

Combined Ipsilateral Oculomotor Nerve Palsy and Contralateral Downbeat Nystagmus in a Case of Cerebral Infarction

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Key Words

Monocular downbeat nystagmus · Oculomotor nerve palsy · Infarction · Superior cerebellar peduncle

Abstract

We report a patient with acute cerebral infarction of the left paramedian thalamus, upper mesencephalon and cerebellum who exhibited ipsilateral oculomotor nerve palsy and contralateral downbeat nystagmus. The site of the infarction was considered to be the paramedian thalamopeduncular and cerebellar regions, which are supplied by the superior cerebellar artery containing direct perforating branches or both the superior cerebellar artery and the superior mesencephalic and posterior thalamosubthalamic arteries. Contralateral and monocular downbeat nystagmus is very rare. Our case suggests that the present downbeat nystagmus was due to dysfunction of cerebellar-modulated crossed oculovestibular fibers of the superior cerebellar peduncle or bilateral downbeat nystagmus with one-sided oculomotor nerve palsy.

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Introduction

Monocular downbeat nystagmus is a very rare manifestation of combined nuclear-supranuclear ophthalmoparesis that is seemingly a secondary dysfunction of cerebellar-modulated crossed oculovestibular fibers of the superior cerebellar peduncle, integrator

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neurons, or posterior commissure crossing fibers originating at the interstitial nucleus of Cajal and mediating vertical ocular reflexes [1]. However, the exact responsible lesion site has not yet been determined [2, 3]. Here we report a patient with acute unilateral infarction of the paramedian thalamus, upper mesencephalon and cerebellum who exhibited ipsilateral oculomotor nerve palsy and contralateral downbeat nystagmus.

Case Report

An 80-year-old man with a history of atrial fibrillation and hypertension was admitted to our hospital because of sudden onset of consciousness disturbance and right hemiparesis. On neurological examination, he was in a stupor with a Glasgow Coma Scale score of 7 (E1V2M4). He had left oculomotor nerve palsy involving mydriasis, the levator, superior, inferior and medial rectus muscles, and right oculomotor nerve palsy involving the levator and superior rectus muscles (fig. 1). Both pupils were unresponsive to light. Downbeat nystagmus of the right eye was found. There was no left eye nystagmus. Attempted convergence of the eyes and head rotation did not increase nystagmus. He also had dysphagia, right hemiparesis and ataxia with a National Institutes of Health Stroke Scale score of 29 in the emergency room.

Laboratory examination revealed mild elevation of LDH, γ -GTP and CRP. Diffusion-weighted magnetic resonance imaging on admission showed high-signal lesions in the left paramedian thalamus, hypothalamus, upper mesencephalon and cerebellum hemisphere containing the superior cerebellar peduncle (fig. 2, arrows). Magnetic resonance angiography did not reveal any abnormality. We diagnosed him with cardiac embolism due to atrial fibrillation. A free radical scavenger, edaravone, and glyceol were administered [4]. His consciousness gradually cleared to a Glasgow Coma Scale score of 14 (E3V5M6) and he became able to communicate 22 days after onset. Dysphagia remained and endoscopic gastrostomy was performed 23 days after onset. Warfarin 3.5 mg/day was initiated from 28 days after onset and PT-INR rose to 1.90 37 days after onset. He was discharged to another hospital for rehabilitation and his monocular downbeat nystagmus lasted approximately 39 days after onset.

Discussion

The present case showed acute unilateral infarction of the paramedian thalamus, upper mesencephalon and cerebellum hemisphere and exhibited ipsilateral oculomotor nerve palsy and contralateral monocular downbeat nystagmus, ptosis and superior rectus muscle disturbance. The site of the infarction was considered to be the paramedian thalamopeduncular and cerebellar regions, which are supplied by the superior cerebellar artery containing direct perforating branches or both the superior cerebellar artery and the superior mesencephalic and posterior thalamosubthalamic arteries [5, 6]. The mesencephalon lesion was located in the paramedian area. The bilateral ptosis and upward gaze palsy were considered to have been caused by a lesion involving the unilateral oculomotor nucleus due to the crossed innervation [7, 8].

Downbeat nystagmus is usually bilateral and is associated with cerebellar ectopia, degeneration, cerebellar vermis hematoma, encephalitis, magnesium depletion, alcohol, lithium, anticonvulsant intoxications or brainstem infarction [9–11]. In contrast, monocular downbeat nystagmus is very rare, and only three cases have been reported in whom ocu-

lomotor nerve palsy in one eye was associated with downbeat nystagmus on the other side. The three previous cases had unilateral paramedian thalamopeduncular infarction and showed monocular downbeat nystagmus in the eye contralateral to the mesencephalon lesion (table 1). Two mechanisms are adduced to explain monocular downbeat nystagmus: (1) Monocular downbeat nystagmus is produced by a unilateral central nervous system lesion. (2) Alternatively, bilateral downbeat nystagmus manifests as monocular downbeat nystagmus because of oculomotor nerve palsy in the other eye. Jacome [3] proposed that monocular downbeat nystagmus was secondary to dysfunction of cerebellar-modulated crossed oculovestibular fibers of the superior cerebellar peduncle. Oishi and Mochizuki's report [12] suggested that downbeat nystagmus in the left eye was masked by complete oculomotor nerve palsy. The site of the present infarction included both the paramedian thalamopeduncular region and the superior cerebellar peduncle. Both mechanisms might be appropriate in our case. The accumulation of further clinical cases is needed.

Disclosure Statement

The authors state that they have no conflicts of interest. They have no financial disclosure to make.

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Table 1. Clinical features of patients with ipsilateral oculomotor nerve palsy and contralateral downbeat nystagmus

Case	Age/sex	Etiology	Responsible lesion	Symptoms	Prognosis	Reference
1	62/M	infarction	thalamus, mesencephalon	ipsilateral oculomotor nerve palsy, contralateral downbeat nystagmus, hemiparesis	good	Jacome [3]
2	66/F	infarction	thalamus, mesencephalon	ipsilateral oculomotor nerve palsy, contralateral downbeat nystagmus, hemiparesis	poor	Jacome [3]
3	53/F	infarction	thalamus, mesencephalon	ipsilateral oculomotor nerve palsy, contralateral downbeat nystagmus, hemiparesis, ataxia	poor	Oishi and Mochizuki [12]
4	80/M	infarction	thalamus, mesencephalon, cerebellum	ipsilateral oculomotor nerve palsy, contralateral downbeat nystagmus, hemiparesis, ataxia	poor	our case



Fig. 1. Our patient showed left oculomotor nerve palsy involving mydriasis, the levator, superior, inferior and medial rectus muscles, and right oculomotor nerve palsy involving the levator and superior rectus muscles.

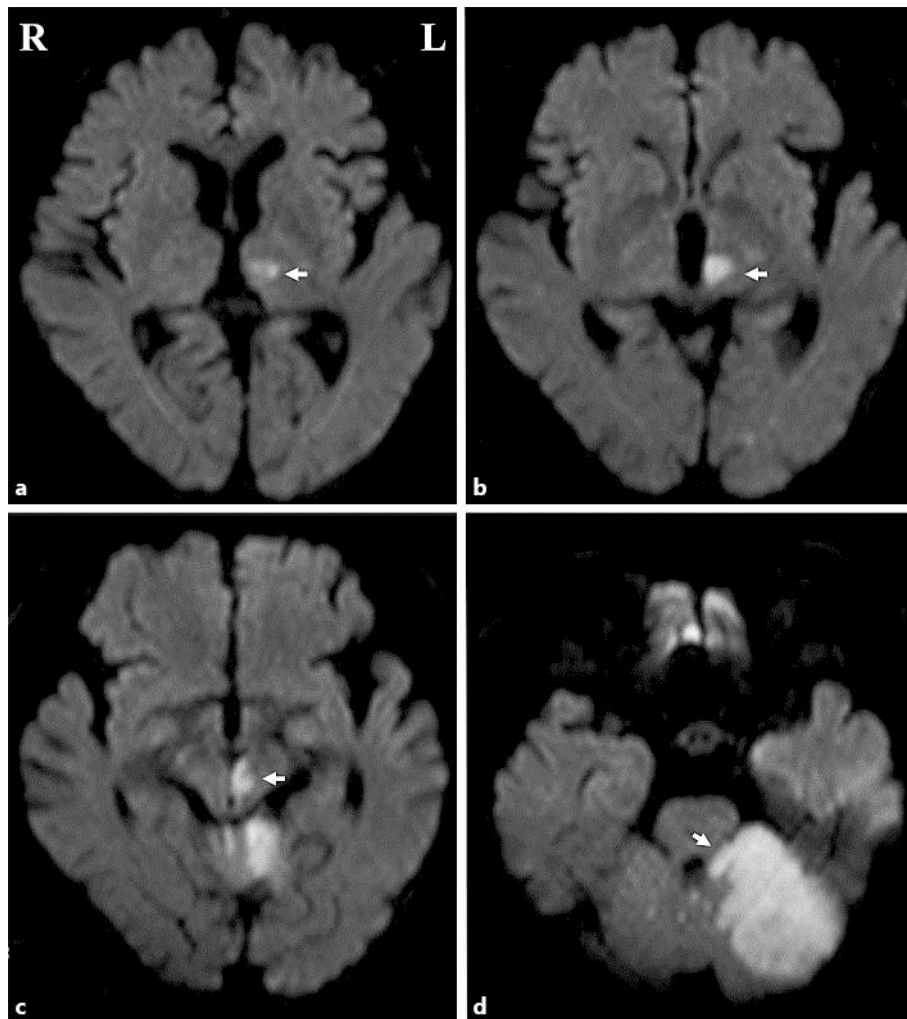


Fig. 2. Diffusion-weighted magnetic resonance imaging on admission showed increased signal intensity in the left paramedian thalamus (a, arrow), hypothalamus (b, arrow), upper mesencephalon (c, arrow) and cerebellum hemisphere including the superior cerebellar peduncle (d, arrow).