A Case of Internuclear Ophthalmoplegia with Transient Rotatory Nystagmus in Facial Colliculus Infarction

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Background: Although internuclear ophthalmoplegia (INO) is a definite sign of an intrapontine or mesencephalic lesion, INO is rarely associated with rotatory nystagmus in pontine lesions. We experienced a case of INO with transient rotatory nystagmus in facial colliculus infarction.

Case Report: An 83-year-old male patient was admitted for acute vertical diplopia. Neurological examination revealed bilateral INO with transient ipsiversive rotatory nystagmus and ipsilateral peripheral type facial palsy. Diffusion weighted image revealed focal infarction in the right facial colliculus. Ocular symptoms were improved within one month.

Conclusion: We report a case of bilateral INO with transient rotatory nystagmus in right facial colliculus infarction.

KEY WORDS: Internuclear ophthalmoplegia · Transient rotatory nystagmus · Facial colliculus infarction.

Introduction

A lesion of the medial longitudinal fasciculus (MLF) results in an ipsilateral adduction deficit, and a contralateral abducting nystagmus and is often called internuclear ophthalmoplegia (INO).1 The interneurons of the MLF are intermixed with the abducens neurons in the sixth-nerve nucleus, which lies dorsally in the pons near the genu of the seventh cranial nerve. A lesion in the facial colliculus produces a combination of INO and peripheral facial palsy. However, INO associated with rotatory nystagmus is rare in pontine lesions and only a few cases have been reported in the literature.2-5 Here we reported a case of INO with transient rotatory nystagmus accompanied by peripheral facial palsy in facial colliculus infarction.

Case Report

An 83-year-old male patient with hypertension and diabetes mellitus was admitted to our neurology department due to vertical diplopia that had developed suddenly. His initial blood pressure was 145/65 mm Hg, blood glucose was 175 mg/dL, and Hb A1c was 9.8%. Ocular examination showed bilateral pupils of normal size with prompt direct light reflexes. But bilateral adduction palsy with normal convergence, rightward gaze limitation with clockwise rotatory nystagmus in the right eye and horizontal nystagmus in the left eye on leftward gaze were observed (Fig. 1). Right peripheral type facial palsy was also present. No other neurologic abnormalities were noted, and skew deviation and head tilting were not observed. On the first day of admission, diffusion weighted image showed acute focal infarction in the right lower pons with restricted diffusion on the apparent diffusion coefficient map and corresponding high signal intensity on T2 weighted image/fluid-attenuated inversion recovery imaging (Fig. 2). He was prescribed a regimen of aspirin and glimepiride. The bilateral INO with ipsiversive rotatory nystagmus had resolved on the sixth day after symptom onset. The ipsilateral lateral gaze limitation and peripheral facial palsy persisted for one month (Fig. 3).

Discussion

INO has diagnostic value in determining the site of the lesion. The pontine center for lateral eye movement regulates lateral gaze via innervation of the ipsilesional lateral rectus muscle and the contralesional medial rectus through the MLF. Unilateral lesions of the MLF between the midpons and the oculomotor nucleus disconnect the ipsilateral medial rectus
subnucleus, causing adduction failure during horizontal gaze. These ocular findings associated with abduction nystagmus of the contralateral eye are collectively referred to as INO. INO is frequently accompanied by a variety of other neurologic deficits due to extension of the MLF lesion into adjacent brainstem structures. A lesion that involves the sixth-nerve nucleus, facial nerve fibers and the interneurons of the MLF near the facial colliculus produces the combination of ipsilateral adduction palsy, abduction limitation and peripheral facial palsy in INO. However, INO is rarely associated with rotatory nystagmus. Lesions responsible for rotatory nystagmus with INO are usually located in the MLF above the level of the abducens nucleus and below the level of the trochlear nucleus. An MLF lesion could inactivate the interstitial nucleus of Cajal (INC). The INC is the integrator for ipsiversive rotatory movements and is situated between the red nucleus and the superior colliculus. The INC receives excitatory inputs from the vertical semicircular canals of the contralateral labyrinth via the MLF. Projections from the vestibular nuclei or vestibulocerebellum to the INC coordinate torsional gaze. It has been suggested that INC inactivation produces contralesional torsional deviation and rotatory ipsilesional nystagmus. MRI of our patient revealed a focal infarction in the right dorsomedial portion of the pontine tegmentum. There were no lesions in the vestibular nuclei or INC. His bilateral adduction gaze palsy and rotatory nystagmus resolved within one week. We suspect that a small facial colliculus lesion involving the MLF may have been responsible for the inactivation of the ipsilateral vertical integrator (the INC), producing INO associated with a transient ipsiversive rotatory nystagmus. In contrast, his ipsilateral lateral gaze limitation and peripheral facial palsy were maintained for one month. It may result from involvement of the abducens nerve nucleus and facial nerve fibers.
REFERENCES