CASE REPORT

Ocular and Truncal Lateropulsion Associated with Caudal Lateral Medullary InfarctionShe-Qing Zhang,¹ Ming-Yuan Liu,² Xiao-Long Ma² & Hui-Min Zheng¹¹ Department of Neurology, Changhai Hospital, Shanghai, China² Department of Neuroimaging, Changhai Hospital, Shanghai, China**Correspondence**

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Received 30 October 2011; revision 13

November 2011; accepted 14 November 2011

doi: 10.1111/j.1755-5949.2011.00284.x

Ocular lateropulsion, referring to ocular motor bias toward (ipsipulsion) or away from (contrapulsion) the lesion side without limitation of eye movement, is uncommon [1]. Truncal lateropulsion, a compelling sensation of being pulled toward the side of lesion, is a well-known transient feature of dorsal lateral medullary infarction (LMI) [2]; and isolated truncal lateropulsion is attributed to lesions in the flocculonodular lobe [3], reticular formation [4], red nucleus of midbrain [5], cerebellar peduncles,

or olivocerebellar fibers at the level of inferior cerebellar peduncle [6]. However, persistent and simultaneous ocular and truncal lateropulsion associated with caudal LMI has not been reported previously, to our knowledge.

A 54-year-old man developed acute onset of dizziness while he was watching TV. He had no nausea, diplopia, tinnitus, and deafness. One hour later, he felt the dizziness becoming more severe and the left-side limbs and trunk becoming numb.

Figure 1 Right pupil is smaller than the left one in dark environment, but both ocular rimae are equal (upper). Severe right lateropulsion on admission (lower left); By slightly pulling his garment, one could support the patient to walk almost normally after one-month treatment (lower middle); MRA shows right vertebral dysplasia and stenosis (lower right).

Figure 2 MR T2W Images of sequential medullary sections showing normal signals at the upper medulla (top **A** and **B**) and hypersignals (arrows) in the periphery of caudal lateral funiculus of medulla (top **C** and **D**). MR DW Images of sequential medullary sections showing normal signals at the upper medulla (bottom **A** and **B**) and hypersignals (arrows) in the periphery of caudal lateral funiculus of medulla (bottom **C** and **D**).

Although feeling giddy, he was still able to walk by a cane. Three hours after the onset, he began vomiting. On the following day, he experienced mild dysphagia and severe hiccup. He had no hypertension, cardiac vascular disease, and diabetes mellitus previously.

Neurological examination showed mild horizontal, sometimes rotatory, nystagmus. His eyes were gazing (biased) to the right at most times, but they were able to move to the left or right, up or down freely, with the target movement. Both of the ocular rimae were the same width. The pupils were equal in bright environment, but the left pupil was apparently larger than the right one in dark environment (Figure 1, top). Both pupils were sensitive to light. The right palate is mildly paraparetic and the right throat reflex is mildly decreased. The tongue moved well and there was no facial weakness. Pain and heat sensation was decreased drastically in the left trunk and limbs, but mildly in the right face. Bilateral corneal reflexes were almost equally sensitive. Bilateral vibration and joint position sensation was preserved. Finger–nose test and the heel–knee–shin test on both sides were surprisingly coordinated when examined on the bed. When standing uprightly without closing the eyes, he suffered severe lateropulsion to the right. He would certainly fall down if not supported by another person (Figure 1, left). There was no weakness of the limbs and Babinski sign on both sides was negative.

Cerebral MRI detected a lacunar infarction in the right mid-lateral periphery of the caudal medulla, with preservation of the upper medulla, inferior cerebellar peduncle and dorsal funiculus (Figure 2). Angiographic sequences disclosed whole length dysplasia and stenosis of the right vertebral artery. (Figure 1, right).

One week after treatment, the symptoms of dysphagia, hiccup, and hypesthesia in the left limbs improved quickly, but the nystagmus worsened and oscilopsia appeared. The latter symptoms lasted for one week. The ocular and truncal lateropulsion still remained. After one-month treatment, the patient could walk almost normally, if his garment was slightly pulled from the left side by another person (Figure 1, middle).

Caudal LMI is less common than rostral LMI. The symptoms of upper LMI are rather different from those of lower LMI. For upper LMI, patients often manifest severe vertigo, nystagmus, limb ataxia, crossed superficial sensory defect accompanied by decreased corneal reflex, Horner sign, even dysphagia and/or dysarthria. However, at the level of lower medulla, the inferior cerebellar peduncle has not formed and the vestibular nucleus has not appeared.

The most prominent feature in the present patient is the long-lasting ocular and truncal lateropulsion. Ocular ipsipulsion has been reported in Wallenberg syndrome by Kim et al. [1], and is ascribed to damage to fibers that project from the contralesional

inferior olivary nucleus via the inferior cerebellar peduncle to the Purkinje cells in the cerebellar cortex. Disinhibition of the Purkinje cells leads to decreased activity of the ipsilateral fastigial nucleus in the vermis. The fastigial nucleus projects, via the uncinate fasciculus in the superior cerebellar peduncle, upon the medial and superior vestibular nuclei and the paramedian pontine reticular formation of the contralateral side and accelerates contralateral saccades. Its inhibition results in a motor bias of horizontal eye movements toward the side of the lesion [7,8]. In our patient, the ocular ipsilateral pulsion did occur, and we thought it was gazing palsy at first; however, the eyes could move freely in all directions. The upper medulla, including inferior olivary nucleus and inferior cerebellar peduncle, was unaffected. Therefore, there must be another mechanism lying behind this phenomenon, which has not been elucidated as yet.

As for the long lasting lateropulsion, we speculate it was caused by the lesion of the right spinocerebellar tracts. Using three-dimensional brainstem mapping, Thomke et al. recently found that involvement of the ascending posterior spinocerebellar tract was associated with lateropulsion with limb ataxia; but the involvement of the descending lateral vestibulospinal tract was associated with lateropulsion without limb ataxia [9]. We cannot analogize that the lateropulsion in our patient is associated with lateral vestibulospinal tract involvement, although the patient manifested lateropulsion without limb ataxia. Because the lesion in the present patient was situated in the periphery of the midlateral medulla, it may damage the spinothalamic tract (the reason for the patient's transient sensory defect on the opposite limbs), rather than the lateral vestibulospinal tract, which descends more ventromedially.

There may be another mechanism concerning the lateropulsion in our case. The fact, that the patient's left pupil was smaller than the right one in dark environment, actually indicated that the right medullary reticular formation had been involved, at least in part. Therefore, the medullary (lateral) reticulospinal tract, which derives from the medial two-third of the reticular formation and descends in the anterior part of the lateral funiculus in lower medulla, may be damaged. The tract can influence muscle tone by acting upon γ motor neurons. The extensor muscle tone will be inhibited if this tract is damaged [10]. This mechanism may, in part, explain the lateropulsion in our case.

In conclusion, persistent ocular and truncal lateropulsion may be associated with the caudal LMI.

Conflict of Interest

The authors declare no conflict of interest.

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