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Case Report

A Report of a Case Involving Body Lateropulsion with Numbness of the Ipsilesional Fingers Caused by a Small Infarction in the Dorsal Part of the Middle Medulla

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Keywords
Body lateropulsion · Finger numbness · Infarction · Middle medulla · Canal paresis · VEMP

Abstract
Based on the complexity of functional anatomy, a small infarction in the medulla can produce various types of clinical symptoms or signs depending on the location of this infarction. We describe the case of a 46-year-old man who presented with sudden onset of body lateropulsion to the left side and numbness of the ipsilateral fingers. 3-tesla diffusion-weighted magnetic resonance imaging with a section thickness of 2 mm revealed a small infarction in the dorsal part of the left middle medulla. To our knowledge, this is the first case report describing vestibular dysfunction apparent upon otoelectrophysiological examination but without vestibular symptoms or signs except for body lateropulsion.

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Introduction

Although lateral medullary syndrome is a well-known disease [1], analyses of lesions identified via magnetic resonance imaging (MRI) have revealed that various types of clinical symptoms or signs may be present depending on the location of a lesion in the medulla [2, 3]. Moreover, some reports have described patients with a small medullary infarction who developed body lateropulsion as their only or predominant symptom [4–6]. In this study, we report the case of a small dorsal medullary infarction that caused the sudden onset of body lateropulsion to the left side and numbness of the left fingers but did not produce other vestibular symptoms/signs.

Case Presentation

A 46-year-old man staggered and almost fell against the wall on his left side when he arose from bed one morning. He also felt numbness in the fingertips of his left hand. When he tried to touch his ear with his left hand, he touched his cheek or mouth instead. After 1 h, he went outside to see his sister. To avoid tilting to the left, he had to lean against the wall on his left side while he was standing, and his sister pulled his right arm rightward while he walked. These symptoms gradually improved but did not disappear, therefore he visited our hospital on the following day. He was neurologically well until the onset of the aforementioned symptoms, and neither vestibular nor cochlear symptoms had manifested during these 2 days.

At his initial visit (on day 2), he was neurologically normal except for body lateropulsion. Although he could walk and stand without assistance, he could not stand on one foot because of a tendency to tilt to the left. The forearm stepping test produced results in the normal range with a tendency to rotate toward the left. He reported numbness in his left fingers, especially the fingertips, but neither superficial nor deep sensory impairment was objectively detected. He was a current smoker and drank alcohol daily. Other than atopic dermatitis, his medical history was unremarkable. With the exception of his triglyceride level (209 mg/dL), his clinical laboratory values, including immunological test results, were in the normal range. No lesion was detected on 1.5-tesla diffusion-weighted (DW) MRI with a section thickness of 3 mm. His electrocardiogram was normal, and no embolic source was detected on intracranial magnetic resonance angiography, carotid ultrasound examination, or transthoracic echocardiogram. However, we tentatively diagnosed the patient with ischemic stroke, and aspirin and low-molecular-weight dextran solution were started.

On day 3, an extremely small lesion with high-intensity signal was detected in the dorsal part of the middle medulla on 3-tesla DW MRI with a section thickness of 2 mm (Fig. 1). On day 4, numbness in his left fingers disappeared, and he was barely able to stand on one foot. He underwent vestibular function tests using video-oculography (VOG). VOG performed with the patient in a supine position revealed no spontaneous nystagmus during the dark phase, and both smooth pursuit and saccade were normal. A cold-air (12°C) caloric test showed left canal paresis (40%), and visual suppression was observed (53%). Ocular vestibular-evoked myogenic potentials (VEMPs) elicited by vibratory stimuli at the forehead showed a 64% decline in amplitude when recorded below the right eye (Fig. 2), whereas no significant differences were observed for cervical VEMPs. Because the patient's audiogram showed bilateral conductive auditory impairment, air-conducted VEMPs were not examined. These oto-electrophysiological examinations showed nuclear- or infranuclear-type left vestibular dysfunction.

Discussion

We have described an extremely rare case involving a patient who presented with sudden onset of body lateropulsion to the left side with numbness of the left fingers but did not produce other vestibular symptoms/signs. We detected a small infarction in the dorsal part of the left middle medulla on 3-tesla DW MRI with a section thickness of 2 mm, and nuclear- or infranuclear-type left vestibular dysfunction was detected via otoelectrophysiological examinations.

Our patient's predominant symptom was body lateropulsion. The dorsal/ventral spinocerebellar tract, the descending lateral vestibulospinal tract (LVST), the vestibulo-thalamic pathway (ascending graviceptive pathway), the dentatoruburothalamic pathway, and the thalamocortical fascicle play important roles in the maintenance of body posture and stability, and a lesion affecting these pathways can cause body lateropulsion [7, 8]. In particular, injuries of the spinocerebellar tract and/or the LVST are known to cause body lateropulsion in patients with lateral medullary infarction or cerebellar infarction [1, 9]. Nevertheless, body lateropulsion as the only or predominant manifestation is rare in patients with these infarctions.

Maeda et al. [4] reported 3 cases in which body lateropulsion was the only or predominant symptom; these cases involved small lesions located on the dorsolateral surface of the caudal medulla. Kim et al. [5] described 6 patients who presented with isolated lateropulsion upon standing or walking and had lesions located on the lateral surface of the lower medulla. Because the lateral surface of the lower medulla contains the ascending dorsal spinocerebellar tract (DSCT), which mediates impulses from the proprioceptors in the ipsilateral lower trunk and leg, impairment of the DSCT is thought to be the cause of body lateropulsion in the aforementioned cases [10]. Moreover, Kim et al. [6] presented a case involving body lateropulsion associated with contralateral decreased pinprick and thermal sensations in the trunk and extremities. They concluded that a lesion that involved the descending LVST and the spinothalamic tract caused the observed symptoms, because the LVST, which is adjacent to the spinothalamic tract, mediates vestibulospinal postural control [6]. Using 3-dimensional brainstem mapping, Thömke et al. [11] analyzed the lesions of 10 patients with body lateropulsion and showed that body lateropulsion with limb ataxia was likely caused by impairment of the DSCT and that body lateropulsion without limb ataxia was likely caused by impairment of the LVST.

Relative to the aforementioned lesions, our patient's lesion was located more dorsally in the middle medulla. The dorsal part of the middle medulla contains the medial vestibular nucleus, the inferior vestibular nucleus, the accessory cuneate nucleus, and the inferior cerebellar peduncle [12]. Moreover, damage to the left vestibular function of our patient was demonstrated by otoelectrophysiological examinations. Therefore, impairment of the left LVST is a potential cause of body lateropulsion in our case.

Our case involved extremely atypical neurological symptoms of medullary infarction; in particular, the patient developed body lateropulsion without other vestibular symptoms and presented with ipsilateral sensory symptoms of the fingers but no contralateral symptoms. Ogawa et al. [2] reported 2 similar cases with the presentation of body lateropulsion and ipsilateral cerebellar ataxia but no vestibular symptoms; one patient developed ipsilateral sensory disturbance of the limbs and body, and the lesion in that case was located in the dorsolateral portion of the left middle medulla. Kim [13] reported 12 patients who presented with ipsilateral sensory symptoms of the limbs/body in addition to lateral medullary syndrome and showed that these patients' sensory symptoms were generally described as

numbness or tightness that predominantly affected the upper limbs, especially the distal fingers. Because these patients' lesions were located in the caudal medulla and tended to extend dorsomedially, he suggested that the ipsilateral dorsal column or decussating lemniscal fibers were likely affected [13]. Cerrato et al. [14] reported a patient who developed an infarction in the right lower medulla and presented with body lateropulsion to the right, decreased vibration sense and 2-point discrimination over the right hand, and impairment of thermal and pain sensation over the left upper limb and neck. Simultaneous impairment of the lateral portion of the archiform fibers and the medial part of the spinothalamic tract was thought to be the cause of this patient's sensory disturbance [14]. Because the lesion in our case was localized to the mid-dorsal part of the left middle medulla, we suggest that numbness of the patient's left fingers was caused by impairment of the most rostral portion of the cuneate nucleus or secondary archiform fibers toward the lemniscal decussation and that dysmetria of the left hand (presumably based on his episode at onset) could be explained by a disturbance in the left cuneocerebellar tract. The cuneocerebellar tract relays impulses from the proprioceptors in the upper trunk and arm to the ipsilateral cerebellum via the inferior cerebellar peduncle, and impairment of this tract can cause dysmetria of the ipsilateral arm [10].

To our knowledge, this is the first reported case in which vestibular dysfunction was revealed by otoelectrophysiological examinations but there were no vestibular symptoms or signs except for body lateropulsion. So far, isolated vestibular nuclear infarction has been reported in 3 patients, including 2 patients described by Kim et al. [15] and 1 patient described by Kim et al. [16]; for these 3 patients, vestibular dysfunction of the affected side was demonstrated via VOG, caloric tests, and VEMP examinations. In addition, Lee et al. [17] reported 18 patients with dorsal medullary infarction and showed that only 5 out of 15 examined patients presented with ipsilesional canal paresis. Compared with the lesions of the 10 patients without canal paresis, the lesions of the 9 patients with canal paresis (8 previously reported cases (2 by Kim et al. [15], 1 by Kim et al. [16], and 5 by Lee et al. [17]) and our case) were located more laterally or more medially. Therefore, we propose that direct injury to the vestibular nucleus, which is located in the mid-dorsal part of the middle medulla, caused canal paresis in these 9 patients. Excepting body lateropulsion, it is not clear why our patient did not develop vestibular symptoms or signs despite exhibiting canal paresis, although all 18 patients reported by Lee et al. [17] presented with vestibular symptoms or signs irrespective of the presence of canal paresis.

Conclusion

We have reported an extremely rare case that involved the presentation of body lateropulsion to the lesional side and numbness of the ipsilesional fingers but no other vestibular symptoms despite canal paresis of the affected side. We propose that an extremely small lesion in the mid-dorsal part of the middle medulla caused these unique neurological manifestations.

Statement of Ethics

The patient consented to the publication of this paper with figures. This case report was approved by the institutional review board.

Disclosure Statement

The authors declare no conflicts of interest regarding the publication of this paper.

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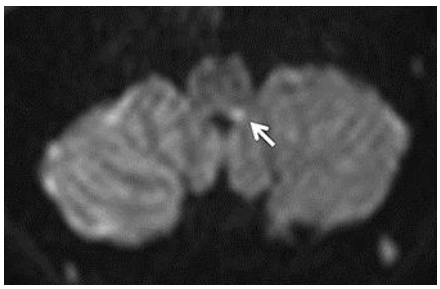

Fig. 1. On day 3, a small lesion with high-intensity signal in the dorsal part of the left middle medulla (arrow) was detected on 3-tesla diffusion-weighted magnetic resonance imaging with a section thickness of 2 mm.

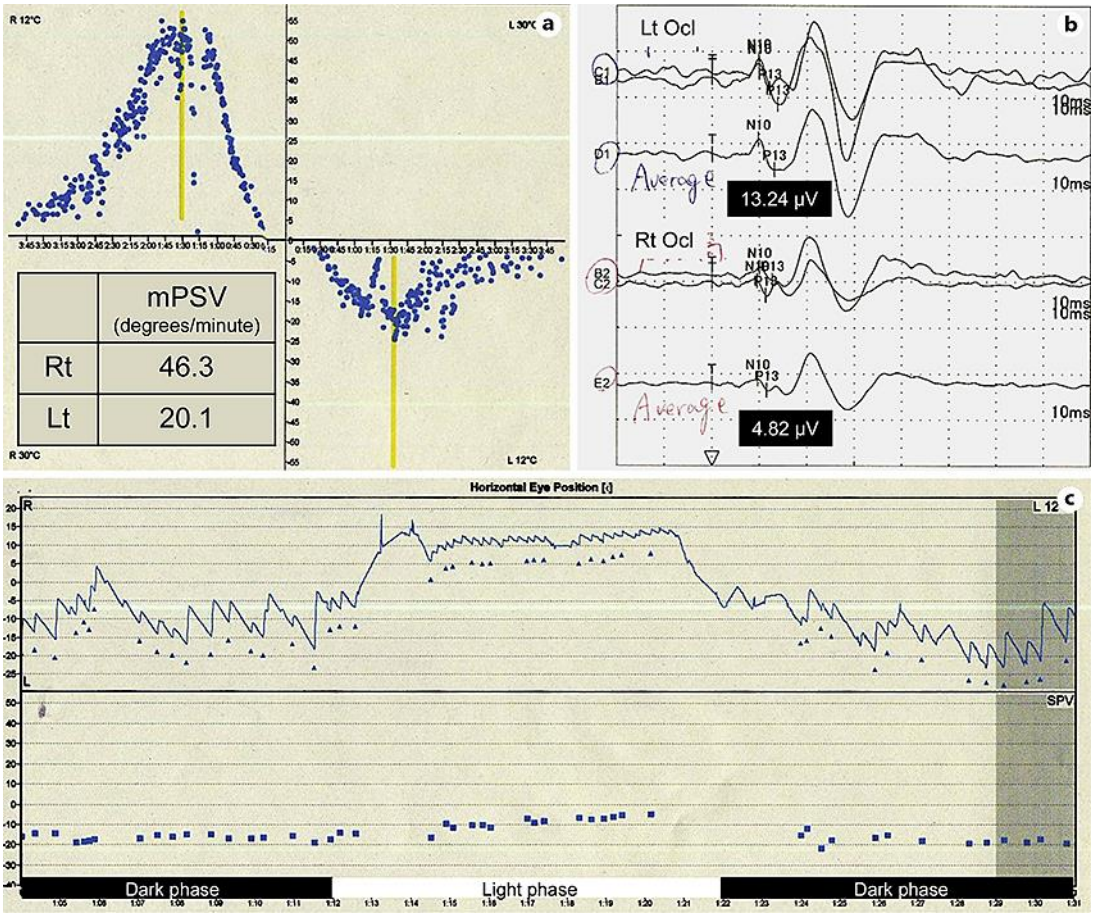

Fig. 2. **a** A cold-air (12°C) caloric test showed left canal paresis (40%). mPSV, maximum slow phase velocity; Rt, right; Lt, left. **b** Ocular vestibular-evoked myogenic potentials elicited by vibratory stimuli at the forehead showed a 64% decline in amplitude when recorded below the right eye. **c** Video-oculography recording showed visual suppression during the light phase (53%).