

# Midbrain Infarction Causing Oculomotor Nerve Palsy and Ipsilateral Cerebellar Ataxia

Makoto Tokunaga, Kimiko Fukunaga, Ryoji Nakanishi,  
Susumu Watanabe and Hiroaki Yamanaga

---

## Abstract

---

We herein report the case of an 81-year-old woman with midbrain infarction causing pupil-sparing oculomotor nerve palsy with ipsilateral cerebellar ataxia. The lesion was located at the rostral end of the decussation of the superior cerebellar peduncle touching the dorsal side, further caudal and dorsal to causal lesions of Claude's syndrome, which presented as oculomotor palsy and contralateral cerebellar ataxia. This is the third report of midbrain infarction causing partial oculomotor nerve palsy with ipsilateral cerebellar ataxia. It may be possible to establish this entity as a new syndrome following the accumulation of more cases.

**Key words:** partial oculomotor nerve palsy, midbrain infarction, cerebellar ataxia, Claude's syndrome, tractography

(Intern Med 53: 2143-2147, 2014)

(DOI: 10.2169/internalmedicine.53.1613)

---

## Introduction

---

The oculomotor nucleus is located at the height of the superior colliculus close to the middle of the central gray region, with fiber bundles running ventrally passing through the red nucleus and exiting from the medial side of the cerebral peduncle (interpeduncular fossa). Because the intramedullary nerve fiber bundles spread out rostrocaudally and mediolaterally, small midbrain infarction can cause partial oculomotor nerve palsy.

Claude's syndrome (1) results in oculomotor palsy on the lesion side with cerebellar ataxia on the opposite side and possibly occurs due to the presence of lesions in the lower red nucleus, the crossing point for fibers from the oculomotor nerve and decussation of the superior cerebellar peduncle. Therefore, Claude's syndrome is also known as "lower red nucleus syndrome." However, Seo et al. (2) reported that, among six patients with Claude's syndrome, all lesions were caudal to the red nucleus, suggesting that the lesions responsible for Claude's syndrome affect the fibers between the decussation of the superior cerebellar peduncle and the red nucleus, not the lower red nucleus.

We experienced a patient with midbrain infarction causing

pupil-sparing partial oculomotor nerve palsy with ipsilateral cerebellar ataxia. Two previous cases of partial oculomotor nerve palsy with ipsilateral cerebellar ataxia have been reported (3, 4); however, the relationship between the site of the lesion and the structure of the red nucleus and/or decussation of the superior cerebellar peduncle in these patients was unclear. In the present case, we used 3-Tesla magnetic resonance imaging (3T-MRI) to scan the midbrain in 1-mm slices and visualized the fibers of the decussation of the superior cerebellar peduncle on tractography. We herein discuss this case in the context of the relevant literature.

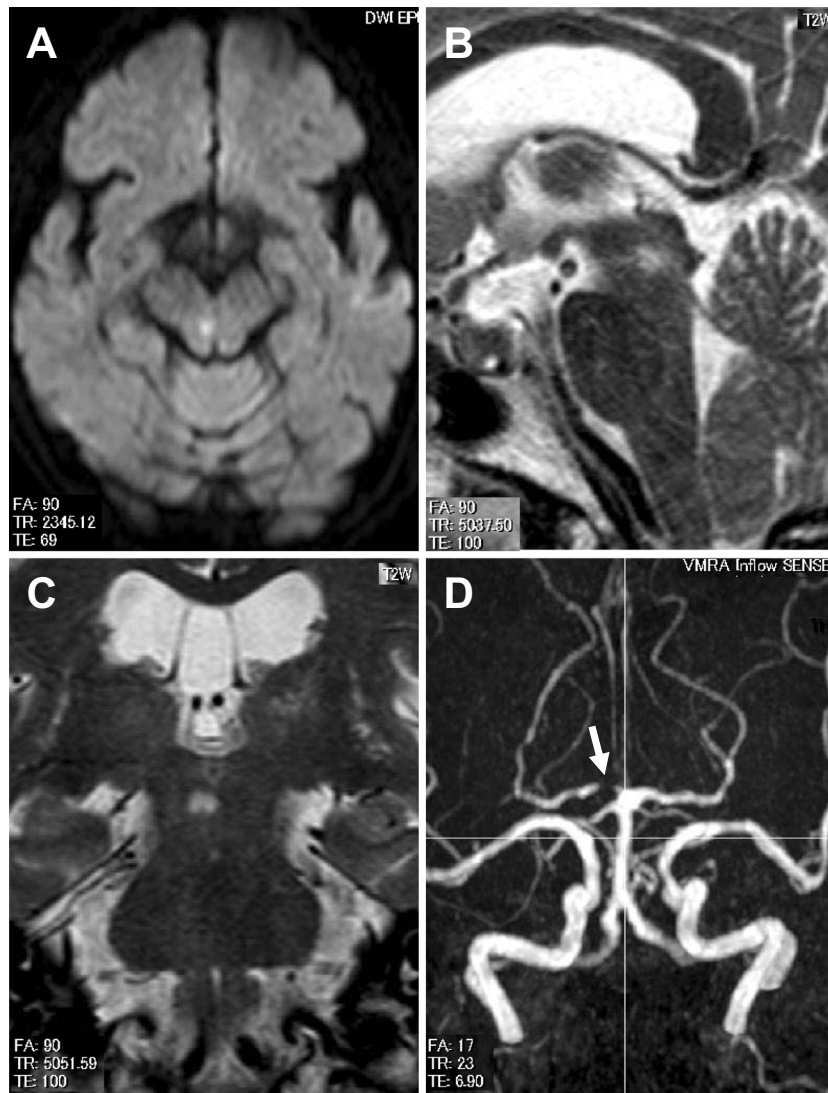
---

## Case Report

---

An 81-year-old woman had been taking oral medication for hypertension since her 60's. She had no history of smoking. On the day of onset (day 1), dizziness appeared soon after the patient woke up, accompanied by double vision and difficulty in grasping objects, and she was referred to our hospital. Cranial magnetic resonance imaging (MRI) showed hyperintensity in the right side of the paramedian midbrain under diffusion-weighted imaging (DWI). Cerebral infarction was diagnosed, and she was admitted on day 2.

The patient's findings on admission were as follows:

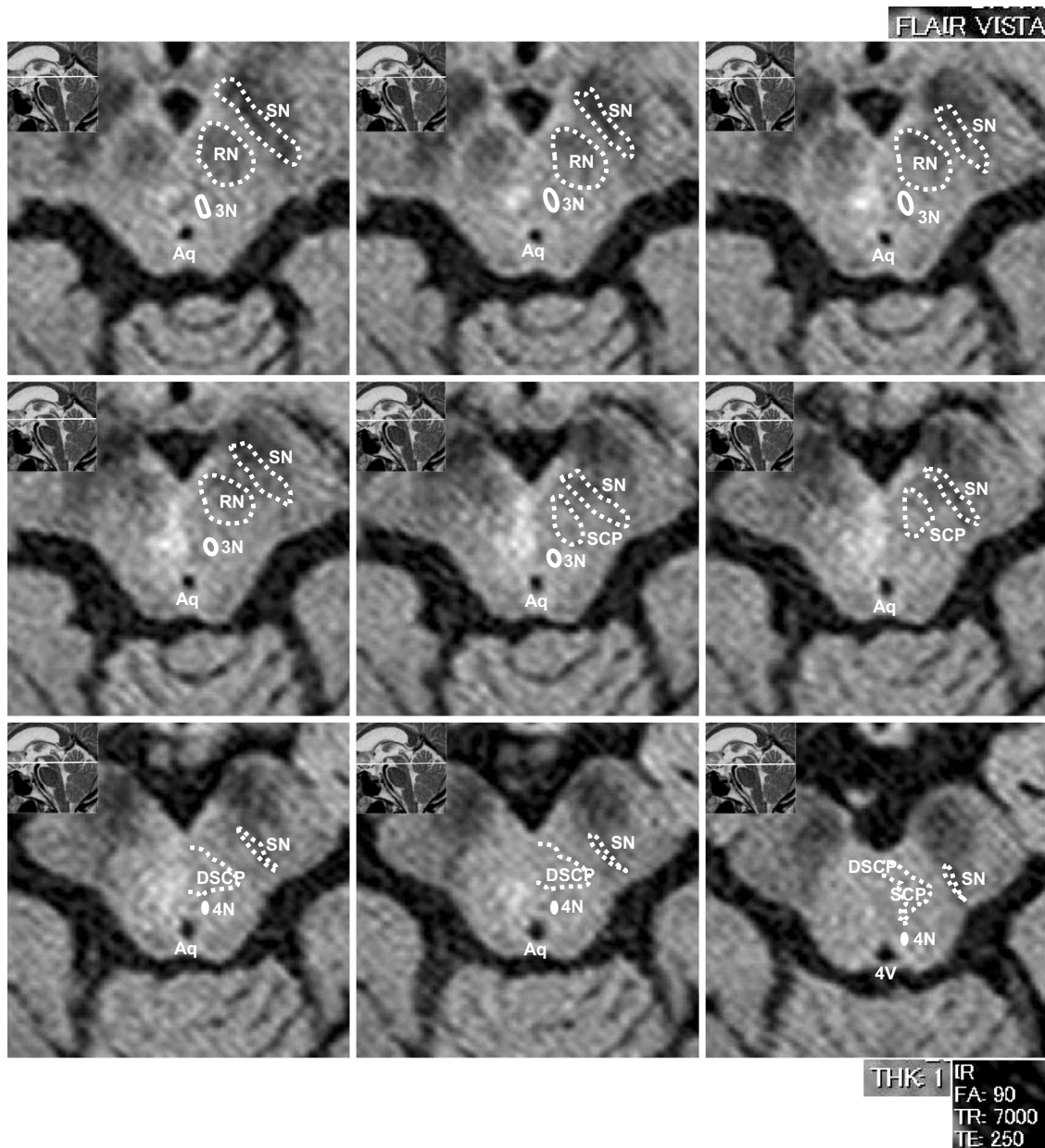


**Figure 1.** Diffusion-weighted (A) and T2-weighted (B, C) magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) (D). Diffusion-weighted (A: day 1) and T2-weighted (B, C: day 4) MRI revealed a increased signal area in the right paramedian midbrain at the level of inferior colliculus, and MRA (D: day 4) demonstrated a severe stenosis in the P1 portion of the right posterior cerebral artery (arrow).

height=140 cm, weight=43 kg (BMI 21.9), blood pressure=144/80 mmHg, pulse=91 bpm (regular). In terms of neurological findings, she was lucid with no dysarthria. Right palpebral ptosis (right palpebral fissure, 4 mm; left palpebral fissure, 8 mm) was present, and there was mild exophoria and adduction failure (-3) of the right eye. The patient was incapable of performing eye convergence, although her vertical movement of the right eye was normal. The pupil diameter in both eyes was 3.5 mm, and her light reflex was normal. Ataxia of the right upper limbs and leg was evident, and she staggered when walking with a stick. Although she was capable of standing with her legs apart (approximately 25-cm stride length), she had difficulty standing with her legs together and was unable to stand on one leg. There was no muscle weakness, sensory disturbances or bladder or bowel dysfunction.

Regarding the test results obtained on admission, the pa-

tient's blood count and urinalysis findings were normal, as were biochemical tests, with the exception of a BUN level of 26 mg/dL, a T-Chol level of 249 mg/dL, a LDL-Chol level of 150 mg/dL, a fasting blood glucose level of 112 mg/dL, an HbA1c (NGSP) level of 6.4% and a BNP level of 18 pg/mL. Electrocardiography showed a sinus rhythm with signs of left ventricular hypertrophy. An ultrasound analysis of the cervical vessels revealed no abnormalities. Cranial MRI performed on days 1 and 4 showed hyperintensity in the right side of the paramedian midbrain under DWI (Fig. 1A), T2 weighted imaging (Fig. 1B, C) and previous microbleeding in the left cerebellum. Meanwhile, magnetic resonance angiography (MRA) disclosed severe stenosis of the right posterior cerebral artery (Fig. 1D). Ataxia of the right upper limbs was evident upon voluntary movement of the upper limbs (5), with mild truncal ataxia on stabilometry. On day 6, the patient was examined at the department



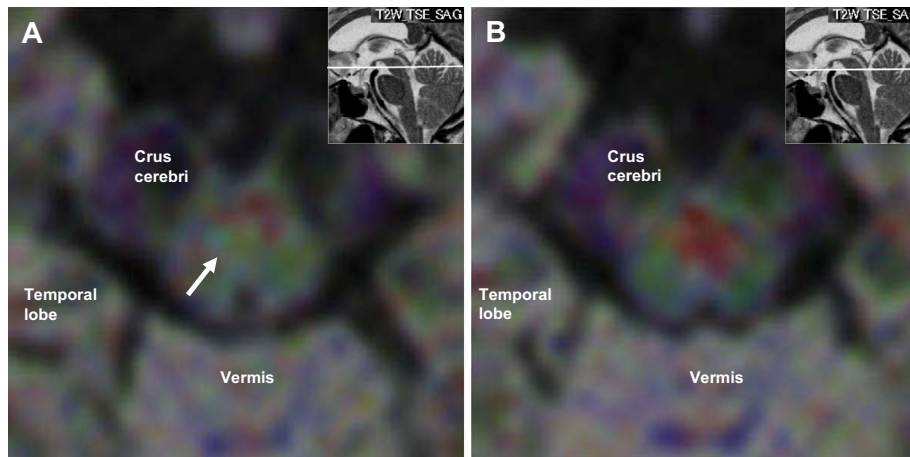
**Figure 2.** The serial axial images acquired using a FLAIR-VISTA sequence by 3T-MRI (day 14). The slice thickness was 1 mm. The high signal lesion was extended from the caudal end level of red nucleus to the decussation of superior cerebellar peduncle. Illustrations were consulted to *The Human Nervous System* edited by Mai and Paxinos (10). 3N: oculomotor nucleus, 4N: trigeminal nucleus, 4V: 4th ventricle, Aq: aqueduct, DSCP: decussation of superior cerebellar peduncle, RN: red nucleus, SCP: superior cerebellar peduncle, SN: substantia nigra

of Ophthalmology, which identified palsy of the medial rectus (MR) and levator palpebrae superioris (LP) in the right eye, with the impossibility of convergence. On day 14, 3T-MRI was performed to scan the midbrain in 1-mm slices (Fig. 2), and the fibers of the decussation of the superior cerebellar peduncle were visualized on tractography (6) in 3-mm slices (Fig. 3). The rostral extremity of the lesion in the right side of the paramedian midbrain was at the height of the bottom of the red nucleus, located medially and dorsally to the red nucleus. The caudal extremity of the lesion was located at the rostral end of the decussation of the superior cerebellar peduncle touching the dorsal side.

With respect to the patient's post-admission course, oral aspirin and ozagrel sodium drops were administered. She was discharged on day 30, with the ability to walk home with a stick. On a follow-up visit to our outpatient department on day 50, only very little subjective LP palsy remained.

## Discussion

Cases of partial oculomotor nerve palsy with ipsilateral cerebellar ataxia are extremely rare. To the best of our knowledge, there are only two previous reports of such pa-



**Figure 3.** Diffusion tensor images (day 14) at the level of the decussation of superior cerebellar peduncle. The first proper vector of each voxel display green; anterior-posterior, red; right-left, and blue; rostral-caudal direction, respectively. The slice thickness was 3 mm. The caudal end of the lesion (arrow of A) revealed the defect of the crossing tensor (red) at the right dorsal part of decussation. In the next caudal slice (B), the crossing tensor of the right superior cerebellar peduncle might be decreased.

tients (3, 4). Schwartz et al. (3), in their letter to the editor, described a patient with right partial oculomotor nerve palsy and ataxia of right upper limbs and leg (which disappeared after 12 hours). The authors inferred that the transient cerebellar ataxia may have been caused by extension of the lesion to the decussation of the superior cerebellar peduncle. Regarding the second case, Verstichel et al. (4) reported, in French, a patient with a variant of Claude's syndrome who exhibited left partial oculomotor nerve palsy and left superior oblique muscle palsy with left cerebellar ataxia. The authors proposed that the ipsilateral cerebellar ataxia may have been due to damage to the fibers before the crossing in the decussation of the superior cerebellar peduncle. In both of these cases (3, 4), neither detailed diagnostic imaging of the extent of the lesion nor tractography were performed, and the positional relationship between the decussation of the superior cerebellar peduncle and the lesion was merely based on assumption.

Tractography is a method of visualizing (using a tensor analysis of DW images) the courses of nerve fibers running within the brain and can be used to clearly visualize the decussation of the superior cerebellar peduncle, which is difficult to achieve with regular MRI (6). For the first time, using MR images of 1-mm slices and tractography of the midbrain, we clearly showed that the lesion in the present case was located at the rostral extremity of the decussation. The fibers originated from the dorsal side (cerebellar side) of the decussation, crossed the midline and ran toward the contralateral ventral side (red nucleus side). Therefore, such lesions located at the height of the rostral extremity of the decussation touching the dorsal side may damage the fibers before the crossing and thus induce ipsilateral cerebellar ataxia. Although this case involves only the third reported patient, this entity should perhaps be recognized as a new syndrome, despite its rarity.

Should the lesion extend further ventral or caudal, affecting a wide area of the decussation of the superior cerebellar peduncle, it may damage the fibers both before and after the crossing, thus causing bilateral cerebellar ataxia. In fact, such cases have previously been reported (7). Mossuto-Agatiello (7) reported five patients with unilateral lower midbrain infarction of the decussation of the brachium conjunctivum (caudal paramedian midbrain syndrome). Bilateral ataxia, prevalent on one side, was the most characteristic and most constant clinical feature, while eye movement disorders were nonspecific (7).

In the present case, MRA revealed severe stenosis in the P1 region of the right cerebral artery, and the paramedian perforating arteries originating at this point were considered to be the responsible vessels (8). However, the wide individual variation observed in the distribution of penetrating branches suggests that midbrain infarction may present with a wide variety of symptoms (9).

This study is associated with several limitations. First, the patient's midbrain was small, and clearly visualizing the lesion was difficult. Second, observing the three-dimensional anatomical structure and courses of the nerve fiber bundles on superimposed MR images was also difficult. Third, it remains unconfirmed whether lesions touching the decussation of the superior cerebellar peduncle actually damage the fibers before the crossing. Finally, more cases must be accumulated in order to establish the entity causing partial oculomotor nerve palsy with ipsilateral cerebellar ataxia as a new syndrome.

**The authors state that they have no Conflict of Interest (COI).**

## References

1. Claude H, Loyez M. Ramollissement du noyau rouge. Rev Neurol

- (Paris) **24**: 49-51, 1912 (in French).
2. Seo SW, Heo JH, Lee KY, et al. Localization of Claude's syndrome. *Neurology* **57**: 2304-2307, 2001.
  3. Schwartz TH, Lycette CA, Yoon SS, Kargman DE. Clinicoradiographic evidence for oculomotor fascicular anatomy. *J Neurol Neurosurg Psychiat* **59**: 338, 1995.
  4. Verstichel P, Chia L, Meyrignac C. Paralyse du nerf moteur oculaire commun et hémisindrome cérébelleux ipsilatéral par infarctus mésencéphalique, variante du syndrome de Claude. *Rev Neurol (Paris)* **155**: 601-602, 1999 (in French).
  5. Nakanishi R, Yamanaga H, Okumura C, Murayama N, Ideta T. A quantitative analysis of ataxia in the upper limbs. *Rinsho Shinkeigaku (Clin Neurol)* **32**: 251-258, 1992 (in Japanese, Abstract in English).
  6. Hong JH, Jang SH. The usefulness of DTI for estimating the state of cerebellar peduncles in cerebral infarct. *NeuroRehabilitation* **26**: 299-305, 2010.
  7. Mossuto-Agatiello L. Caudal paramedian midbrain syndrome. *Neurology* **66**: 1668-1671, 2006.
  8. Vitošević Z, Marinković S, Četković M, Štimec B, Todorović V, Kanjuh V, Milisavljević M. Intramesencephalic course of the oculomotor nerve fibers: microanatomy and possible clinical significance. *Anat Sci Int* **88**: 70-82, 2013.
  9. Kaya AH, Dagainar A, Ulu MO, et al. The perforating branches of the P1 segment of the posterior cerebral artery. *J Clin Neurosci* **17**: 80-84, 2010.
  10. Mai JK, Paxinos G. *The human nervous system*. Academic Press, 2012: 262-299.