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Case Report

Brown-Sequard Syndrome in an 8-year-old Girl : a Case Report

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SUMMARY

Brown-Sequard syndrome is caused by hemilateral damage of the spinal cord, and has rarely been reported in pediatric patients. We report an 8-year-old girl with Brown-Sequard syndrome. Neurological examination showed motor impairment and deep sensation disturbance below the level of the 8th thoracic vertebra on the right side, and impairment of pain and temperature sensation on the left side. Clinical symptoms allowed an early diagnosis of Brown-Sequard syndrome. Steroid pulse therapy was started early, resulting in a daily improvement of motor paralysis of the right lower extremity. However, left-sided sensory dissociation persisted. At 3 years after the onset of the syndrome, the right lower extremity was more slender than the left lower extremity ; although, interestingly, thermography showed that the skin temperature was lower on the right than on the left side. Clinical and laboratory studies failed to reveal any specific causes of the development of Brown-Sequard syndrome.

Key Words : Brown-Sequard syndrome, steroid pulse, thermography, childhood

INTRODUCTION

Brown-Sequard syndrome is caused by hemilateral damage of the spinal cord. The first description of its pathological features dates back to the report by Brown-Sequard in 1861 [1]. Symptoms include ipsilateral motor impairment and deep sensation disturbance, and contralateral impairment of touch, pain, and temperature sensation below the level of the lesion. The causes include trauma, blood flow disturbance, tumors, or degenerative diseases of the spinal cord, but are often unknown. Many cases have been reported in adults [2, 3], but few in childhood [4, 5]. Herein we report an 8-year-old girl with Brown-Sequard syndrome.

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CASE REPORT

The patient was an 8-year-old girl who visited our hospital because of gait disturbance. She was born via a normal delivery, and had no history of previous disease or trauma. Her first symptom was back pain. She went to school the next morning, and had nausea during the day, followed by numbness in the right lower limb in the afternoon. Several hours later, she became unable to move the right lower limb, stand, or walk, and was brought to the outpatient clinic. At the first examination, she had clear consciousness. The movement of her face, eyeballs, neck, and upper extremities were all normal. She was able to maintain a sitting position, urinate, and defecate. Neurologically, abdominal reflexes were absent. The patellar and Achilles tendon reflexes were slightly diminished in the left lower ex-

tremity, with a positive Babinski sign. In the left lower extremity, the iliopsoas, quadriceps femoris, hamstrings, gastrocnemius, and tibialis anterior muscles were all normal in strength, and they completely moved against strong resistance. In contrast, in the right lower extremity, none of these muscles contracted, and no joints moved. Pain and temperature sensation were absent in the skin regions innervated by the nerves below the level of the 8th thoracic spine. In the right lower extremity with motor paralysis, pallanesthesia was noted in the corresponding regions. To investigate the cause of the symptom, we performed blood and urine analysis on admission, which showed no abnormalities. The anti-herpes simplex virus antibody titer and anti-cytomegalovirus antibody titer, as determined by the complement fixation (CF) test, were less than 1 : 4. The anti-mycoplasma (PA) antibody titer was 1 : 80. Cerebrospinal fluid (CSF) analysis showed a cell count of 9/3, protein level of 23 mg/dl, and glucose level of 55 mg/dl, with an IgG index of 0.51 (normal range, 0.34 ± 0.15). On neurophysiological examination, the motor nerve conduction velocity, sensory nerve conduction velocity, F-waves, and H-waves were within normal limits for the age, that is, there were no peripheral nerve abnormalities. Spinal cord MRI, including that with gadolinium enhancement, revealed no abnormalities.

She was neurologically diagnosed with Brown-Sequard syndrome. After informed consent from her parents, steroid pulse therapy with prednisolone (30 mg/kg for 3 days) was instituted. She was orally administered with vitamin B, and was started on rehabilitation therapy from the next day. A period of 3 days after treatment, the quadriceps femoris muscle of the immobile right lower limb showed a slight contraction. Later, the motor function of the right lower limb gradually improved, and she became able to walk at 11 days after onset. However, the sensory dissociation due to impairment of pain and temperature sensation on the left side of the abdomen and the left lower limb persisted. The sense of vibration at the medial malleoli of the right and left tibiae using a tuning fork of 128 Hz was 8-9 sec and 17 sec, respectively. At one and a half months after onset, thermography at a reference temperature of 34.2℃ (Fig. 1) was performed, which showed lower skin temperatures in the right half of the abdomen and the right thigh and leg than in their left counterparts. Three years have passed since onset, and the motor function of the lower extremities has improved so much that the patient rides a bicycle, and walks up and down stairs, but, the impairment of pain and temperature sensation on the left side persists. The right lower extremity is more slender than the left side.

DISCUSSION

Brown-Sequard syndrome is caused through two mechanisms. One mechanism is external compression of the spinal cord or impairment of venous blood flow, resulting in a necrosis or infarction lesion in the cord ; for example, traumatic or idiopathic spinal cord hernia, tumors outside the spinal cord (such as teratoma, schwannoma, meningioma, and lymphosarcoma), arteriovenous malformations with hemorrhage outside the cord, and spontaneous spinal epidural hematoma. The other mechanism involves the direct effects of intraspinal lesions, including spinal cord trauma, demyelinating diseases (such as multiple sclerosis and acute disseminated encephalomyelitis), transverse myelitis, intraspinal tumor, allergic myelitis, and Hopkins syndrome[2, 3]. Many cases have been reported in adults, but few in childhood[4, 5].

Although spinal cord MRI, blood, and CSF findings aid in determining the causes of Brown-Sequard syndrome, we were not able to determine the cause in this patient. However, the neurologically characteristic symptoms allowed us to make an early clinical diagnosis and start early steroid pulse treatment. Since the spinal MRI findings excluded hemorrhage and neoplastic lesions, we selected steroid pulse therapy, which is based on evidence for the treatment of demyelinating diseases developing through neuro-immunological mechanisms. In addition, this therapy is expected to have an antiinflammatory effect on pathological states related to myelitis. We cannot demonstrate how the steroid pulse therapy was effective in this patient ; however, it was clinically effective in achieving a daily improvement of motor function. At present, more than 3 years have passed since onset, she leads the normal life of a typical elementary school student, but, the sensory dissociation due to impairment of pain and temperature sensations persists. Very interestingly, the

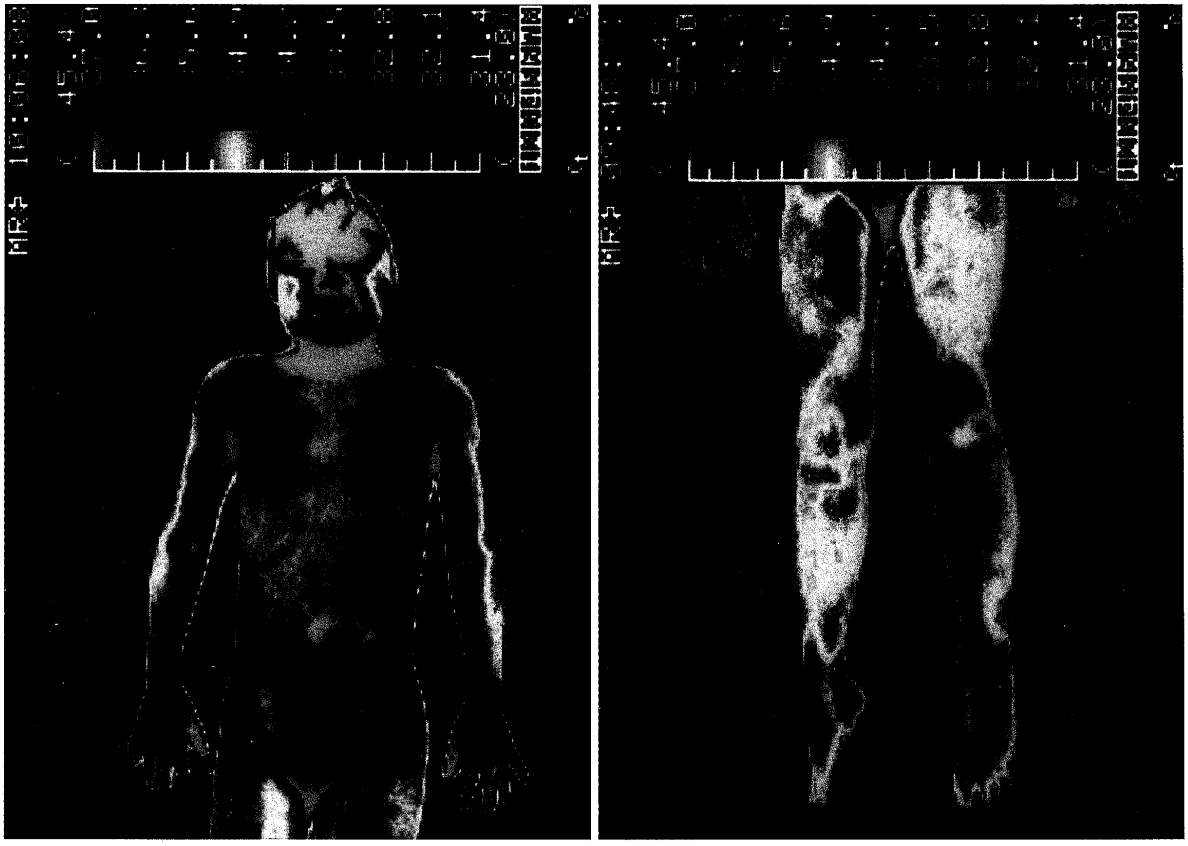

Fig. 1 Entire body thermogram recorded at a reference temperature of 34.2 ℃.

lower temperature in the right lower extremity observed on her thermogram reflects the difference in diameter between both lower extremities that have gradually manifested over a period of 3 years. Since no studies have reported thermographic findings in Brown-Sequard syndrome, more cases need to be accumulated for a better assessment of at least their clinical significance. We are considering the evaluation of the muscles of the slender lower extremity by CT and MRI and MRA to evaluate the state of blood vessels.

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