

Short communication

Isolated body lateropulsion caused by a lesion of the rostral vermis

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Abstract

A 66-year-old man presented with axial lateropulsion, exhibiting sudden falling as the sole clinical sign of cerebellar infarction in the territory of the medial branch of the superior cerebellar artery (mSCA). A brain MRI showed acute infarction selectively involving the centralis of the rostral vermis. This patient illustrates that cerebellar infarction in the territory of the mSCA can present as an isolated body lateropulsion. The possible mechanism of an isolated body lateropulsion is considered.

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1. Introduction

Lateropulsion of the body is the occurrence of an irresistible fall in individuals without vertigo, paresis, sensory loss, or cerebellar deficits. It is a well-known clinical feature of stroke in the posterior circulation that commonly affects the thalamus, midbrain, medulla oblongata and cerebellum [1]. 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 lateropulsion in most cases [2]. Because the rostral vermis is related to body equilibrium, gait, and postural control [3], it has been assumed to produce lateropulsion when affected. The pathology most commonly associated with lesion of the rostral vermis is a restricted form of cerebellar-cortical degeneration that occurs in chronic alcoholic patients [4]. As the rostral vermis is typically supplied by the medial branch of the superior cerebellar

artery (mSCA), however, infarction in the territory of the mSCA also can cause severe gait ataxia with sudden falling [5,6]. Sudden falling due to mSCA territory cerebellar infarction is usually associated with additional clinical signs, such as cerebellar dysarthria, limb ataxia, contrapulsion of saccade, and/or spontaneous posturing of the limbs, neck and trunk [5–8]. I present a patient who had an isolated body lateropulsion as the sole clinical sign of an infarct of the rostral vermis in the territory of the mSCA and discuss the possible mechanism related to body lateropulsion at the level of the rostral vermis.

2. Case description

A 66-year-old man with type 2 diabetes mellitus and hypertension developed the acute onset of severe unsteadiness. He was unable to stand unassisted and fell several times to the left side. Upon neurological examination the day after the onset of symptom, he fell to the left when attempting to stand with his eyes open. He could not sit without falling to the left side. He did not have dysarthria, diplopia, ophthalmoparesis, skew deviation, perceived tilting of the environment, limb weakness, or sensory loss. Spontaneous or gaze-evoked nystagmus was undetectable with or without Frenzel's glasses. There was no limb dysmetria on finger-to-

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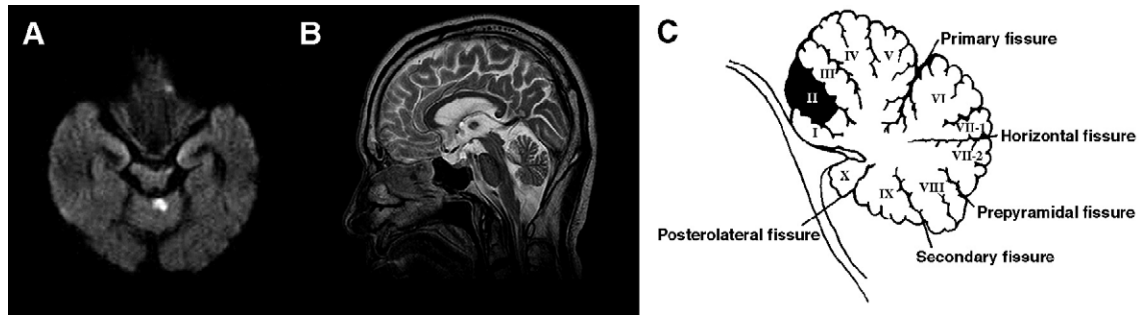


Fig. 1. Diffusion-weighted axial MRI images (A) of the brain showing an acute infarct selectively involving the rostral vermis. Sagittal T2-weighted brain MRI images (B) showing a hyperintense lesion selectively involving the centralis of the anterior lobe of the vermis. An anatomic drawing (C) of the vermal area involved in the lesion (II), as appears in the MRI of the mid-sagittal section: lingula (I), centralis (II, III), culmen (IV, V), declive (VI), folium vermis (VII-1), tuber vermis (VII-2), pyramis (VII), uvula (IX), and nodulus (X).

nose and heel-to-shin testing. The muscle stretch reflexes were normal and the plantar reflexes were flexor bilaterally. His vibration and position senses were intact in the lower extremity. Diffusion-weighted axial MRI images and T2-weighted sagittal MRI images of the brain showed an acute small infarct selectively involving the centralis of anterior lobe of the vermis (Fig. 1). A pure tone audiogram and brainstem auditory evoked potentials were normal. On quantitative electronystagmography testing, horizontal saccades, smooth pursuit, optokinetic nystagmus, and caloric responses were normal. Spontaneous or gaze-evoked nystagmus was also not detected. Five days after admission, the axial lateropulsion subsided and the patient attempted to correct his gait by widening the base of his support and leaning to the left. He rapidly improved over the next several days and at discharge, complained of only mild unsteadiness when walking.

3. Discussion

Body lateropulsion as the only clinical sign of a stroke is rare. I have identified seven previously reported cases with different kinds of lesions in the English literatures: lateral medullary infarction [9–11], infarction of the superior and inferior cerebellar peduncle [12], midbrain infarction involving red nucleus [13], and cerebellar infarction [14,15]. Cerebellar lesion commonly results in a postural instability from mild veering to severe gait ataxia with sudden falling (i.e., axial or body lateropulsion), and the other neurological symptoms or signs are usually associated with gait ataxia. I am aware of only two previous reported cases with cerebellar infarction who presented with an isolated body lateropulsion [14,15]. In 1995, Shan et al. [14] described a patient with a cerebellar infarct in the territory of the lateral branch of the posterior inferior cerebellar artery, who presented with an isolated lateropulsion of the trunk at sitting position; however, the patient could walk without assistance. In 2004, Muley and Bushara [15] reported two patients with cerebellar infarct in the territory of the mSCA who presented with an isolated severe gait ataxia with sudden falling. However, these two previous reports did not describe a

possible mechanism or a detailed account of the anatomic structure responsible for the occurrence of an isolated body lateropulsion.

The rostral vermis is particularly concerned with the control of posture, gait, and body equilibrium. It receives fibers from dorsal spinocerebellar tract (DSCT) that convey the unconscious proprioceptive information from the leg and the lower trunk. The DSCT contains ascending fibers that originate in the ipsilateral column of Clarke, travel the dorsolateral surface of the caudal medulla, enter the cerebellum through the inferior cerebellar peduncle, and terminate in the rostral and caudal portion of the ipsilateral vermis and hemisphere [16]. In the rostral vermis, the dorsal spinocerebellar fibers end in Larsell's lobules I (lingula) to IV (culmen), which is associated with the control of posture and gait in human [16]. It is well known that when cerebellar disturbance is limited to stance and gait, the lesion is most likely to be localized in the anterior part of the rostral vermis. Since many other fibers, in addition to dorsal spinocerebellar fibers, pass and end in the rostral vermis, it was not possible to draw a definite conclusion about the selective involvement of the efferent fibers of the DSCT terminating in the rostral vermis. Because my patient did not have vertigo or nystagmus, however, a lesion in the vestibular pathway passing or ending to the rostral vermis is less likely to be involved. Furthermore, although the ventral spinocerebellar tract (VSCT) also convey unconscious proprioceptive information from the leg and the lower trunk, the VSCT conveys the proprioception of the contralateral lower limb and the VSCT neurons are being more related to attempted movements rather than to pure sensory signals [17]. Considering the lesion on the brain MRI of my patient showed the selective involvement of the centralis of Larsell's lobules II, that the ascending DSCT conveying posture, gait, and muscle tone of the lower limb and the trunk terminate in the rostral vermis [16], and that previous reports of an isolated lateropulsion due to a lesion of the DSCT exist [10,11], I speculated that a lesion of the ascending DSCT at the rostral vermis in my patient is responsible for the observed lateropulsion. A recent functional MRI study, which showed that the leg movement activated area is found

within the central lobule of Larsell's lobules II–III, also supports our hypothesis [18].

In conclusion, I have previously reported an isolated lateropulsion as the sole manifestation of lateral medullary infarction [9]. These reports highlight the importance of acute, severe, isolated gait ataxia with lateropulsion as the sole manifestation of vertebrobasilar ischemic stroke. Clinicians should be aware of the possibility of infarction of the rostral vermis in patients with an isolated lateropulsion.

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