

Divergence Paralysis Caused by Acute Midbrain Infarction

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Abstract

A 41-year-old woman with idiopathic thrombocytopenic purpura and dyslipidemia abruptly developed vertigo, truncal ataxia and divergence paralysis. Cranial magnetic resonance imaging demonstrated the presence of infarction in the left superior paramedian mesencephalic artery involving the vicinity of the periaqueductal gray matter. The symptoms rapidly resolved under the administration of anti-platelet agents. The precise location of the hypothetical divergence center of the ocular motor system remains unclear because the lesions responsible for divergence paralysis are rarely identified on neuroimaging. We emphasize that this is a first reported case of DP caused by acute midbrain infarction and speculate that the mesencephalic reticular formation may be partially involved.

Key words: divergence center, horizontal diplopia, idiopathic thrombocytopenic purpura, ophthalmoplegia, periaqueductal gray matter (mesencephalic central gray)

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Introduction

Divergence paralysis (DP) is characterized by uncrossed horizontal diplopia at far distances with no or minimal deviation at near distances (1-6). In addition, despite the absence of restricted ocular movement, concomitant esotropia in all fields of gaze has been observed (1-6). However, the precise location of the hypothetical divergence center of the ocular motor system (DC) remains uncertain because only six cases of localized lesions responsible for DP identified on neuroimaging have been reported in the English language literature (1-5). Among these cases, Tsuda et al. (1, 2) recently reported two cases of DP caused by old infarctions in the vicinity of the periaqueductal gray matter (PAG) that were detected on magnetic resonance (MR) imaging. We herein describe an additional case of DP secondary to an acute midbrain infarction.

Case Report

A 41-year-old Japanese woman with dyslipidemia, idiopathic thrombocytopenic purpura (ITP) and a smoking habit abruptly developed vertigo, gait disturbance and hori-

zontal diplopia only at far viewing distances in March 2012. The corrected visual acuities were 1.5 in both eyes. The fundoscopic findings did not demonstrate any abnormalities in either eye. The patient's pupil diameter was 3 mm bilaterally in the lightened room. Pupil responses to light and near distances were prompt in both eyes. The palpebral aperture measured 9 mm bilaterally. In the primary position, a 5.5 prism diopter (PD) of esotropia at 0.5 m and a 10 PD of esotropia at 5 m were observed. Despite the absence of any restriction of ocular movements (Fig. 1), the red glass test demonstrated concomitant esotropia in all fields of gaze. The Maddox rod test demonstrated normal ocular torsion. Based on these results, the patient was diagnosed as having DP. Furthermore, truncal ataxia with the patient primarily falling backward was observed, although no limb ataxia, dysarthria or nystagmus were noted. There were no other neurological abnormalities, except for DP and truncal ataxia. A complete blood cell count revealed a decreased platelet count of 71,000/ μ L. Blood chemistry demonstrated dyslipidemia. The patient's thyroid function was within normal ranges. Electrocardiogram, chest radiography and carotid ultrasound imaging demonstrated normal findings. On cranial magnetic resonance (MR) imaging, normal findings were observed on diffusion-weighted images; however, fluid at-

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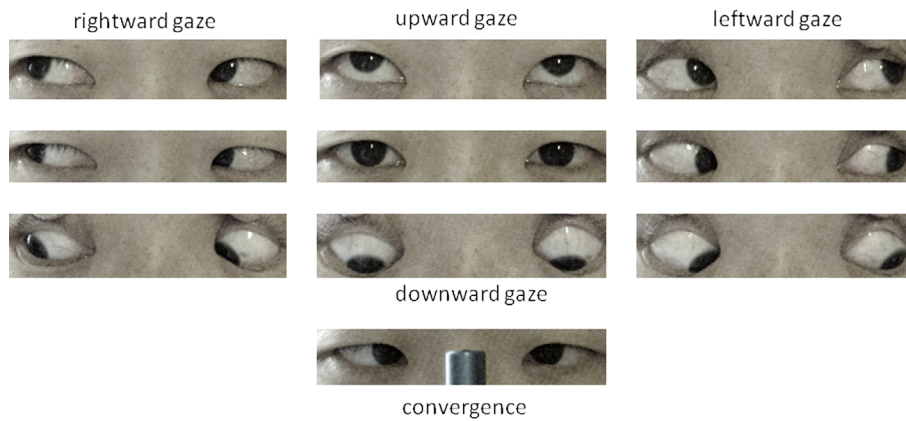


Figure 1. In the primary position, slight esotropia was observed. Ocular movement was not restricted.

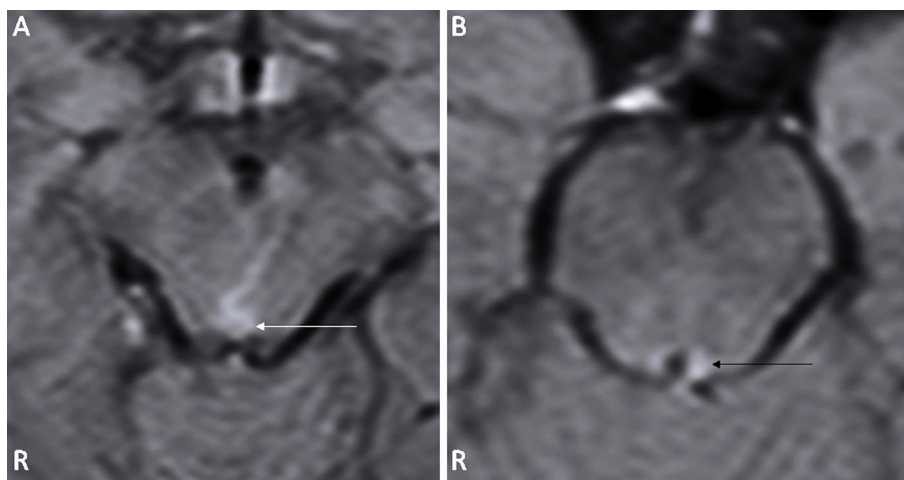


Figure 2. Cranial magnetic resonance imaging on fluid-attenuated inversion recovery images demonstrated the presence of a high-intensity lesion in the territory of the left superior paramedian mesencephalic artery (A, white arrow) that involved the left-sided region of the periaqueductal gray matter (B, black arrow).

tenuating inversion recovery images demonstrated the presence of a high-intensity lesion in the territory of the left superior paramedian mesencephalic artery involving the PAG (Fig. 2). Orbital MR imaging and cranial MR angiography demonstrated normal findings. Thereafter, following the administration of intravenous sodium ozagrel (anti-platelet agent) at a dose of 160 mg/day, the vertigo and truncal ataxia disappeared within one day and the DP disappeared within three days. Three months later, cranial MR imaging demonstrated no abnormalities in the brainstem.

Discussion

A cerebral infarction is a rare, recognized complication of ITP, although its precise etiology remains unknown (7).

Lepore (6) has reported cases of DP secondary to brainstem infarctions and stated that DP might be a non-localizing symptom of brainstem damage because no lesions responsible for DP have been identified. On the other hand, Stern et al. (3) described a case of DP associated with left

abducens nerve palsy secondary to a hematoma in the caudal pons close to the floor of the fourth ventricle. Lee et al. (4) noted a case of isolated DP caused by a localized hematoma in the upper pontine tegmentum extending to the midbrain. Moreover, Tsuda et al. (5) noted cranial MR imaging findings in two cases of isolated DP secondary to multiple sclerosis and acute pontine infarction, respectively, and concluded that the nucleus reticularis tegmenti pontis might be responsible for divergence of the eyes. In contrast, based on a postmortem examination, Bender et al. (8) noted that a small cavernous hemangioma was detected as a lesion responsible for DP in the right-sided region of the PAG at the level between the superior and inferior colliculi. Furthermore, based on the results of stereotactic surgery to destroy the dorsolateral tegmentum extending laterally from the midbrain aqueduct to the lateral border of the midbrain, Nashold et al. (9) wrote that the neurophysiological mechanism responsible for the divergence of the eyes might be located in the midbrain tegmentum. Recently, based on two cases of DP caused by old infarctions, Tsuda et al. (1, 2) speculated

that the DC might be located in the vicinity of the PAG. In both cases, the old infarct lesions were located in the left-sided and posterior regions of the PAG (1, 2). However, in reported human cases (1, 2, 8, 9), the etiology of DP secondary to midbrain tegmentum involvement has not yet been discussed in detail. Based on a primate (rhesus monkeys) study, Mays (10) reported that both convergence and divergence neurons are intermixed in the mesencephalic reticular formation just dorsal or dorsolateral to the oculomotor nucleus.

In our present case, DP was observed and a high-intensity lesion located in the left-sided region of the PAG was detected on MR imaging. Three months later, the midbrain lesion was not observed on MR imaging. Therefore, we considered the possibility that the lesion was an area of edema that developed secondary to brainstem ischemia. We believe that the mesencephalic reticular formation may be associated with divergence of the eyes, not only in rhesus monkeys (10), but also in humans. Consequently, in our present case and in previously reported cases (1, 2), we speculated that the mesencephalic reticular formation might be partially involved in addition to the PAG. On the other hand, truncal ataxia is not an unusual symptom in cases of midbrain infarction, although its precise responsible region remains unclear (11, 12).

In conclusion, we emphasize that this is a first reported case of DP caused by acute midbrain infarction.

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

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