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Bilateral ataxia after tumor resection in a patient with a unilateral thalamic tumor

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Abstract

Patients with thalamic lesions can experience ataxia on the contralesional side. We report here a 24 year old female patient who presented with a left thalamic tumor and experienced bilateral ataxia of the upper and lower extremities after a left thalamic resection. Her right-sided ataxia was more severe than that of the left side. The bilateral ataxia was likely to be from cerebellar ataxia. The right-sided ataxia may have been caused by injury of the dentatorubrothalamic tract originating from the dentate nucleus of the right cerebellum and terminating in the contralateral ventrolateral nucleus of the left thalamus. We believe that the left sided ataxia in this patient was due to an uncrossed dentatorubrothalamic tract, which control the movement of the ipsilateral side of the human body.

Key Words: cerebellar ataxia, brain Tumor, dentatorubrothalamic tract.

INTRODUCTION

Ataxia is one of the most frequent symptoms arising from a thalamic lesion, although its incidence has not been documented.[1] Two types of ataxia can occur after thalamic injury: sensory and cerebellar. Sensory ataxia results from impairment of positional sense, is defined as a poorly controlled direction of finger-to-nose and heel-to-knee movements or by attempts to grasp small objects, which become poorer with eye closure.[2] Cerebellar ataxia is associated with injury to the dentatorubrothalamic tract (DRTT) at the level of the injured ventrolateral nucleus of the thalamus.[3] Lesions interrupting the DRTT may cause cerebellar-like ataxia or tremor, clinically identical to symptoms caused by direct cerebellar damage. Ataxia arising from thalamic injury can occur on the side of the body contralateral to the lesion.[1-6] However, little is known about ipsilateral or bilateral ataxia induced by unilateral thalamic lesions.

We describe a patient who presented with a left thalamic tumor and experienced bilateral ataxia after tumor resection.

CASE REPORT

A 24-year-old, right-handed woman with no history of neurological or psychiatric illness was admitted to the neurosurgery department of our hospital complaining of motor weakness of the right extremities (Medical Research Council [MRC] scale[7]: 4/5). This motor weakness started 1 year previously and had become progressively more severe. On magnetic resonance imaging examinations of the brain on the day of admission, a left thalamic tumor was observed (Figure 1A). One week after admission, she underwent resection of the left thalamic tumor. During the operation, the cerebellum was not touched. Pathologic examination of the resected tumor revealed an extraventricular neurocytoma. Two months after this operation, she was transferred to the rehabilitation department of a university hospital for rehabilitative management. Brain MRI performed at that time revealed a defect on the left thalamus (Figure 1B). Clinically she exhibited bilateral ataxia of the upper and lower extremities, which was not present before the operation. The ataxia was much more severe on the right side than on the left side. Furthermore, she exhibited incoordination in finger-to-nose, finger-to-finger, and heel-to-knee tests, with normal initiation and velocity of movements, but with irregular accelerations and decelerations producing oscillations when near the target. This lack of coordination was severe in her right extremities and mild in her left extremities. It remained unchanged with the eyes open or closed, and was not improved by visual cues. She

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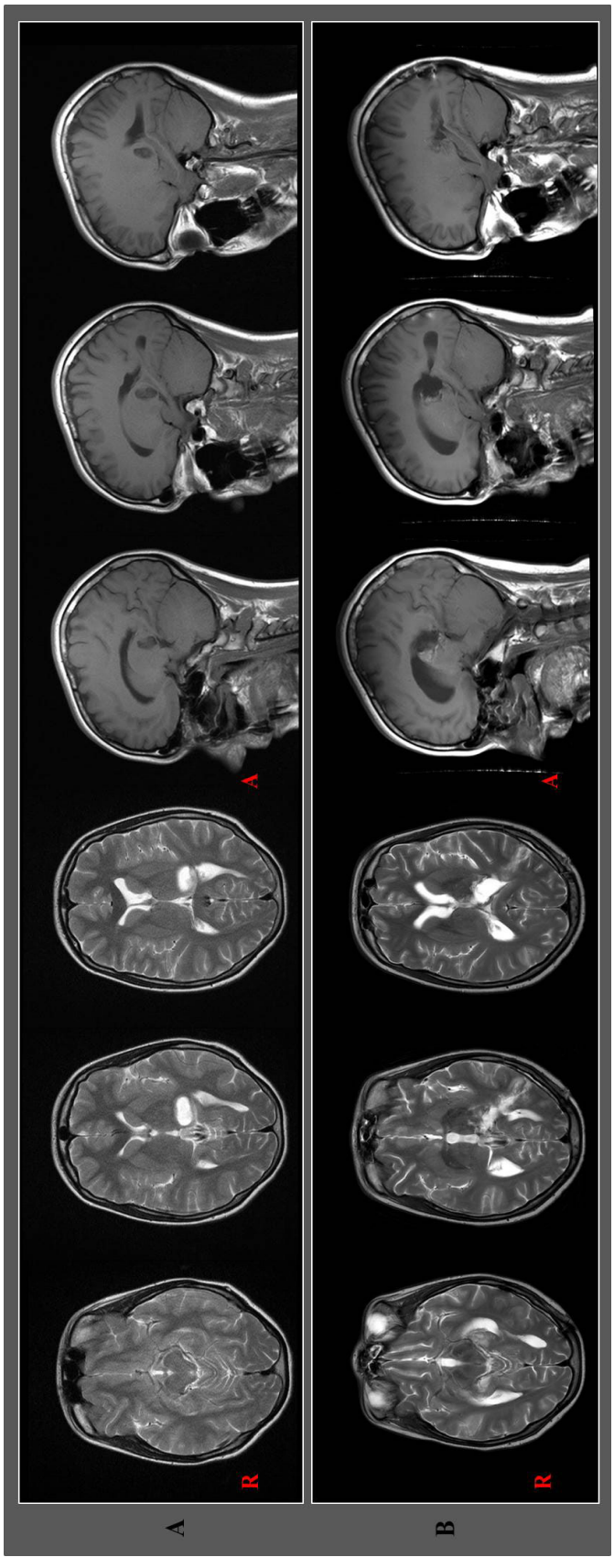

Figure 1 (A) Axial T2-weighted and sagittal T1-weighted MRI of the brain performed on the day of admission and prior to the operation. A tumor was visible in the left thalamus. (B) Axial T2-weighted and sagittal T1-weighted MRI performed 1 month after tumor resection showing a defect of the left thalamus and encephalomalacia in the left temporal lobe as a result of the operation.

also had severe gait ataxia, requiring continuous support when walking. Mild motor weakness of the right extremities was observed (MRC: 4), but she had no weakness of the left extremities. Tactile and kinesthetic sensations on her right side were impaired, with scores of 16 (normal 20) and 18 (normal 24) on the Nottingham Sensory Assessment[8] respectively. In particular, for kinesthetic sense on her right side, the patient was able to recognize and mirror the direction of the movement of her right extremities in all test movements, but often inaccurately mirrored the test movements with the difference of 10-20°. There was no somatosensory deficit in her left extremities. There was also no slurred speech and nystagmus observed.

The patient underwent a rehabilitative management program, including movement therapy (Monday through Friday: 2.5 hours/day and Saturday: 1 hour/day) and neuromuscular electrical stimulation therapy for the right finger extensors, knee extensors, and ankle dorsiflexors (Monday through Saturday: 2×20 minutes/day). This movement therapy focused on improving motor function of the right and left extremities, as well as trunk stability. One month after her transfer to the rehabilitation department, the patient's ataxia was significantly reduced, and she could walk independently on an even floor. However, the degree of motor weakness and sensory impairment of her right extremities were unchanged.

DISCUSSION

We report a case of bilateral ataxia after tumor resection from a left thalamic tumor. As the ataxia on both the right and left extremities did not deteriorate with eye closure, it was likely to be cerebellar ataxia. Cerebellar ataxia can be induced by damage to the DRTT[3] which is involved in the control of movement. Hence, abnormal movement, including ataxia, tremor, and dystonia, can occur when the DRTT is injured.[9] The DRTT originates from the dentate nucleus of the cerebellum and terminates in the contralateral ventrolateral nucleus of the thalamus after decussating to the contralateral red nucleus.[9] In our current patient therefore, the ataxia on the right extremities may have been induced by the DRTT connecting the right cerebellum and left thalamus. Several studies have reported ataxia on the contralateral side after a thalamic infarct or hemorrhage.[1-6] As for the left-sided ataxia of the extremities ipsilateral to the thalamic lesion, this may be due to injury of the uncrossed DRTT. Although the presence of an uncrossed DRTT has not been definitively established, a study by Lee *et al*.[10] supports its existence. The authors described a patient who previously experienced a left thalamic hemorrhage and developed a recurrent right hemiataxia after a right thalamic hemorrhage. That patient experienced a recurrence of prior neurological symptoms after a second stroke occurred in the ipsilateral hemisphere, although thalamic lesions usually cause ataxia on the contralesional side. The authors speculated that the hemiataxia caused by the first stroke improved as a result of compensation by the uncrossed DRTT, and that damage to this tract by the second stroke caused a reappearance of the pre-existing hemiataxia.

Aside from the bilateral ataxia, right motor weakness in our present patient could be explained by a mild injury to the left corticospinal tract in the posterior limb near the thalamus. Furthermore, impairment of touch and proprioceptive sensations in the right extremities of our patient were likely due to damage to the sensory-related regions of the thalamus.

In conclusion, we report a patient who exhibited bilateral ataxia after resection for a left thalamic tumor. Although most previous studies have described patients with thalamic lesions causing ataxia contralateral to the lesion, our patient exhibited ataxia of the ipsilesional side and contralesional side. This report is therefore the first document of bilateral ataxia following a unilateral thalamic lesion. We believe that our findings provide additional evidence for an uncrossed DRTT, which controls movement of the ipsilateral side of the human body. Further studies involving larger numbers of patients are required to validate our findings.

DISCLOSURE

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