Acute cerebellar ataxia with human parvovirus B19 infection

Yoshitaka Shimizu, Tamaki Ueno, Hiroshi Komatsu, Hiroshi Takada, Tadasu Nunoue

Abstract

A 2 year old boy developed acute cerebellar ataxia in association with erythema infectiosum. During the disease, genomic DNA and antibodies against human parvovirus B19 were detected in serum but not in cerebrospinal fluid. Parvovirus B19 associated acute cerebellar ataxia might occur due to transient vascular reaction in the cerebellum during infection.

(Arch Dis Child 1999;80:72-73)

Keywords: acute cerebellar ataxia; erythema infectiosum; human parvovirus B19

Although human parvovirus B19 (PVB19) is well known to cause erythema infectiosum, it also has other pathological manifestations such as aplastic crisis, thrombocytopenia, purpura, myocarditis, and arthritis. In central nervous system (CNS) disease, meningitis and encephalitis have been reported in association with this viral infection, but acute cerebellar ataxia has not previously been documented. We report a case of acute cerebellar ataxia associated with PVB19 infection.

Case report

A 2 year old boy presented to his physician because of frequent vomiting. He was diagnosed with cyclic vomiting and received intravenous fluids. Over the next day he developed truncal ataxia and nystagmus, resulting in an inability to walk and to maintain a sitting position. On the fourth day he was seen in our hospital with bright red erythema on both cheeks and a maculopapular lacy erythema on his extremities. The rash subsided two days later although he still had mild ataxic gait and horizontal nystagmus. As the patient was not cooperative, detailed neurological examinations was difficult except for patellar tendon reflexes, which were normal. He was diagnosed with acute cerebellar ataxia. A toxicology screen was not performed, and there was no recent history of vaccination or drug intoxication.

White blood cell count was $8.0 \times 10^9/l$, with 42.5% polymorphs, 2% eosinophils, 1.5% basophils, 6% monocytes, and 48% lymphocytes. C reactive protein was 0.1 mg/dl and erythrocyte sedimentation rate was 9 mm in the first hour. Cerebrospinal fluid (CSF) was normal with cell count 9/ μ l, protein 8 mg/dl, and glucose 65 mg/dl. Oligoclonal bands were not found and myelin basic protein was < 0.5 ng/ml in CSF. Serum electrolytes and urinary analysis by the stick test and microscopy were normal. Serology was negative for

antibodies to varicella zoster virus, coxsackie B1, B2, B3, B5 viruses, and *Mycoplasma pneumoniae*. IgM and IgG antibodies against PVB19 were positive at a standard dilution of 1:200 in serum but negative at four dilution points between 1:50 and 1:400 in CSF. PVB19 DNA investigated by polymerase chain reaction (PCR)¹ was also positive in serum but negative in CSF. Nested PCR using six different primer sets revealed that the strain of PVB19 in our case was not a special variant virus. Magnetic resonance imaging (MRI) and single photon emission computed tomography (SPECT) performed on days 8 and 10 after presentation respectively showed no abnormal findings. An electroencephalogram on day 10 was also normal. Therefore, this case was thought to be acute cerebellar ataxia caused by PVB19 infection. Ataxia and nystagmus disappeared in a week without any specific treatment. He was discharged on day 12.

Discussion

Acute cerebellar ataxia occurs in association with various viral and non-viral infections and vaccines. It has been assumed that most acute cerebellar ataxia cases result from postinfectious allergic autoimmune reactions. Recently, antineuronal antibodies were detected in a patient with acute cerebellar ataxia following Epstein-Barr virus infection.² Although meningitis and encephalitis related to PVB19 have been reported, it is not clear whether PVB19 directly invades nerve cells in such cases. Okumura and Ichikawa reported the direct invasion of PVB19 into the CNS in a case of aseptic meningitis in which PVB19 DNA, IgM, and IgG antibodies against PVB19 were positive in the CSF.³ In our case, neither PVB19 DNA nor antibodies were detectable in CSF, which suggests that the mechanism for acute cerebellar ataxia does not involve direct viral invasion. A postinfectious allergic reaction, as reported in other viral infections, could be responsible, as the incubation period to acute cerebellar ataxia is almost the same for erythema infectiosum as for other viruses.

Some investigators have suggested the pathophysiology of acute cerebellar ataxia involves oedematous cerebellitis based on MRI⁴ and SPECT⁵ findings. In our case, no abnormalities were noted, possibly because the cerebellitis was mild as suggested by the clinical course. Recently, Brown *et al* demonstrated that the cellular receptor for PVB19 was an antigen of the blood group P.⁶ This P antigen is expressed not only on erythroid progenitor cells but also on endothelial cells. Nunoue has detected

Department of
Pediatrics, Otsu
Municipal Hospital,
Motomiya, Otsu,
Shiga, 520 Japan

Y Shimizu
T Ueno
H Komatsu
H Takada

School of Health
Sciences, Kyushu
University, Fukuoka,
Japan

T Nunoue

Correspondence to:
Dr Shimizu.

Accepted 6 May 1998

PVB19 in endothelial cells of the fetal cerebrum (unpublished data 1997). Therefore, acute cerebellar ataxia could be caused by vascular injury in the cerebellum, which may manifest as oedematous cerebellitis in severely damaged cases.

To the best of our knowledge, this is the first case report of acute cerebellar ataxia associated with PVB19 infection. Further attempts are required to clarify the role of this virus in the development of acute cerebellar ataxia as about 30% of PVB19 infections in children are not manifest as erythema infectiosum.

We thank Dr Shinsaku Imashuku for reviewing this manuscript.

- 1 Umene K, Nunoue T. A new genome type of human parvovirus B19 present in sera of patients with encephalopathy. *J Gen Virol* 1995;**76**:2645–51.
- 2 Ito H, Sayama S, Irie S, *et al.* Antineuronal antibodies in acute cerebellar ataxia following Epstein-Barr virus infection. *Neurology* 1994;**44**:1506–7.
- 3 Okumura A, Ichikawa T. Aseptic meningitis caused by human parvovirus B19. *Arch Dis Child* 1993;**68**:784–5.
- 4 Shoji H, Hirai S, Ishikawa K, *et al.* CT and MR imaging of acute cerebellar ataxia. *Neuroradiology* 1991;**33**:360–1.
- 5 Yazawa S, Ochi T, Sugimoto S, *et al.* A case of acute cerebellar ataxia with abnormal single photo emission computed tomography. *Clin Neurol* 1995;**35**:424–7.
- 6 Brown KE, Anderson SM, Young NS. Erythrocyte P antigen: cellular receptor for B19 parvovirus. *Science* 1993;**262**:114–17.