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Short communication

Ipsilesional limb ataxia and truncal ipsipulsion in isolated infarction of the superior cerebellar peduncle

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ABSTRACT

The clinical features of a lesion confined to the superior cerebellar peduncle (SCP) have not been defined well in human. A 92-year-old woman suddenly developed mild dysarthria and severe imbalance from an isolated unilateral SCP infarction, and examination showed ipsiversive ocular torsion, severe ipsilesional limb ataxia, and truncal ipsipulsion. These findings are well consistent with those observed in monkeys when the SCP was severed. In addition to the dentate-rubro-thalamic projections, the SCP appears to contain the fibers involved in the control of eye motion in the roll plane.

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1. Introduction

The superior cerebellar peduncle (SCP) mostly contains the efferent fibers from the cerebellum [1]. The main cerebellar efferents across the SCP include the cerebellothalamocortical projection fibers from the dentate nucleus to the contralateral red nucleus, and then to the thalamus (the dentate-rubro-thalamic tract) [2]. Previously, clinical features of a lesion restricted to the SCP have been described only in patients with multiple sclerosis [3]. Here, we report dysarthria, and severe truncal ipsipulsion and ipsilesional limb ataxia in a patient with an isolated SCP infarction.

2. Case report

A 92-year-old woman with hypertension for several years presented with dysarthria and imbalance. Examination showed mild dysarthria and marked ataxia in the left upper and lower extremities. She persistently fell to the left on standing. She did not show head tilt, spontaneous nystagmus, ocular lateropulsion, or skew deviation. Vibratory stimuli on the mastoids and horizontal head-shaking induced mild downbeat nystagmus, but within normal range [4]. Horizontal saccades were normal, but horizontal smooth pursuit was symmetrically impaired in both directions (Fig. 1A). Bedside horizontal head-impulse tests were normal in both directions. Remainder of the neurological examinations was unrevealing without any weakness or sensory changes.

Fundus photography documented ipsiversive torsion $(-6.0^{\circ},$ normal range 0–12.6°, negative value indicates an intorsion.) of the right eye, but the torsional position of the left eye was within normal range (8.4°, Fig. 1B). Diffusion- and T2-weighted MRIs disclosed an acute infarction restricted to the left SCP (Fig. 1C) and multiple old lacunae in the deep cerebral white matter. MR angiography was normal with patent left superior cerebellar artery (SCA). Findings of transthoracic echocardiography and prolonged electrocardiogram monitoring were also normal. The presence of patent foramen ovale (PFO) was suspected due to more than 10 embolic signals detected in the middle cerebral artery within 5 s during a saline agitation test using a transcranial Doppler.

She was placed on aspirin 100 mg per day and discharged 10 days later. Follow-up examination 3 months later still showed leftward falling tendency on walking, and she could walk only with a support.

3. Discussion

We, for the first time, describe ipsiversive ocular torsion, mild dysarthria, severe ipsilesional limb ataxia, and truncal ipsipulsion in isolated unilateral SCP infarction.





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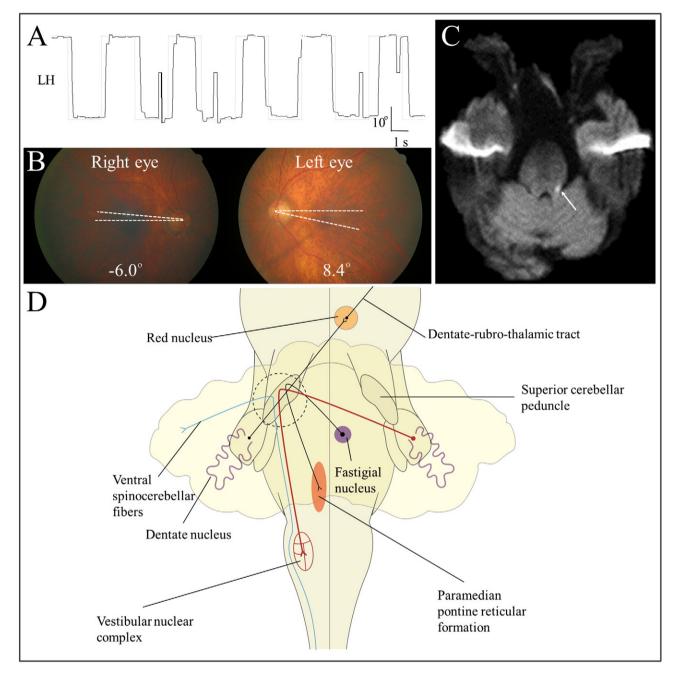


Fig. 1. (A) Video-oculography (SMI, Teltow, Germany) shows normal horizontal saccades in both directions. Upward deflection in the recording indicates rightward eye motion. (B) Fundus photography shows intorsion of the right eye (normal range: 0° -12.6°, negative value indicates intorsion.). (C) Diffusion-weighted MRI demonstrates an acute infarction restricted to the left superior cerebellar peduncle (arrow). (D) Schematic illustration of the fibers passing through the superior cerebellar peduncle (SCP). In view of the ipsiversive ocular torsion observed in our patient, the SCP appears to contain the fibers controlling eye motion in the roll plane presumably by connecting contralateral dentate nucleus and ipsilateral vestibular nuclei (modified from Frohman TC, et al. The neuro-ophthalmology of multiple sclerosis. Continuum 2010;16:122–46.) *LH* = horizontal position of the left eye.

The SCP contains mainly the efferent fibers from the cerebellar nuclei to the red nucleus, thalamus, pontine reticular nuclei, olivary nucleus, and hypothalamus [5]. However, some afferent fibers traveling through the SCP include the ventral spinocerebellar fibers [5]. The findings observed in our patient are in line with the kinetic tremor, severe ipsilesional truncal and limb ataxia that were observed in rhesus monkeys when the unilateral SCP was severed [6]. The severe truncal ipsipulsion and ipsilesional limb ataxia reflect damage to the cerebellothalamocortical projections as they exit through the SCP.

Unlike the caudal cerebellum by which the neural signals from the vestibulocerebellum are passed to the brainstem, the rostral cerebellum is less likely responsible for nystagmus [7]. Indeed, only one-fourth of

the patients with infarctions involving the SCA territory showed nystagmus, which is mostly ipsilesional [7].

In a previous study, saccadic contrapulsion was described in association with ipsilateral ataxia in a patient with unilateral SCA infarction and in patients with a demyelinating lesion restricted to the SCP [3,8]. The saccadic contrapulsion in rostral cerebellar lesions has been explained by damage to the uncinate fasciculus that hooks around the SCP. The uncinate fasciculus carries the efferents from the deep cerebellar nuclei to the brainstem reticular formation and thalamus [5]. The main trunk gives off the ascending and descending limbs after decussation [5]. Involvement of the descending limb targeting the paramedian pontine reticular formation is known to cause saccadic contrapulsion [8]. However, our patient did not show the features of ocular lateropulsion. The uncinate fasciculus running through the dorsolateral border of the SCP may have been spared in our patient [1].

Of interest, our patient also showed ipsiversive ocular torsion, which indicates a vestibular tone imbalance in the roll plane [9]. The ocular tilt reaction (OTR) or perceptual tilt observed in cerebellar lesions has been ascribed to damage to the dentate nucleus [10]. The lesions involving the dentate nucleus or its outflow fibers to the vestibular nuclei may cause contraversive OTR [10]. In contrast, cerebellar lesions involving the biventral lobule, the middle cerebellar peduncle, the tonsil, and the inferior semilunar lobule have caused ipsiversive OTR [10]. The ipsiversive ocular torsion observed in our patient suggests that the SCP also contains the fibers involved in the control of eye motion in the roll plane (Fig. 1D).

The SCP is irrigated by the posterior cerebellar peduncular arteries, which are the end tributaries of the medial and lateral branches of SCA [11]. Cardioembolism is the most common cause of isolated SCA infarction [12], and our patient also showed the findings suggestive of paradoxical embolism through the PFO, which is a common cause of cryptogenic embolism in old age [13]. The rarity of isolated SCP infarction may be explained by the low possibility of selective embolization only to the posterior cerebellar peduncular artery. However, the possibility of a small vessel disease cannot be excluded.

Although dysarthria and unilateral ataxia are non-specific signs of brainstem or cerebellar lesions, accompanying OTR may further localize the lesion to the ipsilateral rostral cerebellum including the SCP. The SCP appears to contain the fibers involved in the control of eye motion in the roll plane in addition to the uncinate fasciculus, dentate-rubro-thalamic projections and ventral spinocerebellar tracts.

Author contributions

Dr. Lee wrote the manuscript, and analyzed and interpreted the data.

Dr. Bae analyzed and interpreted the data, and revised the manuscript. Dr. Kim conducted the design and conceptualization of the study,

interpretation of the data, and revising the manuscript.

Disclosure

Dr. Lee and Bae report no disclosures.

Dr. Kim serves as an associate editor of Frontiers in Neuro-otology and on the editorial boards of the Journal of Korean Society of Clinical Neurophysiology, Journal of Clinical Neurology, Frontiers in Neuroophthalmology, Journal of Neuro-ophthalmology, Journal of Vestibular Research, Journal of Neurology, and Medicine; and received research support from SK Chemicals, Co. Ltd.

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References

- Rasmussen AT. Origin and course of the fasciculus uncinatus (Russell) in the cat, with observations on other fiber tracts arising from the cerebellar nuclei. J Comp Neurol 1933;57:165–97.
- [2] Wakana S, Jiang H, Nagae-Poetscher LM, Van Zijl PC, Mori S. Fiber tract-based atlas of human white matter anatomy 1. Radiology 2004;230:77–87.
- [3] Frohman EM, Frohman T, Fleckenstein J, Racke M, Hawker K, Kramer P. Ocular contrapulsion in multiple sclerosis: clinical features and pathophysiological mechanisms. J Neurol Neurosurg Psychiatry 2001;70:688–92.
- [4] Choi KD, Oh SY, Kim HJ, Koo JW, Cho BM, Kim JS. Recovery of vestibular imbalances after vestibular neuritis. Laryngoscope 2007;117:1307–12.
- 5] Hans J. Clinical neuroanatomy: brain circuitry and its disorders. Springer; 2011.
- [6] Walker AE, Botterell E. The syndrome of the superior cerebellar peduncle in the monkey. Brain 1937;60:329–53.
- [7] Lee H, Kim H-A. Nystagmus in SCA territory cerebellar infarction: pattern and a possible mechanism. J Neurol Neurosurg Psychiatry 2013;84:446–51.
- [8] Ranalli PJ, Sharpe JA. Contrapulsion of saccades and ipsilateral ataxia: a unilateral disorder of the rostral cerebellum. Ann Neurol 1986;20:311–6.
- [9] Brandt T, Dieterich M. Central vestibular syndromes in roll, pitch, and yaw planes: topographic diagnosis of brainstem disorders. Neuro-ophthalmology 1995;15: 291–303.
- [10] Baier B, Bense S, Dieterich M. Are signs of ocular tilt reaction in patients with cerebellar lesions mediated by the dentate nucleus? Brain 2008;131:1445–54.
- [11] Tatu L, Moulin T, Bogousslavsky J, Duvernoy H. Arterial territories of human brain: brainstem and cerebellum. Neurology 1996;47:1125–35.
- [12] Terao S-i, Sobue G, Izumi M, Miura N, Takeda A, Mitsuma T. Infarction of superior cerebellar artery presenting as cerebellar symptoms. Stroke 1996;27:1679–81.
- [13] Handke M, Harloff A, Olschewski M, Hetzel A, Geibel A. Patent foramen ovale and cryptogenic stroke in older patients. N Engl J Med 2007;357:2262–8.