

Isolated axial lateropulsion with ipsilesional subjective visual vertical tilt in caudal lateral medullary infarction

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Abstract. The isolated body lateropulsion has been well recognized in caudal lateral medullary infarction and postulated to result from the involvement of ipsilateral dorsal spinocerebellar tract which is known to convey proprioception of trunk and legs. However, there has been no case accompanied by the tilt of the subjective visual vertical in caudal lateral medullary infarction. Recently, it has been suggested that a lesion in the ipsilateral graviceptive vestibulothalamic pathway can lead to alteration of subjective visual vertical without ocular tilt reaction in various brainstem lesions. Here we describe two cases of caudal lateral medullary infarction with ipsilesional body lateropulsion and subjective visual vertical tilt but without limb ataxia or ocular tilt reaction. It could be hypothesized that the ascending graviceptive information from the spinal cord may run adjacent to the dorsal spinocerebellar tract or perception of the visual vertical can be influenced by ascending spinal proprioception.

Keywords: Lateropulsion, subjective visual vertical, dorsal spinocerebellar tract, vestibulothalamic tract

1. Introduction

The axial lateropulsion is an irresistible falling to one side and has been well described as a symptom of classic lateral medullary infarction (LMI) [11]. In classic LMI, axial lateropulsion is ascribed to the involvement of vestibulo-spinal or vestibulo-ocular reflex (VOR) pathway and is usually accompanied by ocular tilt reaction (OTR). However, isolated axial lateropulsion without any sign of OTR has been well described in caudal lateral medullary infarction (CLMI) [12,14,17]. The dorsal spinocerebellar tract (DSCT) is at the lateral surface of the caudal medulla and is reported to be responsible for the isolated axial lateropulsion without limb ataxia in CLMI [17]. The

DSCT conveys proprioception of body and lower limb but is not known to be associated with VOR pathway.

Altered subjective visual vertical (SVV) has been thought to be associated with crossed graviceptive pathway which interacts with VOR pathway. But recent studies suggest an ipsilateral graviceptive pathway which is independent of VOR pathway [5,18,21].

So far, however, there has been no report on association of the DSCT with the SVV tilt. We present two patients with axial lateropulsion and ipsilesionally tilted SVV but without ocular torsion and head tilt who had acute ischemic lesions in lateral surface of caudal medulla where the DSCT lies in.

2. Case description

2.1. Case 1

A 74-year-old male visited ER with complaining of inability to maintain balance in standing. He said he

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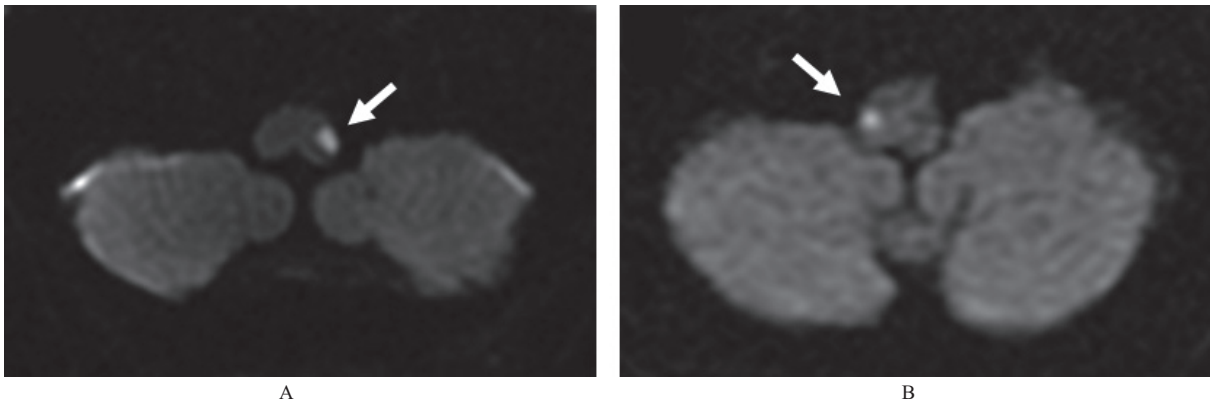


Fig. 1. Diffusion-weighted MR image of case 1 (A) and case 2 (B) showing the acute infarction at the lateral surface of caudal medulla (arrows).

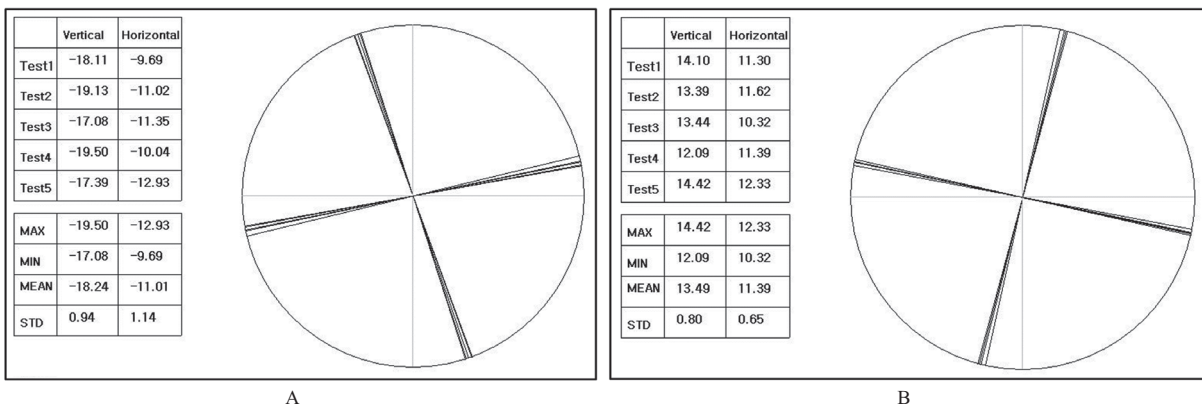


Fig. 2. Result of subjective visual vertical in case 1 (A) and case 2 (B). The mean tilt angle was -18.2° in case 1 and $+13.5^\circ$ in case 2. —, Counterclockwise.

felt transient dizziness for a short time but it soon disappeared. He had no history of hypertension, diabetes, or dyslipidemia. But he was a current smoker with history of 50 pack-years. He noted no other symptoms of dysphagia, dysarthria, vertigo, diplopia, sensory abnormality, weakness, or incoordination. On initial neurologic examination, he was unable to sit straight and fell to the left side. Nystagmus, OTR or limb ataxia was not observed. Brain MRI showed acute infarction involving the left lateral surface of the caudal medulla (Fig. 1A). On test for SVV on the next day, his SVV tilted 18 degree to the left (Fig. 2A). Fundus photography taken 6 days after symptom onset depicted no ocular torsion (right 4.0° extorsion, left 3.3° extorsion). Left falling subsided in 4 weeks.

2.2. Case 2

64-year-old man with hypertension, diabetes mellitus, and chronic renal failure awoke with dysequi-

librium. He was unable to keep standing. On neurologic examination, he had marked body lateropulsion to the right, but abnormality of cranial nerves, weakness, paresthesia, or cerebellar ataxia was absent. Brain MRI depicted right caudal lateral medullary infarction (Fig. 1B). MRA showed a tortuous right vertebral artery with mild distal stenosis. SVV test performed 3 days after symptom onset showed 13.5 degree deviation to the right (Fig. 2B) without ocular torsion on fundus photography taken 4 days after symptom onset (Fig. 3) or skew deviation. He was able to stand without support a week later.

3. Discussion

Our patients presented with axial lateropulsion and SVV tilt without other vestibular symptom such as vertigo, ocular torsion, head tilt, skew deviation or cerebellar dysmetria and without any motor or sensory

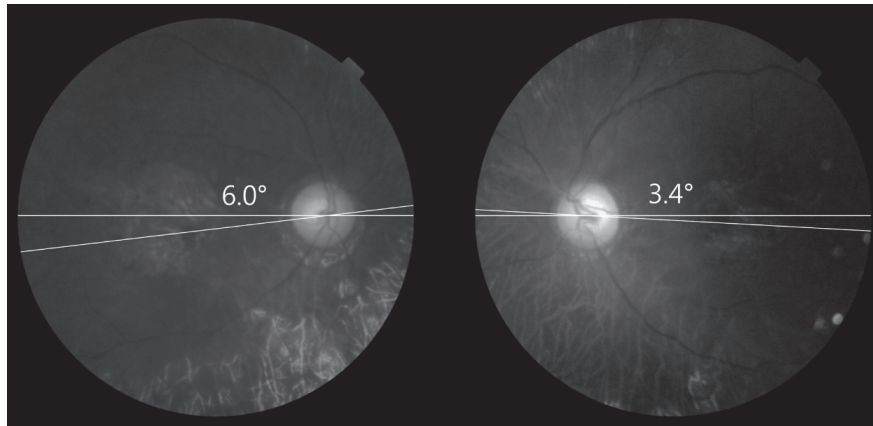


Fig. 3. Fundus photography of Case 2. Ocular torsion is not observed.

function abnormality. Bedside neuro-otologic examinations including head impulse, head shaking, and positional tests didn't show any abnormal findings. Laboratory vestibular function tests including caloric test, rotation chair test, cervical VEMP test were all normal except for ipsilesional tilt of SVV. In both cases, brain MRI showed a small acute ischemic lesion at the lateral surface of caudal medulla on the same side of lateropulsion (Fig. 1). MRA showed stenosis of relevant ipsilesional vertebral artery in both patients.

Brandt and Dietrich reported that prevalence of pathologic deviation of the SVV from the true vertical is as high as 94% in patients with lateral medullary infarction [7]. They found that the direction of SVV tilt was concordant with ocular torsion, but angle of SVV tilt and ocular torsion did not match. They insisted that a vestibular nuclei complex lesion may be responsible for SVV tilt and ocular torsion.

SVV tilt can be caused by a lesion in the crossed graviceptive pathway from the vestibular nuclei through the medial longitudinal fasciculus (MLF) to the contralateral interstitial nucleus of Cajal and posterolateral thalamus [7]. Head tilt, ocular torsion, skew deviation (OTR) can be accompanied with the crossed graviceptive pathway.

Recently, the existence of uncrossed vestibulothalamic pathway for perception of verticality has been reported [5,18,21]. This uncrossed ipsilateral vestibulothalamic tract (IVTT) runs through the medial edge of the medial lemniscus. A lesion in this tract does not produce oculomotor abnormality as the tract bypasses the oculomotor system while a lesion in the crossed graviceptive pathway can cause OTR. Because the IVTT is an ipsilateral pathway, the SVV tilt is toward the lesional side. On the contrary, a crossed pathway

would produce contralateral SVV tilt if the involved lesion is above the decussation at upper medulla [18] or lower pons [8,20].

The isolated axial lateropulsion in CLMI has been well documented in previous reports [1,12,17]. Thömke et al. identified the location of the DSCT and the lateral vestibulospinal tract (LVST) in medulla oblongata as a responsible lesion for axial lateropulsion in patients with ataxia and without ataxia respectively [17]. According to Thömke, DSCT is the more convincing structure for explaining the lateropulsion of our patients as our patients did not have ataxia and the LVST runs more ventromedially than the lesions of our patients.

Cases of lateropulsion with SVV tilt to the ipsilateral side [1,7,10,20] or the contralateral side [15] were reported previously. Lesions in lateral medulla [13], paramedian pons [20], cerebellum [16] was associated with their symptoms. In other studies, the lateropulsion without limb ataxia was reported to result from the DSCT involvement [12,14,17]. But SVV tilt or ocular torsion in patients with DSCT involvement without vestibular nuclei complex involvement has not been reported.

In brain MRIs of our patients, the lesion was located at the lateral surface of caudal medulla oblongata where the dorsal spinocerebellar tract and the ventral spinocerebellar tract (VSCT) run.

The DSCT originates from the ipsilateral column of Clarke, runs along the dorsolateral surface of caudal medulla, enters the cerebellum through the inferior cerebellar peduncle, and terminates in the ipsilateral anterior lobe and paramedian lobule [6,9]. The column of Clarke receives fibers from muscle spindles and Golgi tendon organs of ipsilateral trunk and

lower limb. The DSCT is known to convey unconscious proprioception. The VSCT arising from lower thoracic and lumbosacral segments decussates at the level of the origin, and recrosses as it enters the cerebellum through superior cerebellar peduncle. The ventral spinocerebellar neurons seem to encode motor signals derived from descending pathway in addition to proprioception. In our cases, it is less likely that the VSCT is the responsible structure because it runs along the contralateral side at the level of medulla and its function is more related to descending motor pathway [6].

We believe that this is the first report of cases with lateropulsion and SVV tilt but no ocular torsion in DSCT lesion. As there was no such case, we present three possible mechanisms here. First, there could be a pathway that contributes to the graviceptive system other than proprioception in DSCT. Although there is no reliable evidence that the DSCT conveys graviceptive information other than proprioception, this hypothesis can explain the SVV tilt of our patients. Furthermore, the absence of oculomotor abnormality implies that the involved graviceptive system in our patients may be IVTT rather than crossed graviceptive pathway. Second, it is possible that dorsal spinocerebellar proprioception affects SVV tilt. As maintaining balance is delicate mechanism combining vestibular, visual, and somatosensory system, it can be told that dorsal spinocerebellar proprioception may contribute to graviceptive system. It is known that the tilt of head or body can influence SVV tilt [4]. At intermediate body tilt, overcompensation of subjective vertical is observed (E-effect). But at larger angle tilt, undercompensation occurs and subjective vertical is tilted toward the direction of body roll (A-effect). Modification of these effects by somatosensory dysfunction was described in a patient after complete proprioceptive sensory loss due to polyneuropathy [19] or in patients with central hemianesthesia [2,3]. SVV deviation toward hypesthetic side even in the upright position was observed in central hemianesthesia [2]. These studies support that SVV may be influenced by proprioceptive information. And lastly, the vestibular nuclei complex could be involved in our patients. Although the brain MRI did not demonstrated any other lesion than a lesion at lateral surface of caudal medulla, there could be undetected small lesion between MRI slices.

In conclusion, we described two patients with DSCT lesion who presented with axial ipsipulsion and ipsilateral SVV tilt but no ocular torsion. The findings of this study suggest that the DSCT may convey graviceptive

information or proprioception may have an influence on SVV tilt. A further study for elucidating reasonable basis for our proposed mechanism is suggested.

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