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Medullary lesion revealed by MRI in a case of MS with respiratory arrest

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Summary. A 66-year-old MS patient who suffered respiratory arrest followed by complete remission is reported. Magnetic resonance imaging (MRI) revealed a medullary lesion. Clinicoradiological relationships are discussed.

Key words: Multiple sclerosis – Respiratory arrest – Magnetic resonance imaging – Dual respiratory pathways

Brainstem lesions in multiple sclerosis (MS) can be easily detected by MRI. Lesions in the medulla oblongata are said to be present in 50% of cases with clinically definite MS [1]. Respiratory failure due to involvement of the primary respiratory neurons in the medulla oblongata is rare in MS. Boor et al. [2] reported a MS patient with respiratory failure who had a medullary lesion at autopsy. We report an unusual case of respiratory arrest which recovered, in a patient with MS. A medullary lesion was shown by MRI.

Case report

A 66-year-old woman was admitted to the Tokyo Metropolitan Geriatric Hospital on January 23, 1987, because of progressive paraparesis. She had had an episode of paraparesis with sensory disturbance below the C8 dermatomes bilaterally in 1976. Paraparesis had improved in three months, but numbness had remained. In 1978, she had suddenly developed diplopia and facial paresis on the left side, both of which improved in three months. In late December, 1986, she developed nausea, persistent hiccups, blurred vision, and paresthesia in the face. Thereafter she noticed that position sense was lost in the right upper extremity. On January 20, 1987, she developed weakness of the lower extremities, which progressed until admission.

Physical examination on admission revealed blood pressure of 114/60 mm Hg and pulse rate of 60 beats per min. The temperature was 36.2 °C. She was alert and cooperative. She complained of blurred vision despite normal visual acuity. There was dysesthesia in the face and numbness below the C8 dermatomes bilaterally. The deep tendon reflexes were exaggerated in all extremities and plantar responses were both extensor. Vibration and position sensation were decreased in all extremities, and absent in the right upper extremity.

The hemogram and serum biochemistry were normal. The CSF protein content was 52.9 mg/dl with 5.1 mg/dl of IgG (9.6%). Otherwise the CSF was normal. Brain CT showed no abnormality. EEG demonstrated slow alpha wave activity with small amount of delta and theta activity in the leads from the left hemisphere. Auditory brainstem responses (ABRs) were normal. In the somatosensory evoked potentials (SEPs) recorded after stimulation at the right median nerve, N9 was normal, but N13 and N20 were not detected. The blink reflex showed low amplitude of R1 and R2 in the left side.

Clinical course

Tetraparesis developed rapidly during the 5 days after admission. On January 29, she stopped

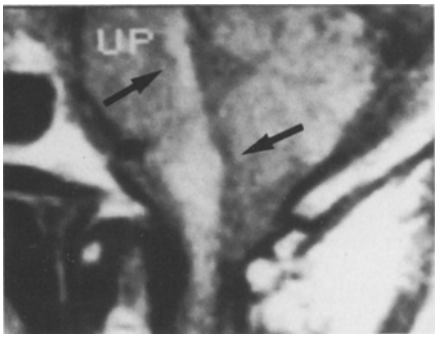

Fig. 1. Sagittal T2-weighted MR image. High-intensity areas are shown in the lower pontine tegmentum along the floor of the fourth ventricle and throughout most of medulla oblongata ventrodorsally. These involve the pyramidal tract ventrally, medial lemniscus centrally and the respiratory neurons and bulbal nuclei dorsally. There are no lesions in the cervical and upper thoracic spinal cord

breathing abruptly. At this time she was alert. Artificial breathing was started at once. She also showed a complete bulbar palsy. Soft palatal, gag and cough reflexes were absent. During the following 4 days weakness of the arms improved, and she could move her arms in a pseudoathetotic manner. Four weeks later spontaneous breathing appeared and became shallow and irregular with drowsiness. She could leave the respirator completely 7 weeks later. Weakness and proprioceptive sensory loss remained in both lower extremities. The CSF after respiratory arrest disclosed an increase of IgG% (38.4%), myelin basic protein (MBP) (8.4 ng/dl), and oligoclonal bands. In September, N 20 was recorded with long latency, but N 13 remained unremarkable. Blink reflex revealed temporal dispersed R 1 in the left side. MR imaging was performed after the respiratory arrest using a 0.5-Tesla superconducting MR imaging unit (Siemens Magnetom system). T2-weighted images were obtained using spin-echo pulse sequences (repetition time, 1600 ms; echo time, 75 ms). A high-intensity area was shown in the medulla oblongata and lower pontine tegmentum (Fig. 1). High-intensity areas were also shown in the white matter of right temporal and occipital lobes. In the cervical and upper thoracic segments of the spinal cord, abnormal findings were not found.

Discussion

The present case had three episodes of remissions and exacerbations of multiple neurological signs during twelve years with CSF findings compatible with the diagnosis of multiple sclerosis. She suffered abrupt respiratory arrest associated with bulbar palsy, tetraparesis and pyramidal signs. Her respiration recovered completely 7 weeks later.

The respiratory failure in multiple sclerosis is usually of ventilatory type caused by paralysis of the respiratory muscles. Respiratory failure due to involvement of the primary respiratory neurons in the medulla oblongata is rare in MS [3].

According to the concept of the dual respiratory pathways to the spinal respiratory neurons [4, 5], the voluntary respiratory pathway descends from the cerebral cortex via the pyramidal tract. The involuntary respiratory pathway arises from the primary respiratory neurons in the dorsolateral reticular formation of the medulla oblongata near the nucleus ambiguus [6]. That pathway crosses near the medial lemniscal decussation [2, 6], and descends just medial to the spinothalamic tract in the spinal cord [4, 7]. The type of respiratory disturbance can be attributed to the location of lesion.

Guthrie et al. [8] described respiratory failure with tetraplegia and sensory loss below the high cervical level in 4 cases of MS. One of them had MS lesions in the pontine tegmentum and in the lower cervical and upper thoracic spinal cord at autopsy, but the medulla oblongata was not involved. Noda et al. [9] reported one MS patient, who showed the selective loss of voluntary breathing with tetraplegia and sensory loss below the high cervical level. The respiratory failure of these five cases has been considered to be caused by the involvement of both pyramidal tracts running to the spinal respiratory neurons.

In contrast, Boor et al. [2] reported a MS patient with selective loss of involuntary breathing characterized by apnea during sleep. Bulbar sign and proprioceptive sensory disturbance in all extremities were observed. At autopsy, MS lesions were found in the central or dorsomedial region of the lower medulla oblongata, involving the primary respiratory neurons, nucleus ambiguus and medial lemniscus, but sparing the pyramidal tract. They considered that the disturbance of involuntary breathing was due to the involvement of primary respiratory neurons.

The present case showed the complete arrest of both voluntary and involuntary breathing. The former improved after 4 weeks, while the latter continued for 7 weeks. The involuntary respiratory arrest, bulbar signs and bilateral proprioceptive sensory loss observed in the present case were probably induced by the lesions of primary respiratory neurons, medial lemniscus and bulbar nuclei localized

mainly in the central and dorsal part of medulla oblongata. The voluntary respiratory arrest and tetraplegia were probably due to a lesion of the pyramidal tract which occupied the ventral part of medulla oblongata. MRI revealed such a lesion, a high intensity area throughout the whole medulla oblongata (Fig. 1). The lesion in the pontine tegmentum on MRI is considered to be responsible for left facial weakness and blink reflex abnormalities of R1.

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