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Case Report

An infant case with hydrocephalus as the initial manifestation of *Mycoplasma hominis*-associated meningitis

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ABSTRACT

We report an infant with hydrocephalus as the initial manifestation of *Mycoplasma hominis*-associated meningitis, who recovered without appropriate antimicrobial treatment. The analysis of the 16S rRNA gene by polymerase chain reaction amplification using universal primers and pathogen-specific primers was useful for the diagnosis and the investigation of serial detection status of the pathogen. This method may be helpful for the assessment of the frequency and the prediction of severity in *M. hominis*-associated central nervous system infection in infants, and investigating the association between *M. hominis* and the development of hydrocephalus.

1. Introduction

Mycoplasma hominis rarely causes severe infection in central nervous system (CNS) [1,2]. The incidence of *M. hominis*-associated CNS infection might be underestimated because of the difficulty of obtaining positive culture on routine bacteriological methods. High morbidity and mortality of the infection were assumed to result from delayed diagnosis and ineffective initial antimicrobial treatment [2–7]. On the other hand, some cases recovered with no treatment or with inappropriate treatment [8,9].

Polymerase chain reaction (PCR) amplification of the 16S rRNA gene is a particularly powerful method for detecting the causative pathogen when bacterial cultures of specimens from affected tissues are negative [10]. This method takes a small amount of work and time, because causative pathogens are identified by the direct sequencing analysis after the PCR amplification [10,11]. In contrast, the analysis of the 16S rRNA gene using pathogen-specific primers can be performed more briefly to investigate serial detection status after the identification of the pathogen.

We herein report an infant case with hydrocephalus as the initial manifestation of *M. hominis*-associated meningitis. The combination of the analyses of the 16S rRNA gene using universal primers and pathogen-specific primers was useful for the diagnosis, the prediction of the time of onset and the investigation of serial detection status of the pathogen.

2. Case report

A male infant weighing 2160 g at 33 weeks of gestation was born by vaginal delivery. The membranes of his mother ruptured 2 weeks before delivery. The cervix was fully dilated at 33 weeks of gestation, and the infant was delivered spontaneously. The infant was admitted to our neonatal intensive care unit for dyspnea, and the hospitalization continued because of prolonged central apnea. Asymmetry of ventricular sizes was revealed in ultrasonography at birth whereas no change of the finding had been observed. The head magnetic resonance imaging (MRI) on 34-day-old showed mild subependymal hemorrhage (Grade I) and slight dilation of the left lateral ventricle (Fig. 1A).

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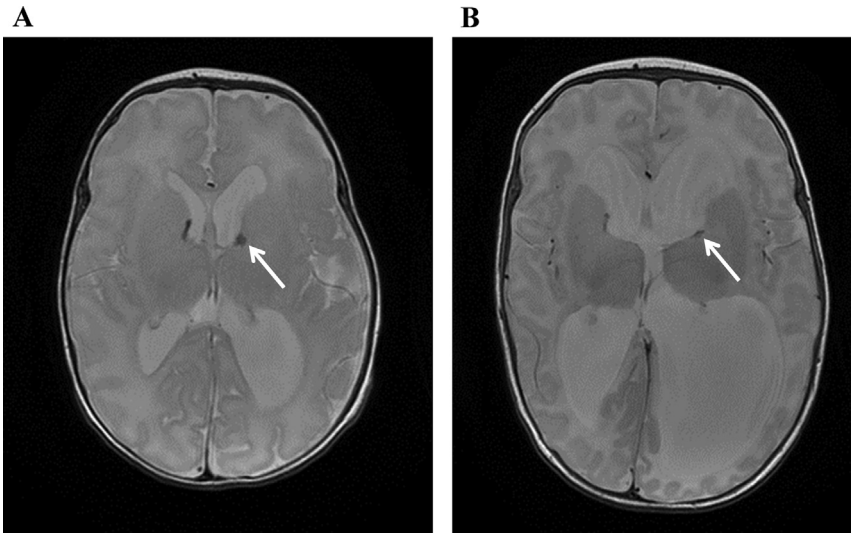

Fig. 1. T2 weighted magnetic resonance imaging on 34-day-old (A) and on 84-day-old (B). Arrows indicate mild subependymal hemorrhage (Grade I).

The frequency of apnea gradually decreased and the abnormal neurological findings did not appear. However, his head circumference rapidly dilated from 72-day-old. The head MRI on 84-day-old showed the progression of the dilations of the lateral ventricles and the third cerebroventricle and the appearance of the septum-like structure in the fourth ventricle, leading to the diagnosis of non-communicating hydrocephalus (Fig. 1B). Neither leukocytosis in peripheral blood nor elevation of serum C-reactive protein (CRP) level was observed. The placement of a ventriculo-peritoneal (VP) shunt was performed on 86-day-old. Cerebrospinal fluid (CSF) analysis obtained on the operation revealed a cell count of 44/μl with 95% of mononuclear leukocytes, a protein level of 157 mg/dl and a glucose level of 24 mg/dl. CSF bacterial culture was negative. Seven days after the operation, he had a fever and redness of skin on abdomen along with the VP shunt tube (Fig. 2). Leukocyte count in peripheral blood was 11,900/μl, and serum C-reactive protein (CRP) level was 3.3 mg/dl. Removal of the shunt tube and extra-drainage were performed on 98-day-old. CSF revealed a cell count of 86/μl, a protein level of 183 mg/dl and a glucose level of 18 mg/dl. Blood, CSF and device (shunt tube) bacterial cultures were negative. VP shunt tube was reinserted after the improvement of all symptoms. However, on 115-day-old, the shunt tube was removed again despite the administration of antimicrobial agents because of the occurrence of the same symptoms (Fig. 2). Bacterial cultures were all negative. We performed PCR amplification of the 16S rRNA gene using universal primers to identify the causative pathogen as previously described [11]. The samples obtained on 115-day-old were initially used for the analysis. *M. hominis* was identified from the

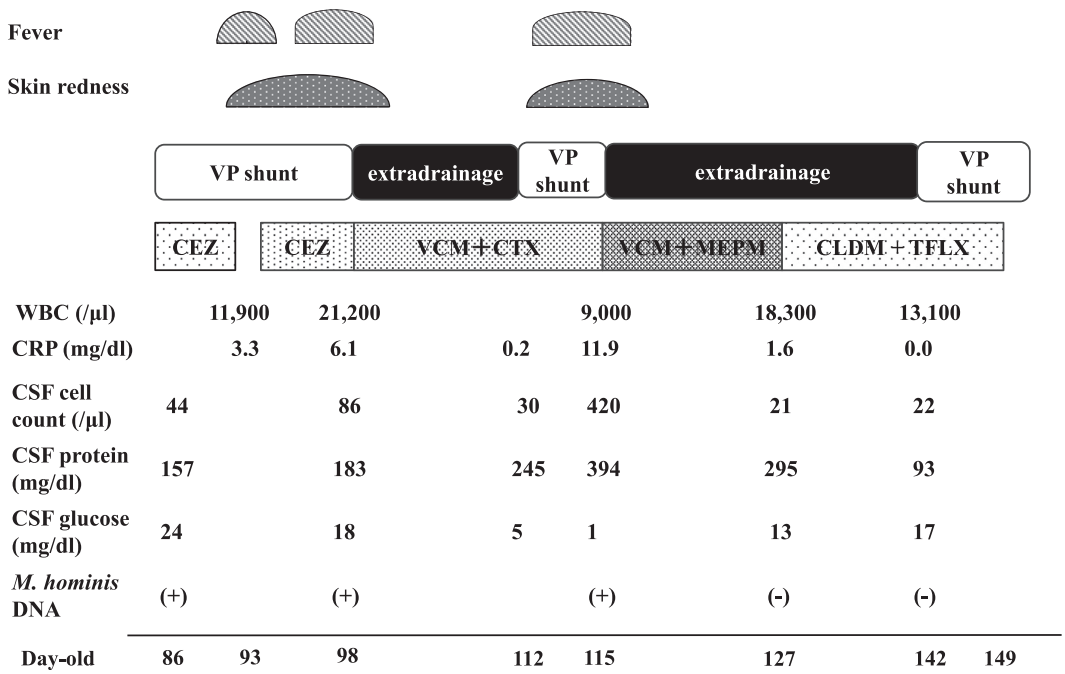

Fig. 2. Clinical course and serial laboratory data. VP shunt: ventriculo-peritoneal shunt, CEZ: cefazolin, VCM: vancomycin, CTX: cefotaxime, MEPM: meropenem, CLDM: clindamycin, TFLX: tosufloxacin, WBC: white blood cell, CRP: C-reactive protein, CSF: cerebrospinal fluid, *M. hominis*: *Mycoplasma hominis*.

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