INTERNAL MEDICINE

□ CASE REPORT □

Body Lateropulsion and Cerebellar Tremor in a Patient with Pontine Infarction

Ai Hosaka[1,2], Ryoya Tsunoda[2], Tetsuto Yamaguchi[2] and Yasuro Shibagaki[2]

Abstract

Body lateropulsion is known to be caused commonly by lateral medullary lesions but rarely by pontine lesions. It is also known to be associated with lesions of the dorsal spinothalamic tract or ascending graviceptive pathways. We herein report the case of a 75-year-old woman presenting with contralateral lateropulsion and cerebellar tremor caused by pons infarction. To our knowledge, this is the first case report of pontine infarction causing both lateropulsion and cerebellar tremor. Our case may be helpful in anatomical studies of ascending graviceptive pathways.

Key words: body lateropulsion, cerebellar tremor, pontine infarction

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Introduction

Body lateropulsion, which is characterized by irresistible falling to one side, is caused by central nervous system lesions in the absence of muscle weakness. Lateral medullary lesions are known to cause ipsilateral lateropulsion (1). Little is known about which other lesions can cause this condition, although there have been a few reports of body lateropulsion caused by lesions in the pons (2-5), midbrain tegmentum (6), cerebellar peduncle (7), and cerebellum (8). Body lateropulsion is also known to be associated with lesions in the dorsal spinothalamic tract or ascending graviceptive pathway (GP). However, the anatomical location of the GP is unclear. We herein report a case presenting as contralateral lateropulsion and cerebellar tremor caused by pons infarction. To our knowledge, this is the first report of these two signs together being caused by pons infarction. We believe our case will be helpful in anatomical studies of the GP.

Case Report

A 75-year-old woman with diabetes mellitus presented with difficulty moving her right upper limb. The day after symptom onset, she recognized tremor of the right hand and was unsteady on her feet. Three days after onset, she was admitted to our hospital. On admission, her blood pressure was 183/83 mmHg, and her pulse was a regular 86 bpm. She was alert. External ocular movement was normal, and there was no ocular deviation, nystagmus, or ocular lateropulsion. She had no motor weakness or sensory disturbance. Limb ataxia was absent because there was no dysmetria, dysdiadochokinesis, or decomposition of movement. However, the patient had an action tremor of the right hand that was evident during voluntary movement, such as in finger-to-nose testing, although there was no resting tremor. More precisely, the tremor was not apparent shortly after her hand started to move. However, as her hand approached the target, the tremor gradually became distinct, with gradually increasing amplitude and a rhythmic frequency (5 Hz). The tremor amplitude increased gradually from when the tremor started and continued for a while after the target had been reached. This sign matched the features of cerebellar tremor. The patient could not stand or walk without assistance; she tended to fall because of body axis deviation to the left, despite the absence of motor weakness, sensory loss, and limb ataxia. These signs matched the features of body lateropulsion. Brain magnetic resonance images revealed a fresh ischemic infarct in the left dorsal part of the middle pons

[1]Department of Neurology, Hitachinaka Medical Education and Research Center, University of Tsukuba Hospital, Japan and [2]Department of Neurology, Hitachi, Ltd. Hitachinaka General Hospital, Japan
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Correspondence to Dr. Ai Hosaka, ahosaka@md.tsukuba.ac.jp

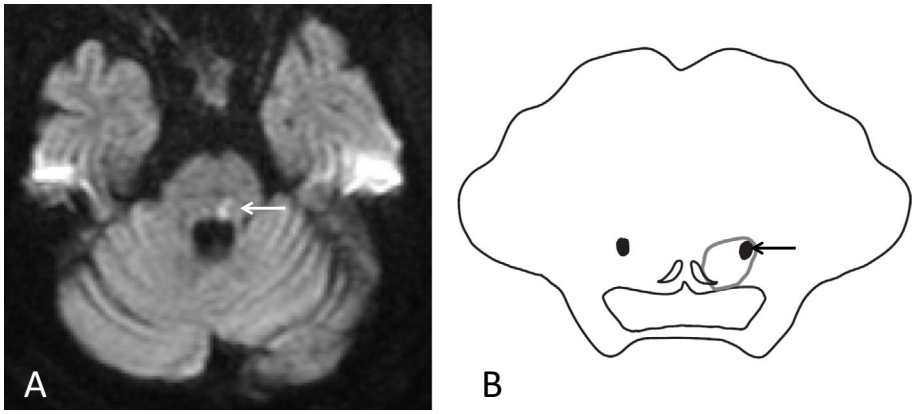

Figure. The patient's brain magnetic resonance imaging (MRI) findings. A: Diffusion-weighted MRI showing fresh infarct in the left middle dorsal pons (arrow). B: A schematic illustration of the middle pons. The arrow indicates the central tegmental tract. The gray lines show the locations of ischemic lesions.

(Figure A). Magnetic resonance angiography showed no severe stenosis in the basilar artery or vertebral arteries, but there was severe stenosis of the left M1 segment of the middle cerebral artery. The patient received antiplatelet therapy (ozagrel) and a cerebroprotective agent (edaravone). Her clinical signs gradually improved. The lateropulsion and action tremor gradually improved and had disappeared by two months after onset.

Discussion

There were two important clinical findings in the present experience. First, pontine infarction can present as body lateropulsion. Second, pontine infarction can present as cerebellar tremor at the same time.

Lateral medullary infarction is known to cause ipsilateral lateropulsion. However, there have been 11 documented cases of pontine infarction causing lateropulsion (2-5). The lateropulsion improved in most of these cases. Yi et al. (2) considered that the GP from the vestibular nuclei crosses the midline at the caudal pontine level, just above the level of the vestibular nuclei. Therefore, infarction at the pontine level could have affected the GP and thus caused lateropulsion in our patient.

Second, pontine infarction can present as cerebellar tremor. Our patient had no resting tremor but did show a 5-Hz action tremor that increased as her right hand approached the target; this feature matched that of a cerebellar tremor (9). Cerebellar tremor is caused mainly by cerebellar lesions. However, there have been some reports of lesions in the cerebellar peduncle and pons (10, 11) causing cerebellar tremor. Alstadhaug (10) reported a case in which bleeding of the pontine tegmentum caused oculopalatal tremor and contralateral cerebellar limb tremor. They considered that pontine lesions caused the cerebellar tremor through the effect of focal lesions in the central tegmental tract on the dentatorubral-olivary pathway. Our case likely had a similar mechanism (Figure B).

The dentatorubral-olivary pathway is often referred to as the Guillain-Mollaret triangle. In general, impairment of the Guillain-Mollaret triangle causes tardive involuntary movements such as cerebellar tremor (10, 11), midbrain tremor (12, 13), and palatal tremor (14). However, our patient presented with cerebellar tremor very soon after disease onset. An alternative contributing factor may have been disrupted cerebellar automaticity, as evidenced by the presence of cerebellar tremor. To our knowledge, this is the first case report of pontine infarction causing both lateropulsion and cerebellar tremor.

In conclusion, pontine infarction can present as lateropulsion and cerebellar tremor. The anatomical pathway of the GP remains unclear. However, the findings in our case suggest that the GP is located close to the central tegmental tract at the pontine level; these findings may help in carrying out anatomical studies of the GP.

The authors state that they have no Conflict of Interest (COI).

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