

Body Lateropulsion as a Presenting Symptom of Rostral Paramedian Midbrain Syndrome

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Dear Sir,

Lateropulsion of the body is the occurrence of an irresistible fall in individuals without vertigo, paresis, sensory loss, or cerebellar deficits [1]. It is a well-known clinical feature of the lateral medullary infarction, but other lesions with cerebellum, midbrain, thalamus, and pons could also result in body lateropulsion [2–6]. Body lateropulsion is usually associated with other neurological symptoms or signs, which vary according to the structures involved. Vestibular dysfunction in the roll plane of the vestibulo-ocular reflex is responsible for body lateropulsion in most cases [2]. There have been only few reports [7, 8] on body lateropulsion as a presenting symptom of rostral midbrain infarction. Furthermore, previous reports [7, 8] have not emphasized the vestibular dysfunction as a possible mechanism of body lateropulsion and did not perform a quantitative posturography test to investigate the mechanism of gait dysfunction. I present a patient who had body lateropulsion as the presenting feature of a small infarct in the rostral paramedian midbrain and discuss the possible mechanism of body lateropulsion at the level of the rostral midbrain.

Case Report

A 66-year-old woman with type 2 diabetes mellitus and hypertension developed the acute onset of severe unsteadiness. She was unable to stand unassisted and fell several times to the right side. Upon neuro-

logical examination, the day after the onset of symptoms, she fell to the right when attempting to stand with her eyes open. She could not sit without falling to the right side. She made no attempt to prevent these falls by widening her stance. In addition to falling, there was a mild limitation in eye movements with adduction and depression, and incomplete ptosis. Both pupils responded to light and accommodative stimuli. Spontaneous or gaze-evoked nystagmus was undetectable with or without Frenzel's glasses. There was no limb ataxia on finger-to-nose and heel-to-shin testing.

The muscle-stretch reflexes were normal and the plantar reflexes were flexor bilaterally. Her vibration and position senses were intact in the lower extremity. T₂-weighted and diffusion-weighted axial MRI images of the brain showed an acute small infarct in the left rostral paramedian midbrain, just dorsal to the red nucleus (fig. 1). A pure-tone audiogram and brainstem auditory-evoked potentials were normal. Five days after admission, the axial lateropulsion subsided and the patient attempted to correct gait by widening her stance and leaning to the right. A quantitative com-

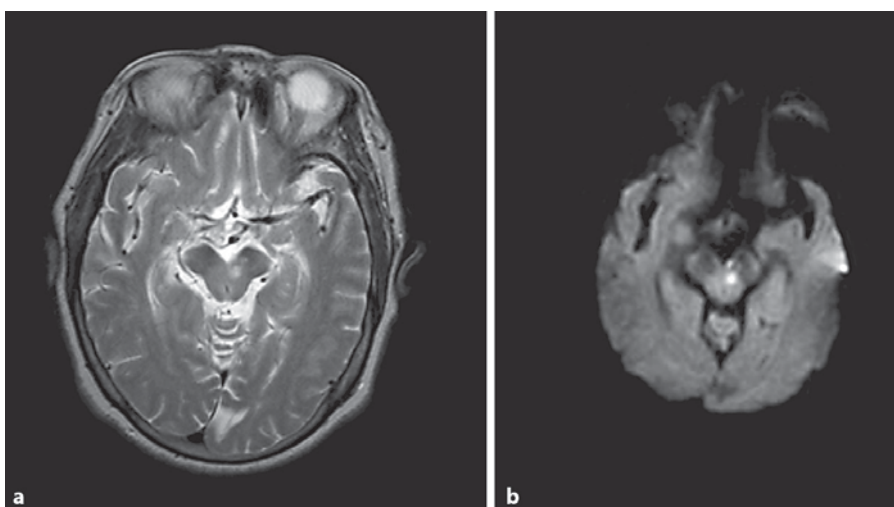


Fig. 1. Axial T₂- (a) and diffusion-weighted (b) images of the brain MRI showing an acute infarct in the left rostral paramedian midbrain, just dorsal to the red nucleus.

puterized dynamic posturography (CDP) test, performed 6 days after the onset of symptoms, showed severe vestibular deficit with falling to the right side when the visual and proprioceptive inputs were interrupted (i.e., conditions 5 and 6 of the sensory organization test) and the center of gravity alignment also tilted to the right side (fig. 2). Motor organization testing showed no abnormality. She rapidly improved within 2 weeks and at discharge, complained of only mild unsteadiness when walking.

Discussion

My patient presented with body lateropulsion due to a small rostral paramedian midbrain infarction. Although the syndrome related to rostral midbrain lesion is the most common type of the mesencephalic one of vascular origin, the classic features of rostral paramedian midbrain syndrome were eye movement disorders and alternating hemiplegia [9]. Documented unilateral paramedian lesions in the rostral midbrain that produce body lateropulsion with falling as a presenting symptom have rarely been reported. In 1990, Felice et al. [7] described a patient with small rostral paramedian midbrain infarct who showed prominent gait ataxia with falling. They postulated that interruption of the ascending fibers of the crossed dentatorubrothalamic pathway at the level of the red nucleus is most likely responsible for severe gait ataxia without tremor, limb ataxia, sensory loss, or weakness. In 2004, Karimi et al. [8] described a patient with Klippel-Feil syndrome who showed body lateropulsion due to rubrothalamic stroke, which resulted from vertebral artery dissection. In two previous reports, the direction of the body lateropulsion was to the side opposite the lesion on brain MRI as in the present case. However, previous reports did not perform a quantitative CDP test, which is essential for evaluating the mechanism of balance dysfunction. Furthermore, previous reports have not emphasized the vestibular dysfunction as a possible mechanism of body lateropulsion.

Among the symptoms of the present patient, ophthalmoplegia in the left eye may have resulted from the lesion that involved either the oculomotor fascicle or nucleus in the paramedian rostral midbrain. However, involvement of the oculo-

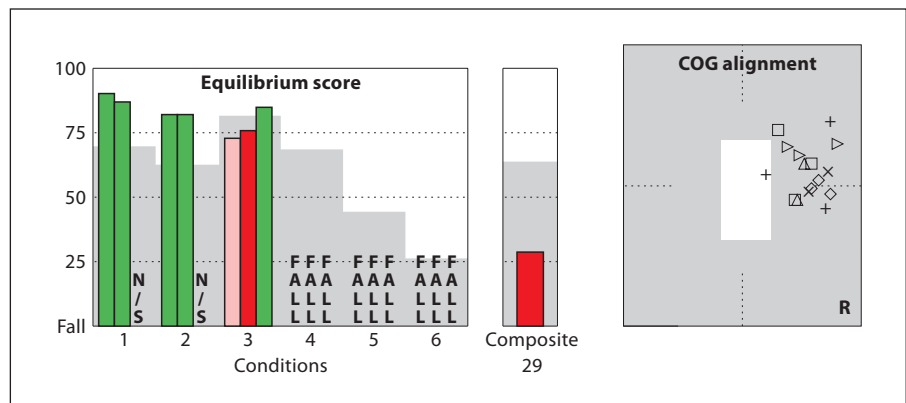


Fig. 2. Sensory organization test in CDP showed a severe vestibular deficit with falling when the visual and proprioceptive inputs were interrupted (e.g., conditions 5 and 6; sway-referenced gain: 1.0). In all conditions, center of gravity (COG) alignment consistently tilted to the right side. Conditions: (1) normal vision and fixed surface; (2) absent vision and fixed surface; (3) sway vision and fixed surface; (4) normal vision and sway surface; (5) absent vision and sway surface, and (6) sway vision and sway surface. Total body equilibrium score was only 29 (normal value: >63 at the same reference age). R = Right; N/S = not studied.

motor nucleus may be excluded in view of the unilateral ptosis.

The cardinal symptom in my patient was body lateropulsion, the tendency to fall laterally due mostly to an acute unilateral vestibular dysfunction in the context of normal motor function, sensation, coordination, and no sensation of vertigo [2]. In the rostral paramedian area, the anatomic structures most likely responsible for sudden falling were ascending fibers of the cerebellar and vestibular pathways. Like previous papers [7, 8], the ascending cerebellorubrothalamic pathway, originating from the dentate nucleus in the deep cerebellum that passes through the rostral paramedian midbrain and ends in the ventral thalamus, may explain body lateropulsion. However, the vestibular pathway is also likely associated with body lateropulsion because, in the CDP test, the patient showed severe vestibular deficit with falling when other sensory components (e.g., visual and proprioceptive inputs) were interrupted.

The ascending graviceptive input from the otoliths converges with that from the vertical semicircular canals at the level of the vestibular nuclei to subservise vestibular function in the roll and pitch planes of the vestibulo-ocular reflex. These signals are passed on to the oculomotor nuclei in the brainstem and cortical centers for control of body position and perception of verti-

cality. Damage to the graviceptive pathway ascending through the paramedian brainstem tegmentum may lead to sudden falling [4].

Without pathological confirmation, it is impossible to precisely determine the involved neural pathway(s) responsible for body lateropulsion. However, considering the result of the CDP test with severe vestibular deficit, that the ascending pathways conveying posture and gait join adjacent to the red nucleus at the level of the rostral paramedian midbrain, and that previous reports [7, 8] of body lateropulsion due to a lesion of the rostral paramedian midbrain exist, I speculated that a lesion of the ascending vestibulothalamic and/or cerebellothalamic pathways at the rostral paramedian midbrain is responsible for the observed body lateropulsion.

In conclusion, I have previously reported body lateropulsion as an isolated or predominant symptom of medullary [3], pontine [4] or rostral vermal [5, 10] infarctions. Together, these reports highlight the importance of acute, severe, gait ataxia with body lateropulsion as the presenting manifestation of vertebrobasilar ischemic stroke. Rostral paramedian midbrain syndrome should be considered in the differential diagnosis of the body lateropulsion without vertigo, sensory loss, or cerebellar deficits.

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