

## Laboratory and Epidemiology Communications

### Detection of the Rotavirus A Genome from the Cerebrospinal Fluid of a Gastroenteritis Patient: A Case Report

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From March to April of 2006, rotavirus A accounted for more than a half of the viruses isolated from the fecal specimens of gastroenteritis patients treated in the pediatric sentinel hospitals of Kanagawa Prefecture, Japan. We report here a clinical case in which the patient developed gastroenteritis followed by encephalitis, possibly caused by rotavirus A.

The patient was a female aged 3 years and 7 months whose first symptoms, diarrhea, vomiting and abdominal pain,

appeared on April 24. Her subsequent clinical course is summarized in Table 1. The cerebrospinal fluid, throat swab and stool specimens, obtained on April 29, May 1 and May 3, respectively, were subjected to reverse transcriptase-polymerase chain reaction (RT-PCR) using the primers described by Gouvea et al. (1). In the case of the stool specimens, rotavirus A genome was detected in both the first and second PCRs, while in the case of the cerebrospinal fluid and throat swab specimens, it was detected only in the second PCR. The primer pairs that showed positive amplification were those for rotavirus A type G3. Given that the same rotavirus A genome, type G3, was detected in both the cerebrospinal fluid and in the throat swab and stool specimens, and that clouding of consciousness followed gastrointestinal symptoms

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Table 1. Clinical course of the patient

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Patient:	3-year and 7-month-old female
April 24, 2006:	First symptoms, diarrhea, vomiting and abdominal pain, appeared. On consultation of a hospital, she was diagnosed as rotavirus gastroenteritis, as her stools were found positive for the virus.
April 26:	She was admitted to the hospital with complaints of fever and abdominal pain. The gastrointestinal symptoms became ameliorated after treatment.
April 28:	Clouding of consciousness appeared.
April 29:	The cerebrospinal fluid was collected for laboratory tests including RT-PCR. The diagnosis was aseptic meningitis.
May 1:	Throat swab specimens were collected for RT-PCR.
May 3:	Stool specimens were collected for RT-PCR.

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(Table 1), we concluded that the patient's acute encephalitis was caused by the rotavirus. During this period, there was an epidemic of rotavirus A infection in Kanagawa Prefecture, during which the G1 and G3 serotypes showed comparable frequency.

The amplified VP7 region of the virus was directly sequenced and the sequence was found to be similar to that of the 98'B31 strain isolated in 2000 in China (accession no. AF260958). Electron microscopy confirmed the presence of rotavirus

particles in the patient's feces.

Patients who suffer acute encephalitis or encephalopathy associated with rotavirus A infection, even when the cerebrospinal fluid is positive for the rotavirus A genome, generally recover without sequelae, but serious neurological disturbances, such as developmental problems or paralysis have been reported in some cases (2). In cases of meningitis or encephalitis following gastroenteritis, we recommend investigation of the cerebrospinal fluid for rotavirus A.

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