Isolated axial lateropulsion caused by an acute lateral medullary infarction involving the dorsal spinocerebellar tract: A case report

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Abstract:
Lateral medullary syndrome encompasses a broad spectrum of symptoms and signs depending on the bulbar localization of the lesion. Body lateropulsion (BL) can occur without vestibular and cerebellar symptoms, as a unique manifestation of a lateral medullary infarction. However, it is relatively rare and challenging to diagnose. We report a case of a 72-year-old woman who presented with a tendency to fall to the right. She denied having vertigo, cerebellar signs, sensory loss, or motor weakness. No signs of vestibular dysfunction were found on the ENT examination. Neurological evaluation was unremarkable, except for mild ataxia of the right limbs along with BL to the right side when standing and walking. Brain magnetic resonance (MR) imaging showed an acute small infarct in the right lateral aspect of the medulla extending from the rostral to the caudal level. MR angiography found no stenosis or vascular occlusions. We believe that ipsilateral axial lateropulsion shown by our patient may be related to a selective ischemic lesion of the dorsal spinocerebellar tract in its medullary course. A lateral medullary infarction should be seriously considered in patients who present with isolated BL without further signs of bulbar involvement.

Keywords:
Isolated body lateropulsion, medulla oblongata, posterior spinocerebellar tract

Introduction
Lateral medullary syndrome (LMS) is a neurological condition caused by an acute ischemic infarction in the lateral segment of the medulla, posterior to the inferior olivary nucleus. Body lateropulsion (BL), i.e., the phenomenon of tilting the body to one side when standing, may rarely be the initial or unique symptom of LMS, a syndrome that includes a variety of symptoms depending on the extent of the lesion. In this study, we report a case of isolated BL (IBL) associated with an acute ischemic infarction in the lateral aspect of the medulla extending from the rostral to the caudal level.

Case Report
A 72-year-old woman presented with a tendency to fall to the right while standing and walking, 4 days after onset. She had a history of hypertension, diverticulosis of the colon, chronic cholecystitis, and renal lithiasis. She did not complain of vertigo, diplopia, hiccups, dysphagia, speech disturbances, tinnitus, hearing loss, numbness, or muscle weakness. No signs of vestibular dysfunction were found on the ENT examination: the patient was able to sit on the side of the bed without support, the head was not tilted, had no lateral eye

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deviations with short lid closure or lateral saccade, and had no skew deviation during the cross-cover test when fixing, and subjective visual vertical test was normal (angle of deviation from vertical = 1.0°). Except for a standing and walking right BL along with a slight right dysmetria in the finger–nose and heel–knee–shin test, neurological evaluation was unremarkable. Laboratory tests at admission found a hyposideremia (31 µg/dl) and an increase in C-reactive protein (4.9 mg/dl) and erythrocyte sedimentation rate (113 mm/h). Hgb was 10.6 g/dl and Hct – 31.0%. Brain magnetic resonance (MR) with diffusion-weighted imaging (DWI) showed an acute small infarct in the right lateral aspect of the medulla, extending from the rostral to the caudal level of the medulla [Figure 1]. T2 axial image analysis revealed a conjugate ocular deviation to the right of 14°, calculated by a method previously reported. MR angiography (MRA) found no stenosis or vascular occlusions [Figure 1]. The echocardiogram (ECG) was normal while the transthoracic ECG found only a parietal hypertrophy of the left ventricle. Carotid ultrasound revealed a plaque in the left and right internal carotid artery, causing lumen stenosis of 25% and 10% according to the NASCET method, respectively. No occlusions or atherosclerotic plaques were found in the extracranial vertebral arteries. The patient was started with antiplatelet therapy (aspirin at 100 mg/day) and discharged with a diagnosis of acute lacunar cerebral infarction. Complete remission of the BL was observed over the subsequent few weeks.

Discussion

In the present work, we described a rare case of a small infarction in the lateral aspect of the medulla causing an ipsiversive IBL. BL may be a transient feature of LMS, a syndrome that includes a broad spectrum of clinical symptoms and signs based on the rostro-caudal and horizontal location of the lesion. IBL, i.e., BL without other signs of cerebellar or bulbar involvement, has been described in limited case reports where it has been associated with lesions involving the cerebellum, cerebellar peduncles, red nucleus, and medulla. In a few other cases caused by a bulbar lesion, IBL has been reported as an initial symptom of LMS. In all cases of BL of bulbar origin, the lesion was found in the ipsilateral part of the medulla.

The laterality of BL in brain stem strokes has been shown to depend on the pathways involved and the location of the lesions. Ipsiversive BL has been related to medullar lesions involving either the descending lateral vestibulospinal tract (LVST) or the ascending dorsal spinocerebellar tract (DSCT). Recently, Thömke, using a three-dimensional brainstem mapping, found that BL without limb ataxia may be attributed to an impaired vestibulospinal posture control caused by a lesion of the LVST, whereas BL with limb ataxia is probably related to a lesion of the DSCT, which carries unconscious proprioceptive stimuli from the ipsilateral lower trunk and leg. Contraversive BL, on the other hand, occurs with infarcts involving the medial longitudinal fasciculus in the upper pons or the red nucleus in the midbrain. MRI lesions involving the medulla and associated with LMS have been classified rostrocaudally as rostral, middle and caudal. Since the ascending DCST is localized on the lateral surface of the lower medulla, it is believed that the lesion of this spinal tract is the cause of the ipsiversive BL in all these cases.

DWI is a commonly performed MRI sequence for the evaluation of acute ischemic stroke and is very
sensitive in the detection of small and early infarcts. In our case, DWI showed an acute small infarct in the right lateral aspect of the medulla extending from the rostral to the caudal level [Figure 1]. In addition, T2 axial image analysis revealed an ipsilesional conjugate eye deviation, commonly associated with acute lateral medullary infarction. Therefore, the slight degree of ocular deviation (14°) found probably depends on the small size of the infarct lesion which did not allow to damage the olivary projections to the contralateral vestibulocerebellar structures.

Because DSCT carries major proprioceptive information ipsilaterally in the medulla and may be affected at any level during its course to the cerebellum, we believe that a lesion of this descending tract in its entire path through the medulla, as not previously demonstrated, could be responsible of the ipsiversive IBL of our patient. Furthermore, the absence of vestibular symptoms, the ipsiversive type of IBL, as well as the mild ipsilesional limb ataxia, confirm in our case the exclusive involvement of the DCST. In fact the absence of signs of vestibular dysfunction excludes the damage of the LVST, while the presence of a mild limb ataxia ipsilateral to the BL makes the involvement of the ventral spinocerebellar tract very unlikely, as this tract carries the proprioceptive sensitivity of the contralateral lower limb.

In summary, lateral medullary infarction should be seriously considered in patients who present with IBL. The absence of vestibular symptoms together with the neuroradiological finding of a small lesion in the lateral aspect of the medulla, homolaterally to the IBL, is indicative of a DSCT lesion.

**Declaration of patient consent**
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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

**References**

1. Wallenberg A. Acute bulbar affection (Embolie der art. cerebellar post. inf. sinistra?) Arch Psychiatr Nervenkr 1895;27:504-40.
2. Kim JS. Pure lateral medullary infarction: Clinical-radiological correlation of 130 acute, consecutive patients. Brain 2003;126:1864-72.
3. Ramaswamy S, Rosso M, Levine SR. Body lateropulsion in stroke: Case report and systematic review of stroke topography and outcome. J Stroke Cerebrovasc Dis 2021;30:105680.
4. Lee H, Sohn CH. Axial lateropulsion as a sole manifestation of lateral medullary infarction: A clinical variant related to rostral-dorsolateral lesion. Neurol Res 2002;24:773-4.
5. Kim SH, Cho J, Cho JH, Han SW, Kim SM, Park SC, et al. Isolated lateropulsion by a lesion of the dorsal spinocerebellar tract. Cerebrovasc Dis 2004;18:344-5.
6. Maeda K, Saikyo M, Mukose A, Tomimatsu H, Yasuda H. Lateropulsion due to a lesion of the dorsal spinocerebellar tract. Intern Med 2005;44:1295-7.
7. Nakazato Y, Tamura N, Ikeda K, Yamamoto T. Isolated body lateropulsion caused by lower lateral medullary infarction. eNeurologicalSci 2017;7:25-6.
8. Chetana N, Jayesh R. Subjective visual vertical in various vestibular disorders by using a simple bucket test. Indian J Otolaryngol Head Neck Surg 2015;67:180-4.
9. Yang YJ, Choi JE, Kim MT, Kim SH, Lee MY, Yoo DS, et al. Measurement of horizontal ocular deviation on magnetic resonance imaging in various disease with acute vertigo. PLoS One 2019;14:e0224605.
10. North American Symptomatic Carotid Endarterectomy Trial Collaborators, Barnett HJ, Taylor DW, Haynes RB, Sackett DL, Peerless SJ, et al. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Engl J Med 1991;325:445-53.
11. Sacco RL, Freddo L, Bello JA, Odel IC, Onesti ST, Mohr JP. Wallenberg’s lateral medullary syndrome. Clinical-magnetic resonance imaging correlations. Arch Neurol 1993;50:609-14.
12. Day GS, Swartz RH, Chenkin J, Shamji AI, Frost DW. Lateral medullary syndrome: A diagnostic approach illustrated through case presentation and literature review. CJEM 2014;16:164-70.
13. Shan DE, Wang V, Chen JT. Isolated lateropulsion of the trunk in cerebellar infarct. Clin Neurol Neurosurg 1995;97:195-8.
14. Arai M. Ipsilateral axial lateropulsion as an initial symptom of vertebral artery occlusion. J Neurol Neurosurg Psychiatry 2004;75:1648.
15. Bertholon P, Michel D, Convers P, Antoine JC, Barral FG. Isolated body lateropulsion caused by a lesion of the cerebellar peduncles. J Neurol Neurosurg Psychiatry 1996;60:356-7.
16. Felice KJ, Keilson GR, Schwartz WJ. Rubra’ gait ataxia. Neurology 1990;40:1004-5.
17. Li H, Wei N, Zhang L, Liu X, Han J. Body lateropulsion as the primary manifestation of medulla oblongata infarction: A case report. J Int Med Res 2020;48:0300060520970773.
18. Akdal G, Thurtell MJ, Halmagyi GM. Isolated lateropulsion in acute lateral medullary infarction. Arch Neurol 2007;64:1542-3.
19. Kim HJ, Kwon HM, Huh YE, Oh MY, Lee YS. Ipsilateral axial lateropulsion as an initial symptom of lateral medullary infarction: A case report. J Clin Neurol 2007;3:197-9.
20. Yamaoka Y, Kishishita S, Takayama Y, Okubo S. A report of a case involving body lateropulsion with numbness of the ipsilesional fingers caused by a small infarction in the dorsal part of the middle medulla. Case Rep Neurol 2018;10:54-9.
21. Thömke F, Marx JJ, Iannetti GD, Cruccu G, Fitzek S, Urban PP, et al. A topodiagnostic investigation on body lateropulsion in medullary infarcts. Neurology 2005;64:716-8.
22. Yi HA, Kim HA, Lee H, Baloh RW. Body lateropulsion as an isolated or predominant symptom of a pontine infarction. J Neurol Neurosurg Psychiatry 2007;78:372-4.
23. Karimi M, Razavi M, Fattal D. Rubral lateropulsion due to vertebral artery dissection in a patient with Klippel-Feil syndrome. Arch Neurol 2004;61:583-5.
24. Teufel J, Strupp M, Linn J, Kalla R, Feil K. Conjugate eye deviation in unilateral lateral medullary infarction. J Clin Neurol 2019;15:228-34.