 40 (4.0%) of the 983 encephalopathy cases reported between 2007 and 2010 in Japan were caused by RV infection. RV-induced encephalopathy is not uncommon, ranking third after influenza virus (26.6%) and HHV-6 (17.0%). Furthermore, 18 of the encephalopathy cases (45%) caused by RV were MERS. Compared with the incidence of influenza virus (20.2%) and HHV-6 (1.8%), the incidence of MERS is high [3].

Few reports have investigated the genotypes of RV associated with encephalopathy. The genotypes of the 14 RV encephalopathy cases diagnosed between 2005 and 2010 in Japan were all group A RV, with 4 cases of G3, 3 cases of G1, 1 case of G2, and 6 cases untyped (detected from stool samples); all were highly prevalent human RV genotypes. To date, only five G5P[6] RV strains have been isolated worldwide, all of which were detected in human gastroenteritis patients [5, 8]. To the best of our knowledge, this is the first instance of G5P[6] RV detection in an encephalopathy patient.
It is unclear whether RV readily causes encephalopathy, and individual human factors may contribute to the pathogenesis.

This was the first G5P[6] RV sample isolated from an encephalopathy patient.

Moreover, G5P[6] is not covered by existing vaccines. Understanding RV genotypes in encephalopathy is important for defining this pathological condition and enabling the development of a suitable vaccine.

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References


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