eNeurologicalSci 7 (2017) 25–26

Contents lists available at ScienceDirect

eNeurologicalSci

journal homepage: http://ees.elsevier.com/ensci/

Letter to the Editor

Isolated body lateropulsion caused by lower lateral medullary infarction

Keywords:
Isolated body lateropulsion
Lateral medullary infarction
Magnetic resonance imaging
Cerebral infarction

Dear Editor,

Body lateropulsion (BL) is a symptom of lateral medullary syndrome, also known as Wallenberg syndrome. It is usually associated with ipsilateral Horner syndrome, ipsilateral limb ataxia, superficial sensory disturbance of the ipsilateral face and the contralateral limbs, dysarthria, dysphagia, and vertigo [1]. Here we present two cases of ipsilateral body lateropulsion as an initial symptom of lower lateral medullary infarction and review the existing literature regarding isolated body lateropulsion (iBL) caused by lateral medullary infarction. A brief description on the Case 1 has been published in Japanese [2].

1. Case reports

Case 1. A 64-year-old woman noticed a tendency to fall to the left upon standing. She had hypertension, diabetes mellitus, and hyperlipidaemia. She denied having vertigo, diplopia, hiccup, dysphagia, speech disturbances, numbness, or muscle weakness. Neurological findings were unremarkable except for body lateropulsion to the left. Brain magnetic resonance imaging (MRI) showed a small infarction in the left lower part of the medulla (Fig. 1a). She was diagnosed with acute atherothrombotic cerebral infarction, and antiplatelet therapy was started. She no longer swayed on her fifth day at the hospital.

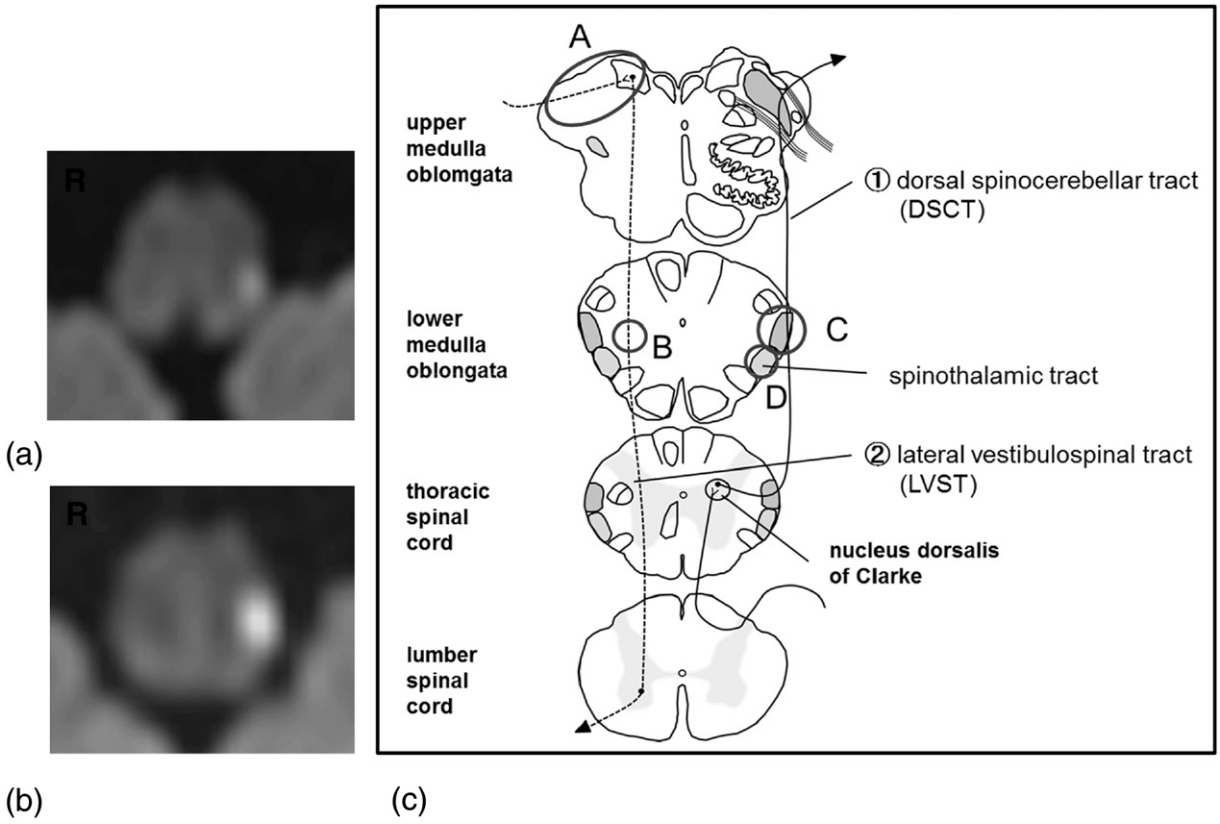

Fig. 1. Diffusion-weighted magnetic resonance imaging for Cases 1(a) and 2(b) Anatomical map related to body posture and its stability (c).

http://dx.doi.org/10.1016/j.ensci.2017.03.004

Case 2. A 67-year-old man presented with a sudden inability to stand and tendency to fall to his left. He noticed no other symptoms, such as vertigo, diplopia, dysarthria, paraesthesia, numbness, weakness, or incoordination. On examination, the sole abnormality found was that he leaned to the left and fell down upon standing without support. Decreased pain and temperature sensation in the right side of the body developed 3 days after the onset of the initial symptoms. Brain MRI showed a small infarct in the left lower part of the lateral medulla (Fig. 1b). The patient was diagnosed as having acute atherothrombotic cerebral infarction, and antiplatelet therapy was started.

2. Discussion

Since iBL is accompanied by no other symptoms, it may often be overlooked unless the physicians recognise it as a manifestation of an organic disorder. The 2 cases of iBL presented here were confirmed to have lesions in the lower lateral medulla by MRI. Patients with BL have been reported to have lesions in the dorsal spinocerebellar tract (DSCT), [3,4] the descending lateral vestibulospinal tract (LVST), the vestibulo-thalamic pathway (ascending graviceptive pathway), [5] the dentatorubrothalamic pathway, or the thalamocortical fascicle [6]. The DSCT and the LVST may be impaired by infarction in the medulla oblongata. Lesions leading to Wallenberg syndrome are located in the upper lateral medulla oblongata (A in Fig. 1c). These types of lesions involve many nuclei and neural pathways, and lead to many neurological symptoms, such as vertigo, diplopia, and nystagmus, as well as lateropulsion. Lesions in the lower medulla oblongata also lead to iBL (B or C in Fig. 1c), as demonstrated by our 2 cases, wherein no nuclei or neural pathways other than the DSCT and LVST were involved at this level. The reason for the appearance of contralateral sensory disturbances Case 2 was that the lesion expanded to involve the spinothalamic tract, which is located near the DSCT and the LVST (D in Fig. 1c).

There are 19 cases of infarction in the lower medulla oblongata leading to an initial symptom of iBL described in the literature, including our 2 cases (14 men and 5 women, mean age [± standard deviation] of 65 ± 14 years) [2]. The patients in these cases are significantly older than those with Wallenberg syndrome (57 ± 8 years old, $P < 0.01$) [1]. This may be because all cases of iBL due to lower lateral medullary lesions were due to atherosclerosis, while the cases of Wallenberg syndrome included juvenile patients with vertebral artery dissection. Direction of lateropulsion was ipsilateral to the lesion side in all cases. Ipsiversive lateropulsion occurs with medullary lesions involving either the DSCT or the LVST. Spinothalamic sensory deficits appeared a few days after the onset of lateropulsion (third to sixth disease day) in 11 cases (58%), including Case 2 in our report. In clinical practice, this combination strongly suggests a lesion in the lower lateral medulla.

The present study raises the possibility that iBL is attributable to lesions in the lower lateral medulla oblongata.

Author contribution statement

The authors contributed to and approved the final manuscript.

Disclosures

The authors report no disclosures.

Conflicts of interest

The authors declare that they have no conflict of interest.

References

[1] J.S. Kim, J.H. Lee, D.C. Suh, et al., Spectrum of lateral medullary syndrome. Correlation between clinical findings and magnetic resonance imaging in 33 subjects, Stroke 25 (1994) 1405–1410.
[2] Y. Nakazato, N. Tamura, K. Ikeda, et al., Neuroanatomy of isolated body lateropulsion (in Japanese), Brain Nerve 68 (2016) 263–270.
[3] M.C. Smith, The anatomy of the spinocerebellar fiber in man. I. The course of the fibers in the spinal cord and brain stem, J Comp Neurol 108 (1957) 285–352.
[4] M.C. Smith, The anatomy of the spinocerebellar fiber in man. II. The distribution of the fibers in the cerebellum, J Comp Neurol 117 (1961) 329–354.
[5] A. Zwergal, J. Büttner-Ennever, T. Brandt, et al., An ipsilateral vestibulothalamic tract adjacent to the medial lemniscus in humans, Brain 131 (2008) 2928–2935.
[6] M. Dieterich, T. Brandt, Thalamic infarctions: differential effects on vestibular function in the roll plane (35 patients), Neurology 43 (1993) 1732–1740.

Yoshihiko Nakazato*
Naotoshi Tamura
Kei Ikeda
Toshimasa Yamamoto
Department of Neurology, Saitama Medical University, Japan
*Corresponding author at: Department of Neurology, Saitama Medical University, 38 Morohongo Moroyama, Iruma-gun, Saitama 350-0495, Japan.
E-mail addresses: nakachan@saitama-med.ac.jp (Y. Nakazato), tamuran@saitama-med.ac.jp (N. Tamura), k_ikeda@saitama-med.ac.jp (K. Ikeda), ytoshi@saitama-med.ac.jp (T. Yamamoto).

9 March 2017