A case of bacterial meningitis with burst waves of local onset on ictal Electroencephalography

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Bacterial meningitis is a common childhood infection of the central nervous system. However, after Haemophilus influenzae type b (Hib) vaccine and 7-valent pneumococcal conjugated vaccine (PCV7) were introduced in 2008 and 2010, respectively, the rate of bacterial meningitis decreased in Japan.¹ On the other hand, there are a few unvaccinated children.

There are many typical symptoms of meningitis such as fever, vomiting, irritability, and convulsions. However, infants frequently display only the symptom of feeling unwell. Seizures occur before or during the first several days after admission to the hospital in as many as one-third of the cases of meningitis.² This study reports the first case of bacterial meningitis with burst waves of local onset on an ictal electroencephalogram (EEG).

The patient was a 5-month-old female. She had no family history and no perinatal history. Her developmental history up to 5 months also recorded no complications. Her vaccination history included only bacille Calmette-Guérin (BCG). She had convulsions with fever twice a day just before hospital admission. The first seizures occurred after 12 h from fever onset. She did not have a stiff neck or anterior fontanelle bulging. A neurological examination also produced normal findings. Her consciousness was clear between convulsions. Cranial magnetic resonance imaging (MRI) was also normal. The third convulsion occurred during the recording of the EEG. It showed normal findings in the natural sleep state before the convulsion (Fig. 1a). The paroxysmal discharges were recorded with a central focus on F3 polar; however, she had no outward changes (Fig. 1b). These discharges broadened out to the left hemisphere area when convulsions appeared only on the fingers of the right hand (Fig. 1c). The spike and waves appeared predominantly on the left hemisphere when the convulsions from the right hand were generalized (Fig. 1d). Laboratory findings showed high white blood cell (WBC) counts (33 500/mm³) and C-reactive protein levels (CRP) (3.69 mg/dL). Cerebrospinal fluid (CSF) cell counts revealed increased leukocytes (816/3), including 33% lymphocytes and 67% neutrophils. The patient was diagnosed with bacterial meningitis. Intravenous vancomycin (60 mg/kg/day) and ceftriaxone (100 mg/kg/day) were administered immediately after the diagnosis. On day 5, penicillin-susceptible S. pneumoniae (PSSP) was detected in the blood and in the CSF culture specimens taken on the day of admission. We substituted ampicillin (400 mg/kg/day) on the same day based on the results of the antibiotic sensitivity test.

On day 6, her body temperature dropped to 36.0 °C. Antibiotic drugs were administrated for 14 days in total. The auditory brain-stem response was normal on day 12. She was discharged from the hospital on day 17. A sharp F3 polar wave appeared on an interictal EEG after 1 year. Then there were no sequelae for 2 years. However, she had another seizure 3 years after suffering from meningitis. Interictal EEG showed repeated epileptic discharges in the same area as shown on the ictal EEG at 5 months old. Her seizure was considered to be secondary generalized based on the findings of previous ictal EEG and recent interictal EEG. We therefore administered carbamazepine to her.

It has been assumed that meningitis is the inflammation of the entire brain and meninges; however, paroxysmal discharges on ictal EEG were partly the cause in this case. A previous report reported that the majority of patients had partial-onset seizures after bacterial meningitis.³ We presume the following three hypotheses regarding that consistency. First, the epileptogenicity of our patient existed at birth. She actually developed epilepsy 3 years after the onset. The paroxysmal discharges she suffered in relation to meningitis correspond with the epileptic discharges in a 3 year old. Second, the whole inflammation might be the partial cause of origin. Pomeroy et al. described how acute seizures may occur as a response to injuries sustained by the cerebral hemispheres during meningitis.⁴ The expected changes were not seen on the MRI. Nevertheless, we surmise that it could be a subtle inflammation corresponding with the electrode location at the paroxysmal discharge on the EEG findings. Third, it also relates to maturation and / or development. The paroxysmal
Discharges in a certain limited area become fully generalized in many cases on the ictal EEGs of infants and/or neonates. There are also some reports that paroxysmal discharges are started by the local area of infantile and/or neonatal convulsions. We think it is related to the immaturity of brain nerve development. It is difficult to prove which hypothesis is right. However, we think that ictal EEG, in this case, enables us to infer a pathology of childhood convulsions.

Informed consent was obtained from the patient’s parents for this presentation.

Disclosure

The authors declare no conflict of interest.

Author contribution

HisY wrote the paper. HisY and YM carried out patient care. HitY contributed to the interpretation of seizures and EEG findings. All authors read and approved the final manuscript.

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