Acute purulent meningitis associated with chronic subdural hematoma and subdural hygroma

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SUMMARY: Imataka G, Miyamoto K, Fujiyama Y, Mitsui M, Yoshida A, Yamanouchi H, Arisaka O. Acute purulent meningitis associated with chronic subdural hematoma and subdural hygroma. Turk J Pediatr 2007; 49: 437-440.

A male infant aged nine months with meningeal irritation and +3.5SD expansion of the circumference of the head was admitted. Brain computed tomography (CT) detected right chronic subdural hematoma and contralateral subdural hygroma. Since the cell count was increased on a cerebrospinal fluid test, acute purulent meningitis was diagnosed. A rapid latex test and culture of cerebrospinal fluid identified *Streptococcus pneumoniae* (PSSP) as the pathogen. Panipenem/betamiprom (PAPM/BP) was administered at 100 mg/kg/3 times for 14 days and dexamethasone was administered at 0.6 mg/kg/4 times for 4 days, and the patient recovered without sequelae. Acute purulent meningitis complicated by chronic subdural hematoma and subdural hygroma is rare. In this case, acute subdural hygroma may have concomitantly developed with acute purulent meningitis in the presence of the chronic subdural hematoma, and rapid disequilibrium of intracranial pressure may have been the developmental mechanism.

Key words: magnetic resonance imaging, shaken baby syndrome, Streptococcus pneumoniae, panipenem/betamiprom.

Meningeal sign was noted in an infant brought to our hospital for fever, vomiting, and increased head circumference. Brain computed tomography (CT) detected unilateral chronic subdural hematoma and subdural hygroma on the opposite side. Lumbar puncture was performed, and a diagnosis of acute purulent meningitis was made based on the findings of cerebrospinal fluid. There has been no report of acute purulent meningitis complicated by chronic subdural hematoma and subdural hygroma. We report the clinical course and characteristic imaging findings of the patient.

Case Report

The patient was a nine-month-old male infant who was born by cesarean section because of a narrow pelvis at 39 weeks of gestation; his weight was 3,695 g. Asphyxia was not noted at birth. The patient's father had been hospitalized for a long period for viral encephalitis. The patient lived with his mother and a four-year-old sister. His mental and motor developments were normal. Past medical history of the patient revealed no obvious trauma. A slight increase in the head circumference was noted about two months after birth. Bacterial otitis media had repeatedly occurred for six months. Regarding the present illness, rhinitis occurred in the spring of 2004, and the patient was brought to an otorhinolaryngologist. Since fever and recurrent vomiting started on the 7th hospital day, and activity decreased, the patient was referred to the emergency outpatient department of our hospital, and admitted.

At admission, the length, body weight, and head circumference were 75 cm (+0.9 SD), 11.6 kg (+1.1 SD), and 50 cm (+3.5 SD), respectively. The consciousness level was 2 on the Japan Coma Scale. The patient was not doing well, and was easily irritated by surrounding sounds. The anterior fontanel

was distended, and neck stiffness and Kernig sign were noted. No hemorrhage was noted in the retina or vitreous body in either eye. Flare of the pharynx and bilateral bacterial otitis media were noted. There were no abnormalities in the thoracic or abdominal region. No injury or bruise suggesting abuse was noted on the external surface skin of any part of the body. Whole bone X-ray radiography was normal. Brain CT and magnetic resonance imaging (MRI) (Fig. 1A) detected a subdural high density area in the right anterior temporal region, and a low density area in the left anterior temporal region. The cerebral ventricles were not extended, and myelination corresponded to his month of age.

On peripheral blood testing, white blood cell count was 34,500/μL, and C-reactive protein (CRP) was 19.8 mg/dl, showing strong inflammatory reactions. No anemia, hypoglycemia, liver function disorder, or abnormal electrolyte was noted. Coagulation test including vitamin K was normal. On cerebrospinal fluid test, the initial pressure was 40 mmHg, and the cell count was 328 (neutrophil: 270, lymphocyte: 20). The cerebrospinal fluid protein and glucose levels were 20 mg/dl and 77 mg/dl, respectively. Cocci were observed in gram-stained cerebrospinal fluid under a microscope. On rapid latex diagnosis of microorganisms in cerebrospinal fluid, *Streptococcus pneumoniae* antigen reaction was positive. Cerebrospinal fluid culture test was performed a few days later, and *S. pneumoniae* (PSSP) was detected and determined to be the etiologic bacteria.

Subdural puncture was performed through the anterior fontanel to reduce intracranial pressure (Fig. 1B). The cerebrospinal fluid contained old blood clots, consistent with the finding of chronic subdural hematoma. For treatment of meningitis, 100 mg/kg/day divided three

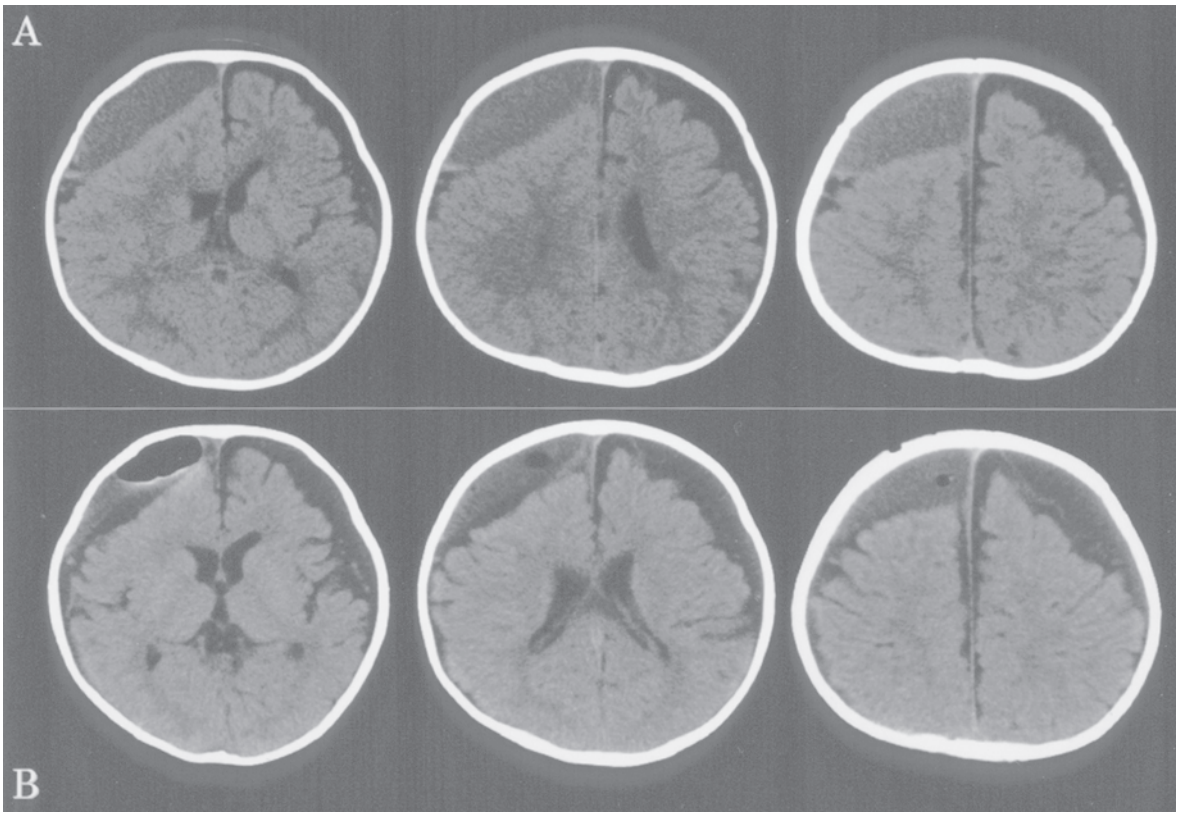

Fig. 1A: Upper three slices are brain CT images on admission. A chronic subdural hematoma was noted in the right anterior temporal region. The cerebral cortex of the right anterior temporal region was compressed, and the right lateral ventricle was narrowed. No midline shift was noted. In the contralateral subdural cavity, subdural hygroma was present.

Fig. 1B: Lower three slices are brain CT images after subdural puncture. Air due to puncture was noted.

doses of panipenem/betamiprom (PAPM/BP) was administered daily for 14 days and 0.5 mg/kg/day divided four doses dexamethasone was administered for 4 days with concomitant mannitol. The symptoms of meningeal signs were resolved within a few days.

The patient was followed for 1 year and 6 months after the occurrence by brain MRI, and abnormal MR imagings were improved. Bilateral auditory brainstem responses (ABR) showed normal findings and the patient had good hearing ability. No sequelae were noted in movement or speech as of three years of age, nor was there any progression of expansion of the head circumference. No convulsion occurred during the clinical course.

Discussion

The cause of subdural hematoma is rupture of the cerebral bridging veins of the skull and brain surface. Since the bridging veins are thin and easily injured in neonates and infants, the incidence of subdural hematoma is high in this developmental period[1]. For example, birth trauma causes subdural hematoma in neonates[2]. However, no abnormality was noted in the birth course of the patient. Occurrence of chronic subdural hematoma after very minor head trauma, despite the absence of an obvious trauma, has been reported in infants[3]. Since chronic subdural hematoma is easily missed in the infantile period without a past history of trauma, and causes complications of macrocephalia, developmental disorder, and consciousness disorder, caution is necessary. Although the patient's mother reported that the patient had no past history of trauma, the mother may not have noticed trauma of the head of her child. In addition, the patient was an infant and could not complain of headaches. Shaken baby syndrome is known to develop subdural hematoma without a history of trauma in infants aged less than two years, particularly at the age of 3-6 months[4]. Shaken baby syndrome is accompanied by eye fundus hemorrhage in 75-90% of cases, and diagnosed upon status epilepticus, differing from the course of this patient. However, observation, including for abuse, is necessary because the possibility of shaken baby syndrome could not be completely excluded.

Symptoms of chronic subdural hematoma in infants include recurrent vomiting, convulsion, distention of the anterior fontanel, and expansion of the head circumference. The patient's chronic subdural hematoma may have been the cause of the increased head circumference. The patient's head circumference had increased due to acute purulent meningitis before admission, although the time of development of chronic subdural hematoma diagnosed based on the images and punctate was not clear.

In subdural hygroma, fluid is retained in the space between the dura mater and arachnoid, and this frequently occurs in neonates to infants. It should be differentiated from external hydrocephalus, in which the subarachnoid space expands. The most frequent cause in the neonate and infant with subdural hygroma is associated with trauma and acute purulent meningitis has also been reported[5]. The main pathology of purulent meningitis with subdural hygroma is perivascular inflammation in the arachnoid containing abundant blood vessels, and when inflammation ruptures the arachnoid, bacteria may invade below the dura mater and cause leakage of exudates. Cytokines such as interleukin (IL)-6 and tumor necrosis factor (TNF)-α are considered to be the cause of subdural hygroma[6].

The mechanism of concomitant development of acute purulent meningitis with chronic subdural hematoma and subdural hygroma in this patient was unclear. Since the head circumference had increased in the patient, and CT detected chronic subdural hematoma, the dura mater and arachnoid may have already been likely to rupture before the development of the symptoms. In addition, the chronic subdural hematoma was unilateral and subdural and subarachnoid pressures on the opposite side may have been likely to lead to rupture to maintain intracranial pressure equilibrium, readily inducing subdural hygroma in this case with acute purulent meningitis.

In Japan, a clinical trial of a 7-valent pneumococcal conjugate vaccine for infants and children has been in progress since 2004. However, the vaccine has not yet been introduced in Japan as of July 2006[7]. Therefore, this patient did not receive the vaccine. The 7- or 11-valent conjugate vaccines for prevention of various pneumococcal infections have been recently launched in several countries[8]. Early introduction of the pneumococcal conjugate vaccine is necessary not only in Japan but also throughout the world for prevention of pneumococcal infections.

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