Contraversive Ocular Tilt Reaction After the Lateral Medullary Infarction

Kyoo Ho Cho, MD, Young Dae Kim, MD, PhD, Jinkwon Kim, MD, Byoung Seok Ye, MD, Ji Hoe Heo, MD, PhD, and Hyo Suk Nam, MD, PhD

Introduction: The triad of ocular tilt reaction (OTR) is conjugated ocular torsion, skewed deviation, and head tilt. A lesion involving below the inferior pons develops ipsiversive OTR, whereas a lesion above the inferior pons leads to contraversive OTR. A lateral medullary infarction usually causes ipsiversive OTR. This is the first report of contraversive OTR following the lateral medullary infarction.

Case Report: A 58-year-old woman was admitted due to sudden-onset vertigo and gait disturbance. A neurological examination revealed OTR to the left side (left head tilt, leftward ocular torsion, and skew deviation). Lateropulsion to the right and dysmetria on the right arm and leg were present. Diffusion-weighted MRI revealed an acute infarction of the right lateral medulla and the posterolateral pons. The patient slowly improved and can walk without sway after 1 week. Neither head tilt nor ataxia was observed after 3 months.

Conclusions: Lateral medullary lesion typically results in ipsilateral OTR. Contraversive OTR of our patient might be derived from the disruption of the cerebellovestibular inhibitory pathway by the lesion in the juxtarestiform body in the lateral medulla.

Key Words: ocular tilt reaction, medulla, cerebral infarction

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Ocular tilt reaction (OTR) is a type of vestibular dysfunction in the roll plane. Patients with OTR present with a characteristic triad of conjugated ocular torsion, skewed deviation, and head tilt.¹ In addition, OTR is frequently accompanied by a deviation of the subjective visual vertical field. Unilateral lesions of the graviceptive pathway from the otoliths to the midbrain can result in OTR.² Medullary lesions below the intersection of the graviceptive pathway usually results in ipsiversive OTR.³ We report a patient with the lateral medullary infarction, who subsequently developed contraversive OTR.

CASE REPORT

A 58-year-old woman was admitted with sudden-onset vertigo and gait disturbance. She had been treated for hypertension for the previous 3 years. The blood pressure and heart rate were 200/92 mm Hg and 79/minute, respectively. Neurological examination revealed a leftward OTR. The patient's head was tilted toward the left side and counterclockwise cyclotorsion was observed (Fig. 1A). The degree of

intorsion in the right eye was 3.5 degrees and the degree of extorsion in the left eye was 16.2 degrees (Fig. 1B). The patient's subjective visual vertical field was deviated to the left side (from 4 trials: +4.6 degrees/ +4.3 degrees/+3.1 degrees/+2.0 degrees; the average was +3.5 degrees, normal is ± 1.8 degrees). A skew deviation was present with left hypotropia. Gaze evoked nystagmus was shown during the right lateral gaze. The patient's pupil sizes and reflexes to light were normal. No facial palsy, facial sensory changes, or hearing impairments were detected. Motor sensory examination and the deep tendon reflexes were normal. Lateropulsion was present; she fell to the right side when she attempted the tandem gait. Dysmetria was shown in the right arm and leg. Diffusion-weighted MRI revealed acute infarctions on the posterolateral regions of the right medulla and pons (Figs. 1C, D). MR angiography, carotid ultrasonography, and transcranial Doppler assessments were normal. The patient's EKG was normal and transesophageal echocardiography revealed the presence of patent foramen ovale. Total cholesterol (6.7 mmol/L) and low-density lipoprotein (4.9 mmol/L) levels were high. After 1 week, the patient slowly improved and can walk without sway. Neither head tilt nor ataxia was observed after 3 months.

DISCUSSION

Our patient showed contraversive OTR after brainstem infarction involving the medullar and the pons. Development of ipsiversive OTR after the lateral medullary infarction of Wallenberg syndrome is well known. However, the development of contraversive OTR from the lateral medullary infarction has not been reported.

OTR may result from irritative or destructive lesions of the central or peripheral graviceptive vestibular pathways.² A lesion involving below the inferior pons develops ipsiversive OTR, whereas a lesion above the inferior pons leads to contraversive OTR.³ Graviceptive information, which is perceived by the vertical semicircular canal and the otolith organ, is delivered to the vestibular nucleus, and the information crosses to the opposite side in the inferior pons and the medial longitudinal fasciculus, which is connected to the interstitial nucleus of Cajal. Stimulation of the vestibular nucleus on one side elicits OTR on the opposite side and stimulation on the interstitial nucleus of Cajal elicits an ipsilateral OTR.

A contraversive OTR had been reported after unilateral cerebellar lesions; the OTR may be under inhibitory control of the posterior cerebellum, possibly the nodulus. Mossman and Halmagyi⁴ described 2 patients with OTR that developed following cerebellar stroke involving the nodulus. They speculated that interruption of the nodular inhibitory projections to the ipsilateral vestibular nuclei might have accounted for the contraversive OTR. Disinhibition of the vestibular nucleus can increase in the activity of the ipsilateral graviceptive pathway, consequently producing contraversive OTR.^{5,6} Because recent report demonstrated that the dentate nucleus is also crucial for contraversive OTR in the cerebellar stroke, the dentate nucleus may be one of relay site between the cerebellum and the vestibular nucleus.⁷

From the Department of Neurology, Yonsei University College of Medicine, Seoul, Korea.

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Reprints: Hyo Suk Nam, MD, PhD, Department of Neurology, Yonsei University College of Medicine, 50-1 Yonsei-ro, Seodaemun-gu, Seoul 120-752, Korea. E-mail: hsnam@yuhs.ac.

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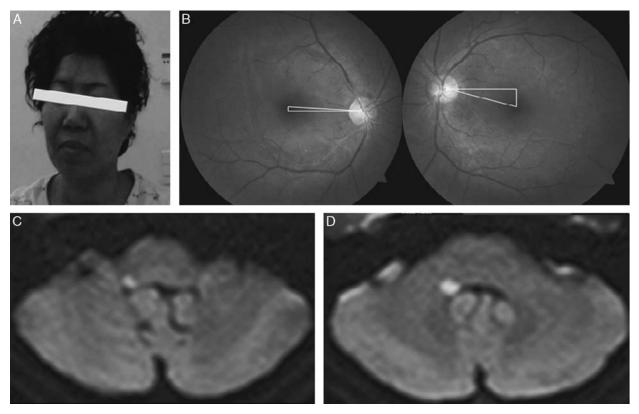


FIGURE 1. A, The patient showed head tilt to the left side. B, Fundus photography revealed 3.5-degree intorsion of the right eye and 16.2-degree extorsion of the left eye. Diffusion-weighted MRI revealed acute infarctions on right posterolateral regions of the medulla (C) and pons (D).

Taken together, lesion involving the cerebellovestibular inhibitory pathway can cause the contraversive OTR. The medullary lesion of our patient might be the inferior cerebellar peduncle (ICP), which connects the cerebellum with the medulla and the spinal cord. The ICP has 2 parts: the restiform body and the juxtarestiform body. The juxtrarestiform body is lying just medial to ICP. The restiform body is afferent system and the juxtarestiform body is mainly efferent. Primary component of the juxtarestiform body is the cerebellovestibular fibers from the flocculonodular lobe and the vermis.8 In our patient, it can be postulated that the lesion involving the juxtarestiform body in the lateral medulla causes disruption of the cerebellovestibular inhibitory pathway and results in contraversive OTR. Our patient had another lesion in the pons; the contraversive OTR can be elicited by the pontine lesion involving the medial longitudinal fasciculus. Because the pontine lesion of our patient was far from the midline, the possibility of the contraversive OTR by the lateral pontine lesion might be low.

The vascular territory of our patient was the anterior inferior cerebellar artery (AICA) area. The AICA supplies the middle cerebellar peduncle, the inferolateral portion of pons, the anterior-inferior cerebellum, and the flocculus. While the AICA usually supplies the flocculus, other areas are more variable and depend on the dominance of the AICA, superior cerebellar artery and posterior inferior cerebellar artery. The inferolateral pons is usually involved in most patients experiencing AICA infarctions. The infarctions by AICA occlusion sometimes extends up to the middle third of the lateral pons and down to the superior part of the lateral medulla.^{9,10} A recent report on OTR following AICA infarction revealed that 9 of 12 patients experiencing AICA infarctions presented with ocular

torsion. Six patients showed ipsiversive ocular torsion, and the remaining 3 patients showed contraversive ocular torsion.¹¹ In patients with ipsiversive ocular torsion, hearing loss and skew deviation were observed. This may be explained by the fact that the internal auditory artery branched from the AICA and supplies the inner ear.5,12 In contrast, none of the patients with contraversive ocular torsion presented with head tilts, skew deviation, or hearing loss. The authors suggested that a lesion in the flocculus might be more likely to result in contraversive ocular torsion. However, in our patient, no cerebellar lesion including the flocculus was found. It can be speculated that a lesion in the lateral medulla, which is supplied by the AICA, can cause contraversive OTR. Our patient showed head tilt and skew deviation, but all previous 3 patients with contraversive ocular torsion did not have either head tilt or skew deviation.11 Differences of the lesion location from previous report might be associated with the presence of head tilt and skew deviation.

CONCLUSIONS

This is the first report of contraversive OTR following the lateral medullary infarction. Contraversive OTR shown in our patient might be derived from the disruption of the cerebellovestibular inhibitory pathway by the lesion in the juxtarestiform body in the lateral medulla.

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