

Body lateropulsion as the primary manifestation of medulla oblongata infarction: a case report

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Abstract

Background: Isolated body lateropulsion is a possible predominant manifestation of medulla oblongata infarction, and can occur without vestibular and cerebellar symptoms. However, it is relatively rare and challenging to diagnose.

Case presentation: A 67-year-old woman was admitted to the Harris International Peace Hospital complaining mainly of instability when standing and walking for the previous 8 hours. Based on the neural localization and multiple head magnetic resonance imaging (MRI) examinations, a diagnosis of cerebral infarction (vertebrobasilar system) was made. Consequently, the patient was managed using therapy aimed at preventing platelet aggregation, lowering plasma lipids, stabilizing plaques, protecting mitochondria, and improving circulation and brain function. The patient's gait improved and she was discharged after 14 days because she was able to walk unaided. The patient was followed up for 6 months and had no noticeable undesirable side effects or signs of neurological deficits.

Conclusion: The possibility of lateral medulla oblongata infarction should be considered when patients present with isolated body lateropulsion, without other signs or symptoms of brainstem damage.

Keywords

Infarction, body lateropulsion, medulla oblongata, instability, vestibular system, diffusion-weighted imaging, localization analysis

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Background

The somatosensory and vestibular systems aid in body gait and balance. Perturbations in the function of these systems are observed in infarction or injury to brain structures such as the medulla oblongata, pons, and cerebellum.¹ Horner's syndrome also affects the function of the somatosensory and vestibular systems, with accompanying clinical manifestations that include ataxia, hoarseness, coughing, loss of facial and limb sensations, dizziness, nausea, and vomiting.

Case presentation

A 67-year-old woman was admitted to the Harris International Peace Hospital complaining of difficulties with standing and walking for the previous eight hours. The patient was able to stand but walked unsteadily, without any obvious inducement, and her gait tilted to the right side. Other observed features include palpitations and an inability to perceive rotating objects, but she did not experience a headache, dizziness, nausea, or vomiting. In addition, the patient did not complain of numbness or weakness in her limbs. She was confirmed to have a history of hypertension for 18 years, with the highest blood pressure reading of 180/100 mmHg. Furthermore, she acknowledged having a 10-year history of coronary heart disease.

The patient's physical examination revealed consciousness, with free eye movement and no observable features of nystagmus. She was able to form bilateral forehead wrinkles, her nasolabial fold was symmetrical without changes in shallowness, and her tongue was centered. Her limb strength score was 5, with normal muscle tone when deep or shallow pressure was applied. In addition, the patient's tendon reflexes were symmetrical, with neither hyperactivity nor weakness.

Test results were negative for the bilateral Babinski sign, neck resistance, and bilateral finger-to-nose test. The bilateral heel-knee-shin test was stable; however, although the test for closed eyes was positive, the patient had difficulties opening her eyes after closing them.

Blood biochemical examination on admission revealed that the patient's triglycerides were 1.92 mmol/L and her homocysteine was 25.75 μ mol/L. No abnormalities were found in coagulation or blood tests.

Examination of the head using magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and diffusion-weighted imaging (DWI) revealed (1) multiple softening foci in the pons, left thalamus, and bilateral basal ganglia; (2) few chronic ischemic changes in both the frontal and parietal lobes; and (3) narrowing of the lumen at the A4 segment of the left anterior cerebral artery and the P1 segment of the bilateral posterior cerebral artery, with non-uniform signal. Furthermore, the bilateral posterior cerebral arteries, basilar arteries, and vertebral arteries had thinned, while number of the distal branches had decreased. Ultrasonography of the neck vessels revealed a plaque in the right subclavian artery. No abnormalities were observed with a video eye-tracker. The MRI of the spinal cord revealed no obvious abnormal signals. Head DWI re-examination (on day 3 after onset, Figure 1) suggested acute infarction of the lateral medulla oblongata. The definite diagnosis was cerebral infarction (vertebro-basilar system). The treatment strategy for this patient was aimed at preventing platelet aggregation, lowering plasma lipids, stabilizing plaques, protecting mitochondria, and improving circulation and brain function. Compared with the features observed at admission, the patient's standing and walking ability and gait were greatly improved at 14 days after treatment initiation. The patient was discharged from the



Figure 1. Diffusion-weighted imaging of the acute infarction of the lateral medulla oblongata.

hospital when she regained the ability to walk unaided. During 6 months of follow-up, no signs or symptoms of neurological deficits were noted in the patient.

Discussion

The main clinical feature of body lateropulsion is the involuntary leaning of the body to one side while standing; this is a specific manifestation of trunk ataxia. The maintenance of normal balance—whether mobile or stationary, conscious or unconscious—relies on precise coordination by the vestibular, visual, and proprioception structures.² Incoherent or inconsistent signal transmission to the cerebral cortex leads to vertigo. When a patient presents with a balance disorder that is unaccompanied by proprioception or visual disorders, the vestibular system and unconscious proprioception are highly likely to be involved. The mechanisms by which the vestibular system achieves body balance and posture are complex, and are coordinated mainly through the vestibulospinal reflex, vestibular reflex,

and vestibulo-cerebellar pathways.³ Vestibulo-ocular function can be detected by video eye-tracker. Difficulties in closing the eyes represent the reflex function of the vestibulospinal cord. Unconscious proprioception is mainly transmitted to the vestibulocerebellum through the anterior and posterior tracts of the cerebellum. Therefore, lesions that involve the vestibular pathway and the unconscious proprioceptive pathway can theoretically lead to lateropulsion of the body. Furthermore, lesions in other brain areas, such as the dorsolateral medulla oblongata, ventral pons, medial longitudinal fasciculus, mid-brain, vestibular cortex, superior cerebellar peduncle, inferior cerebellar peduncle, and flocculonodular lobe, can also lead to body lateropulsion. The fibers involved in these defects include those in the vestibulospinal, spinocerebellar, and vestibulothalamic tracts.⁴⁻⁷ Challenges with maintaining a standing posture and walking gait among affected patients can limit their visual abilities.

The patient in the present report was admitted to hospital with isolated body lateropulsion. Physical examination revealed a positive Romberg's sign when the patient's eyes were closed or open. No dizziness, nausea, vomiting, diplopia, or other symptoms or signs were observed. The finger-to-nose and heel-knee-shin tests were negative, and video eye-tracker analysis revealed no abnormalities. Together, these results suggest that the vestibulo-ocular, vestibulo-autonomic, and vestibulo-cortical pathways were not abnormal in this patient, whereas the vestibulospinal and vestibulo-cerebellar pathways were more likely to be involved. Unconscious proprioceptive abnormalities were ruled out on the basis of the negative results from the finger-to-nose and heel-knee-shin tests. Lesions of ascending fibers in the posterior spinocerebellar tract may result in limb ataxia because of impaired unconscious proprioceptive function or a

lack of input information. The vestibulo-cerebellar pathways are mainly regulated by the vestibulospinal and medial longitudinal tracts. Comprehensive analysis indicated that the lesion was located in the vestibulospinal tract of our patient. The vestibulospinal tract runs from the lateral vestibular nuclei in the brainstem to the descending tract of the spinal cord, between the end of the posterior spinocerebellar tract and the beginning of the inferior cerebellar peduncle, and between the lateral spinal cord. However, the first MRI of our patient's head and spinal cord revealed no obvious lesions, which initially made the diagnosis quite challenging. Based on the localization analysis, we decided to re-examine the DWI to identify the lesion site. Eventually, a lateral acute infarction of the medulla oblongata was identified, enabling us to make a clear diagnosis of the patient's condition. The first negative head MRI examination may be correlated with the onset of the infarction or may have been caused by skull base interference. According to Kim et al., our case would be categorized as the caudal lateral type of medulla oblongata infarction.⁸ Although the vestibulospinal and posterior spinocerebellar tracts are adjacent to the medulla,⁹ our patient only had involvement of the vestibulospinal cord. This conclusion was supported by the location of the lesion and the plane anatomy, as well as by the negative results of the finger-to-nose and heel-knee-shin tests, suggesting that the lesion only damaged the vestibulospinal cord.

In summary, in the era of rapidly developing neuroimaging techniques, neurologists often rely excessively on imaging examinations and ignore the value of localization analyses. However, precise localization analysis remains particularly important for the diagnosis of neurological diseases. The possibility of lateral medulla oblongata infarction should be considered when patients present with isolated body

lateropulsion, without other signs or symptoms of brainstem damage.

Author contributions

HL, NW, and LZ contributed to the conception and design; NW and LZ generated the study data; NW and XL analyzed and interpreted the data; HL, NW, and JH drafted and revised the manuscript, including critical appraisal for intellectual content; all authors gave approval of the final version to be published.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

Ethics statement

This study was approved by the Institutional Review Board and the Ethics Committee of the Harrison International Peace Hospital (2018-1-004). Written informed consent was obtained from the patient for the publication of this report.

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