

## Unilateral Isolated Trochlear Nerve Palsy due to Ipsilateral Midbrain Infarction

Toshiyuki Hayashi<sup>1,2</sup>, Koichi Nomura<sup>1,2</sup>, Yasuhiro Nishiyama<sup>1</sup> and Kazumi Kimura<sup>1</sup>

<sup>1</sup>Department of Neurology, Graduate School of Medicine, Nippon Medical School, Tokyo, Japan

<sup>2</sup>Department of Neurology, Shioda Hospital, Chiba, Japan

Ischemic stroke is a very rare etiology in cases of isolated trochlear nerve palsy, and no reports of ipsilateral trochlear nerve palsy caused by unilateral stroke have so far been published. However, we now report a case of isolated trochlear nerve palsy due to ipsilateral dorsal small midbrain infarction in a 70-year-old woman who presented with acute onset of diplopia. There were no other clinical manifestations, but brain magnetic resonance imaging revealed a small ischemic lesion in the right dorsal midbrain, showing that isolated trochlear nerve palsy can be caused by stroke.

(J Nippon Med Sch 2021; 88: 561–563)

**Key words:** ischemic stroke, trochlear nerve palsy, cranial nerve palsy, magnetic resonance imaging

### Introduction

Trochlear nerve palsy can be caused by demyelinating disease, infection, tumor, trauma, or stroke<sup>1,2</sup>. Fourth cranial nerve palsy usually originates in the contralateral midbrain, because the fourth cranial nerve crosses the dorsal midbrain and innervates the opposite side of the superior oblique muscle<sup>3</sup>. There have as yet been no reports of isolated ipsilateral trochlear nerve palsy caused by unilateral stroke.

Thanks to the development of magnetic resonance imaging (MRI), small lesions invisible on computed tomography can be detected in early neuroimaging for the initial evaluation of acute neurological symptoms<sup>4–7</sup>. Early diagnosis of stroke is important for secondary prevention by administering antithrombotic drugs and controlling risk factors.

We report a case of isolated trochlear nerve palsy due to ipsilateral dorsal small midbrain infarction.

### Case Report

A 70-year-old woman who had immediately consulted an ophthalmologist after experiencing sudden onset diplopia was referred to us with a diagnosis of right trochlear nerve palsy, because the ophthalmologist suspected stroke. The patient had experienced no recent head injuries or infections, but she had a history of hypertension,

dyslipidemia, left retinal branch vein occlusion, and mitral valve replacement with an artificial valve for severe mitral regurgitation. She was taking warfarin, furosemide, bisoprolol, atorvastatin, esomeprazole, magnesium oxide, and kallidinogenase. She had never smoked and was a social drinker.

On examination, the patient was found to have normal higher mental functions. The diplopia was worse and the trochlear nerve palsy was more evident on left downward gaze (**Fig. 1**); it also increased when the head was tilted to the right (positive Bielschowsky head tilt test). Her pupils were equal, normally reacting to light, and no other cranial nerves were affected. There were no motor or sensory abnormalities in the limbs, and no meningeal signs were present. All clinical findings were compatible with right isolated trochlear nerve palsy. Her National Institutes of Health Stroke Scale score was 0, and her pre-stroke modified Rankin Scale score was 0.

The results of laboratory tests were as follows: NT-proBNP 2,064 pg/mL, PT-INR 2.09, and D-dimer 0.3 µg/mL. Brain MRI showed a small hyperintense lesion located in the right dorsal midbrain and right side of the cerebellum on diffusion-weighted imaging (DWI) (**Fig. 2**). Magnetic resonance angiography results were normal, but carotid ultrasonography showed some plaque in the bilateral carotid arteries and no plaque on bilateral verte-

Correspondence to Toshiyuki Hayashi, MD, Department of Neurology, Graduate School of Medicine, Nippon Medical School, 1-1-5 Sendagi, Bunkyo-ku, Tokyo 113-8602, Japan

E-mail: toshi-h-06@nms.ac.jp

[https://doi.org/10.1272/jnms.JNMS.2021\\_88-515](https://doi.org/10.1272/jnms.JNMS.2021_88-515)

Journal Website (<https://www.nms.ac.jp/sh/jnms/>)



Fig. 1 Limited movement of the right superior oblique muscle on left downward gaze is evident.

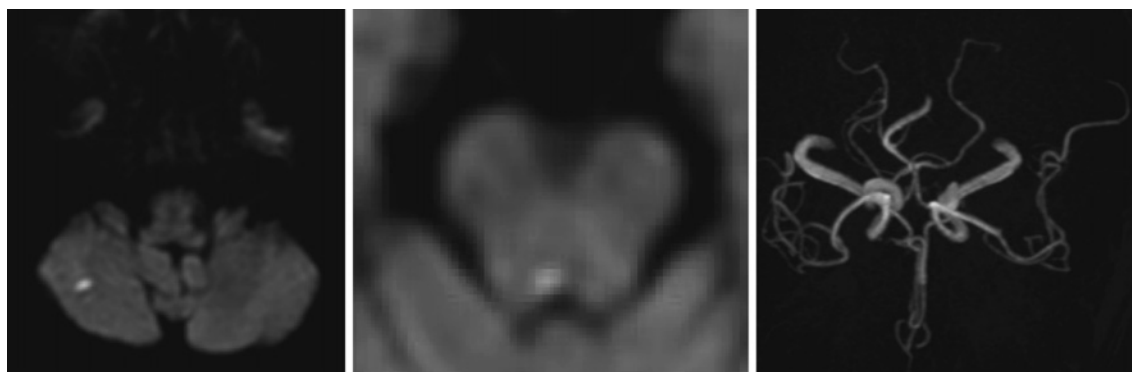


Fig. 2 Head magnetic resonance imaging shows hyperintense lesions located in the right dorsal midbrain and right side of the cerebellum on diffusion-weighted imaging. Magnetic resonance angiography findings are normal.

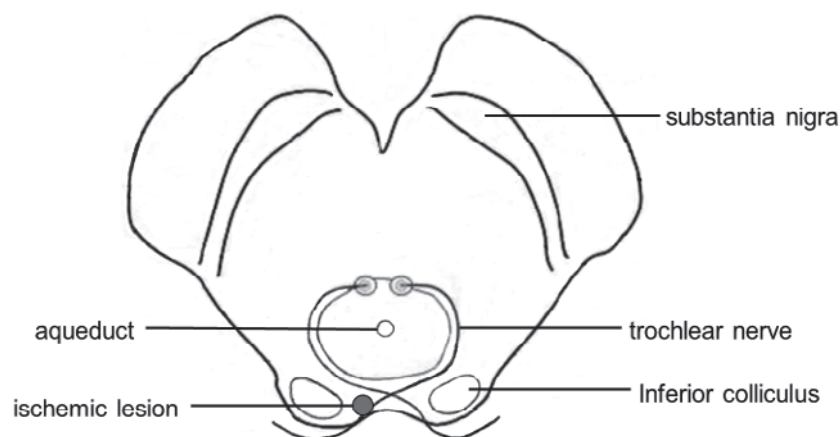


Fig. 3 Schematic drawing represents the ischemic lesion which involves the right trochlear nerve after crossing.

bral arteries as embolic sources. Cardiac ultrasound revealed no intracardiac thrombi, but 24-hour electrocardiogram monitoring carried out on admission showed paroxysmal atrial fibrillation.

The patient was treated with edaravone 30 mg twice a day and warfarin, with a target PT-INR range of 2.0-3.0. The diplopia resolved completely the day after admission, and the high-intensity lesions in the midbrain and cerebellum were no longer visualized on 5 days after onset. Twelve days after onset she was discharged from our hospital with no recurrence.

### Discussion

The case of isolated trochlear nerve palsy caused by ipsilateral dorsal small midbrain infarction described herein is, to the best of our knowledge, the first reported case of isolated ipsilateral trochlear nerve palsy due to unilateral stroke. The midbrain is supplied by branches arising from the posterior cerebral artery, upper basilar artery, and the superior cerebellar artery. The isolated trochlear nerve palsy is rare because the occlusion of these arteries often involves other neurological deficits such as ataxia, dysesthesia and ocular motor nerve palsy. The trochlear

nucleus lies ventrolateral to the aqueduct, at the level of the inferior colliculus. The trochlear nerve courses around the aqueduct to cross the dorsal midbrain and exits on the contralateral side. The trochlear nucleus is the only cranial nerve whose fibers cross over as they emerge from the midbrain and innervate the contralateral superior oblique muscle. Therefore, the trochlear nerve palsy due to disturbance of that nucleus usually affects the contralateral side. In the present case, however, MRI revealed the trochlear nerve palsy to be on the ipsilateral side of the infarction, which we believed to be because the trochlear nerve was damaged after crossing (**Fig. 3**).

Our patient had several risk factors for stroke, including not only hypertension, dyslipidemia but also the fact that she had undergone mitral valve replacement with an artificial valve and atrial fibrillation. Therefore, the mechanism of stroke was unclear, but embolic stroke was suspected because multiple lesions were found in the midbrain and cerebellum on MRI. In cases of trochlear nerve palsy, stroke should be considered as a possible cause, as well as trauma, infection, demyelinating disease, and tumor.

**Conflict of Interest:** none.

#### References

1. Keane JR. Fourth nerve palsy: historical review and study of 215 patients. *Neurology*. 1993;43:2439-43.
2. Richards BW, Jones FR, Younge BR. Causes and prognosis

in 4278 cases of paralysis of the oculomotor, trochlear, and abducens cranial nerves. *Am J Ophthalmol*. 1992;113:489-96.

3. Lee SH, Park SW, Kim BC, Kim MK, Cho KH, Kim JS. Isolated trochlear palsy due to midbrain stroke. *Clin Neurol Neurosurg*. 2010;112:68-71.
4. Tamhankar MA, Biousse V, Ying GS, et al. Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: a prospective study. *Ophthalmology*. 2013;120:2264-9.
5. Akagi T, Miyamoto K, Kashii S, Yoshimura N. Cause and prognosis of neurologically isolated third, fourth, or sixth cranial nerve dysfunction in cases of oculomotor palsy. *Jpn J Ophthalmol*. 2008;52:32-5.
6. Chou KL, Galetta SL, Liu GT, et al. Acute ocular motor mononeuropathies: prospective study of the roles of neuroimaging and clinical assessment. *J Neurol Sci*. 2004;219:35-9.
7. Warwar RE, Bhullar SS, Pelstring RJ, Fadell RJ. Sudden death from pituitary apoplexy in a patient presenting with an isolated sixth cranial nerve palsy. *J Neuroophthalmol*. 2006;26:95-7.

(Received, July 13, 2020)

(Accepted, December 7, 2020)

(J-STAGE Advance Publication, January 19, 2021)

Journal of Nippon Medical School has adopted the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International License (<https://creativecommons.org/licenses/by-nc-nd/4.0/>) for this article. The Medical Association of Nippon Medical School remains the copyright holder of all articles. Anyone may download, reuse, copy, reprint, or distribute articles for non-profit purposes under this license, on condition that the authors of the articles are properly credited.