

A Small Dorsal Pontine Infarction Presenting with Total Gaze Palsy Including Vertical Saccades and Pursuit

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A small localized infarction in the dorsal pontine area can cause various eye-movement disturbances, such as abducens palsy, horizontal conjugate gaze palsy, internuclear ophthalmoplegia, and one-and-a-half syndrome. However, complete loss of vertical saccades and pursuit with horizontal gaze palsy has not been reported previously in a patient with a small pontine lesion. We report a 67-year-old man with a small dorsal caudal pontine infarct who exhibited total horizontal gaze palsy as well as loss of vertical saccades and pursuit.

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A small localized dorsal pontine infarction can produce abducens palsy, horizontal conjugate gaze palsy, internuclear ophthalmoplegia (INO), and one-and-a-half syndrome by damaging the abducens nucleus and its fascicle, the paramedian pontine reticular formation (PPRF), or the medial longitudinal fasciculus (MLF).¹ However, complete ophthalmoplegia in an isolated pontine lesion has not been described previously. We report a case of complete ophthalmoplegia with facial diplegia caused by a small localized dorsal caudal pontine infarction.

CASE REPORT

A 67-year-old man was referred for evaluation of gaze disturbances and facial diplegia. Two weeks prior

to admission he had experienced sudden general weakness for approximately 20 minutes without loss of consciousness while working on his farm. The following day, the patient experienced dysarthric speech and visual obscuration, and his family members noticed that his eyes were deviated to one side. The next day he was admitted to a local hospital, and diagnosed as having a pontine infarction. During the admission at the local hospital, his neurological symptoms did not progress until he experienced sudden bilateral facial weakness 11 days after the initial symptom onset. He was referred to our hospital for further management. He denied any history suggestive of recent upper respiratory or gastrointestinal infection. He was a social drinker and current smoker with a 40-pack/year history. He also had hypertension.

A neurological examination revealed that he was

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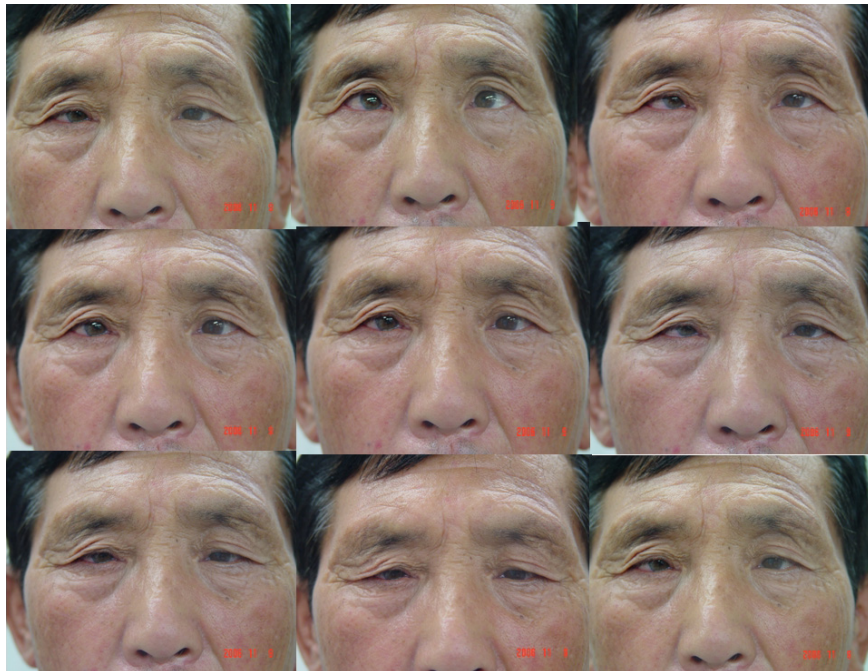


Figure 1. Photographs of eye motion in each cardinal directions demonstrate complete voluntary gaze palsy.

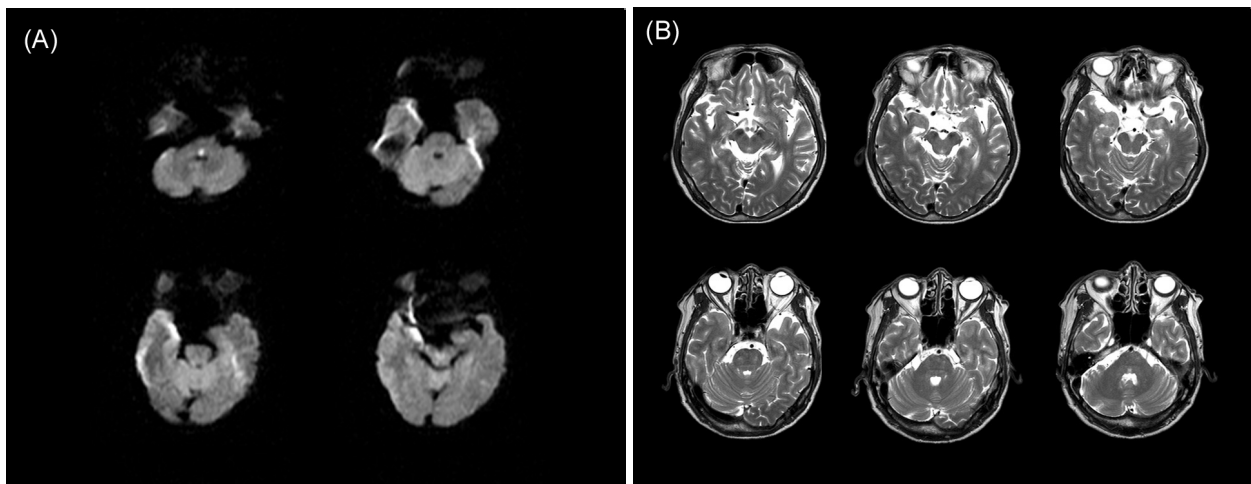


Figure 2. Initial diffusion-weighted MRI (A) and follow-up T2-weighted MRI (B) show a midline pontine infarction at the level of the abducens nuclei.

alert and fully oriented. He showed esotropia of both eyes and complete paralysis of smooth pursuit and saccades in both the horizontal and vertical directions (Fig. 1, The patient signed a consent form.). Optokinetic stimuli elicited no horizontal or vertical movements. The oculocephalic maneuver did not induce horizontal eye movements. However, the vertical vestibulo-ocular reflex (VOR) and convergence were considered intact. He also showed facial diplegia and mild dysarthria. Other findings of a neurological examination were

normal.

Initial diffusion-weighted MRI performed 1 day after the initial symptom onset (7-mm-thick slices, 1.5 tesla) revealed an acute small infarction in the dorsal caudal pons at the level of the abducens nucleus (Fig. 2-A). Follow-up T2-weighted MRI performed 19 days after the initial symptom onset (4-mm-thick slices, 3.0 tesla) revealed a high signal intensity in the same location (Fig. 2-B). The results of a cerebrospinal fluid examination and nerve conduction studies

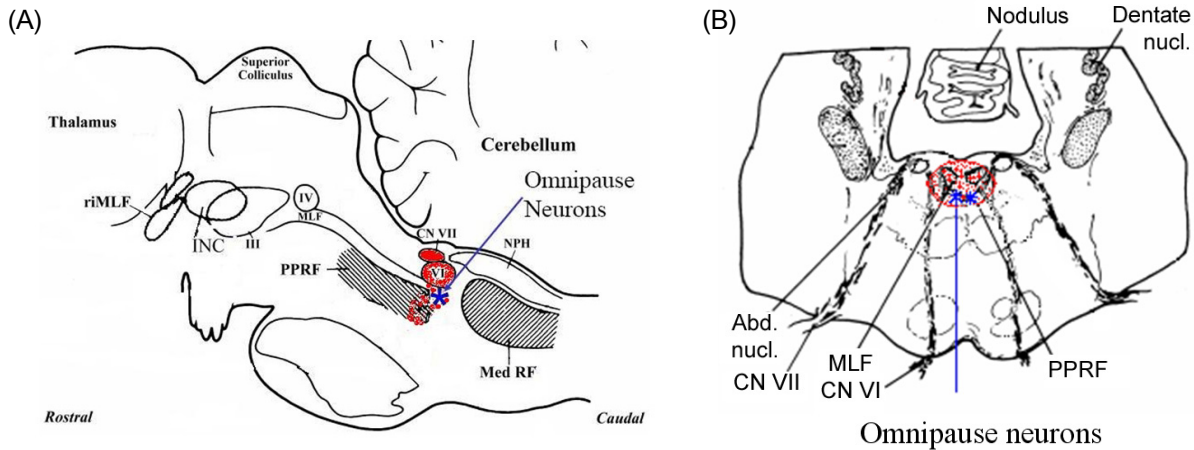


Figure 3. Neural substrates (A, sagittal view of brainstem B, axial view of lower pons) for eye movement. Involved lesions (red color) in this patient may have been omnipause neurons in the nucleus raphe interpositus, bilateral fascicles of facial nuclei, MLF, bilateral abducens fascicles, and/or PPRF (Asterisk indicates the location of omnipause neurons in the nucleus raphe interpositus; III, oculomotor nucleus; IV, trochlear nucleus; IV, abducens nucleus; INC, interstitial nucleus of Cajal; NPH, nucleus interpositus hypoglossi; Modified from Leigh and Zee¹).

were normal. Serum anti-GQ1b IgG and IgM antibodies were negative. He was given antiplatelet agents. His vertical gaze palsy and facial diplegia had improved 2 months later, while the esotropia and horizontal gaze palsy remained unchanged.

DISCUSSION

Our patient presented with complete voluntary gaze palsy and facial diplegia. The infarction was located in the dorsal caudal pons between the facial colliculi. This location could indicate damage to the abducens or facial nuclei, or any pathway traveling through the dorsal pontine tegmentum. The pontine tegmentum contains neural structures for controlling horizontal eye movements, including the abducens nucleus and fascicle, PPRF, and MLF.¹ A pontine tegmental infarction frequently impairs horizontal gaze. In view of the concurrent impairment of the horizontal VOR², damage to bilateral MLF and fascicles of abducens nuclei may have caused the bilateral horizontal gaze palsy in our patient. In addition to the above mechanism, bilateral PPRF lesions can also induce total horizontal gaze palsy including impaired horizontal saccades, horizontal VOR, and pursuit.

This patient showed esotropia of both eyes in primary gaze. This was probably due to additional damage to the bilateral abducens fascicles rather than an in appropriate MLF vergence signal on the medial rectus motoneurons³ (which produces abnormal convergence), because no mesencephalic lesions were found in follow-up MRI (4-mm-thick slices, 3.0 tesla).

Bilateral pontine lesions may also impair vertical saccades, especially slow vertical saccades in the presence of a discrete pontine lesion because omnipause cells project to both horizontal burst neurons in the pons and vertical burst neurons in the midbrain.⁴⁻⁶ However, complete voluntary gaze palsy involving the saccades and smooth pursuit has not been reported previously in focal pontine tegmental stroke.

Vertical gaze palsy is a typical finding of a lesion in the pretectum, which contains burst neurons (rostral interstitial nucleus of the MLF [riMLF]) and the neural integrator (interstitial nucleus of Cajal) for vertical and torsional eye movements. However, our patient had a small lesion in the pontine tegmentum¹ (Fig. 3). Vertical saccadic palsy in our patient may have been attributable to the damage to the omnipause neurons or to disruption of the pathways from the omnipause neurons to the riMLF. Omnipause neurons are located in the nucleus raphe interpositus at the

level of the abducens nucleus and project to burst neurons in the PPRF or riMLF, which they tonically inhibit.¹ They cease discharging just before saccade onset and during saccades.⁴ Experimentally induced damage to the omnipause neuron slows both horizontal and vertical saccades.⁶ Our patient also showed impairment of vertical smooth pursuit. The lesion in our patient was located in the pontine tegmentum where the MLF resides, even though the findings of INO may have been masked by the complete horizontal gaze palsy. The discharge rates of some MLF fibers reflect the vertical head or eye position,⁷ and disruption of these fibers may have been responsible for the impaired vertical smooth pursuit in our patient. The sparing of the vertical VOR in the early phase in our patient is consistent with results of previous studies that extra-MLF pathways can be involved in the vertical VOR⁸ or the early compensatory nature of the VOR. Nonetheless, an anterior canal pathway is known to be an extra-MLF pathway, which in this patient might have spared the downward but not the upward VOR. However, we could not find abnormalities such as downward limitation or decreased gain in the VOR, because electrooculography was not performed. Facial diplegia and ophthalmoplegia may develop in a variant of Guillain-Barre syndrome or in Fisher's syndrome.⁹ However, this might be excluded by normoactive deep tendon reflexes, the absence of ataxia, normal nerve conduction studies, and the absence of serum anti-GQ1b antibody.

This patient had interesting ocular motor findings of total horizontal gaze palsy and loss of vertical saccades and pursuit in the acute phase, and subsequently improved vertical gaze palsy. We consider

that these distinctive ocular motor findings of our patient were indicative of the involvement of lesions of omnipause neurons in the nucleus raphe interpositus, bilateral fascicles of facial nuclei, MLF, bilateral abducens fascicles, and/or PPRF.

REFERENCES

1. Leigh RJ, Zee DS. *The neurology of eye movements*. Fourth edn. New York: Oxford University Press, 2006.
2. Pierrot-Deseilligny C, Goasguen J. Isolated abducens nucleus damage due to histiocytosis X. Electro-oculographic analysis and physiological deductions. *Brain* 1984; 107:1019-1032.
3. Gamlin PD, Gnadt JW, Mays LE. Lidocaine-induced unilateral internuclear ophthalmoplegia: effects on convergence and conjugate eye movements. *J Neurophysiol* 1989;62:82-95.
4. Hanson MR, Hamid MA, Tomsak RL, Chou SS, Leigh RJ. Selective saccadic palsy caused by pontine lesions: clinical, physiological, and pathological correlations. *Ann Neurol* 1986;20:209-217.
5. Slavin ML. A clinicoradiographic correlation of bilateral horizontal gaze palsy and slowed vertical saccades with midline dorsal pontine lesion on magnetic resonance imaging. *Am J Ophthalmol* 1986;101:118-120.
6. Kaneko CR. Effect of ibotenic acid lesions of the omnipause neurons on saccadic eye movements in rhesus macaques. *J Neurophysiol* 1996;75:2229-2242.
7. King WM, Lisberger SG, Fuchs AF. Responses of fibers in medial longitudinal fasciculus (MLF) of alert monkeys during horizontal and vertical conjugate eye movements evoked by vestibular or visual stimuli. *J Neurophysiol* 1976;39:1135-1149.
8. Cremer PD, Migliaccio AA, Halmagyi GM, Curthoys IS. Vestibulo-ocular reflex pathways in internuclear ophthalmoplegia. *Ann Neurol* 1999;45:529-533.
9. Willison HJ, Yuki N. Peripheral neuropathies and anti-glycolipid antibodies. *Brain* 2002;125:2591-2625.