Isolated truncal contrapulsion as a rare presentation of acute thalamic infarct

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Introduction

Truncal lateropulsion is the tendency to fall laterally in the context of normal motor function, sensation, and coordination.[1] It is seen ipsilateral to lesions of the labyrinth, vestibular nerve, vestibular nuclei, and pontomedullary areas and contralateral to pontomesencephalic and thalamic lesions.[2] Thalamus is an important strategic site and infarcts involving the thalamus can yield many syndromes depending on the thalamic vascular territory involved.[3] The most common syndromes related to pure thalamic infarction include sensory syndromes, altered consciousness, cognitive disturbances, and gaze abnormalities.[3,4] Thalamic lateropulsion, a rarely reported syndrome, results from a lesion in the ventrolateral thalamic area.[3] We hereby report the case of a patient with a right thalamic infarct, presenting with an isolated truncal contrapulsion to the left side.

Case History

A 64-year-old woman with a background history of systemic hypertension and type 2 diabetes mellitus presented with acute onset of a tendency to fall to the left side. She could not even sit without support due to leftward falling. She could stand only by holding onto the side rails of her bed; however, on attempting to stand by releasing her hands, she immediately fell to the left side [Figure 1]. She had no blurring of vision, diplopia, drooping of eyelids, facial deviation, dysphagia, or dysarthria. No limb weakness/sensory symptoms were noted.

On examination, she was conscious, well oriented, with a blood pressure was 150/80 mmHg and heart rate of 78 beats/minute. Cranial nerves were normal on examination. She could not stand without support due to leftward falling and was fearful of standing [Figure 1 and Video 1]. She had no blurring of vision, diplopia, drooping of eyelids, facial deviation, dysphagia, or dysarthria. No limb weakness/sensory symptoms were noted.
and extremities was normal. The deep tendon reflexes were normal, with no pathological reflexes. Superficial sensation, joint position senses, and vibratory senses were not impaired. No limb ataxia was noted.

An electrocardiogram and complete blood counts and chemistry did not reveal any abnormal findings. Diffusion-weighted magnetic resonance imaging (MRI) sequence of the brain revealed an acute infarction in the right lateral thalamus [Figure 2]. MR angiography was normal. Findings from two-dimensional echocardiography were normal. She was started on antiplatelet, statin, and other drugs with supportive physiotherapy and gait training.

At review 1 month later, her pulsion had improved; however, she still required the support of one person to walk, with a score of 3 on modified Rankin scale.

**Discussion**

The thalamus is composed of several nuclei having multiple roles in the sensory, motor functions, behavior and in eye movement control.[5] Hence, lesions of the thalamus can result in several syndromes, depending on the nuclei affected. Truncal lateropulsion, the tendency to fall laterally in the context of normal motor function, sensation, and coordination, is rarely reported with pure thalamic infarction. Kumral et al. and Bogousslavsky et al. in their study on pure thalamic strokes have not reported even a single case of truncal contrapulsion.[3,4]

The dorsal/ventral spinocerebellar tract, the descending lateral vestibulospinal tract (LVST), the vestibulo-thalamic pathway (ascending graviceptive pathway), the dentatorubrothalamic 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.[6] Truncal lateropulsion may be seen in lesions of the lateral medulla, flocculonodular lobe, reticular formation, red nucleus of midbrain, cerebellar peduncles, or olivocerebellar fibers at the level of inferior cerebellar peduncle, which disrupt the aforementioned pathways.[5] Pulsion in thalamic lesions is believed to interrupt the projections that run from the ventrolateral nucleus of the thalamus to the medial portion of the precentral gyrus where the trunk and leg are represented.[6] The involvement of the fastigial projections through which the thalamus receives information from the vestibulocerebellum is the presumed mechanism of thalamic pulsion. Injuries of the spinocerebellar tract and/or the LVST are known to cause body lateropulsion in patients with lateral medullary infarction or cerebellar infarction. Thömke et al. 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.[7] Truncal lateropulsion without limb ataxia reflects an impairment of vestibulospinal postural control caused by a lesion of the descending lateral vestibulospinal tract, whereas body lateropulsion with limb ataxia reflects the consequence of impaired or absent proprioceptive information caused by a lesion of the ascending dorsal spinocerebellar tract. The lateral vestibulospinal tract is considered to play an important role in the maintenance of posture by exerting strong excitatory influences on extensor muscles and inhibitory influences on flexor muscles.[8] Thus, interruption of the lateral vestibulospinal tract decreases extensor muscle tone of the trunk and lower limb on the side of the lesion, which is likely to cause ipsilateral axial lateropulsion.

Masdeu et al., in their study, describe six patients with thalamic infarcts, who developed inability to stand, in the absence of motor weakness.[9] Saiki et al. have described two patients with thalamic infarcts who had falls toward the contralateral side.[10] Our patient had a truncal contrapulsion resulting from a lateral thalamic infarct.

We would like to conclude that isolated truncal contrapulsion can be a very rare manifestation of an isolated thalamic infarct.

**Declaration of patient consent**
The authors certify that appropriate patient consent was obtained.

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**Conflicts of interest**
There are no conflicts of interest.

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