CASE REPORT

Oculomotor Nerve Palsy Presented with Isolated Unilateral Ptosis and Minimal Upgaze Palsy

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ABSTRACT

This case with unilateral complete blepharoptosis and slight limitation of upgaze as ocular findings due to midbrain infarction represents an unusual form of intra-axial oculomotor nerve involvement. A 65-year-old man was admitted with acute unilateral ptosis of the left eyelid and gait ataxia. He had chronic hypertension in the medical history. The neurologic examination revealed that he had a complete ptosis of the left eyelid, slight limitation of elevation in the left eye, bilateral dysmetria, and gait ataxia. Magnetic resonance imaging showed acute infarction in the central part of midbrain tegmentum. Previous models for arrangement of oculomotor fascicle in the midbrain suggest that a lesion involving the most caudal fibres for levator palpebrae and superior rectus without affecting other extraocular muscles and pupillomotor fibres is the most likely aetiology.

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Introduction

Midbrain lesions affecting oculomotor nuclear complex may result in bilateral blepharoptosis secondary to damage of the central caudal nucleus.¹ Fascicular lesions of the third nerve cause unilateral blepharoptosis with complete or partial involvement of other oculomotor fibres.² Isolated complete ptosis of the eyelid with an elevation limitation caused by midbrain lesion has been rarely reported.^{2,3} We present an extraordinary case of unilateral complete blepharoptosis as the main manifestation of oculomotor nerve involvement due to midbrain infarction.

Case report

A 65-year-old man was admitted with the complaints of droopy eyelid and ataxic gait that he noticed during awakening in the morning. He had chronic hypertension in the medical history. The neurologic examination revealed a complete ptosis of the left eyelid, bilateral dysmetria, and gait ataxia. Pupillary diameters were 4 mm bilaterally, and also pupil reactions to light and near reflex were normal. His eyes were aligned in primary position, and there was no other ocular movement abnormality except

slightly limited elevation of the left eye (Figure 1). The remainder of the physical examination was unremarkable. Routine blood tests displayed increased levels of creatinine and uric acid. Electrocardiography, chest radiography, and cranial computerized tomography were normal. Magnetic resonance imaging showed an acute infarction in the left paramedian part of the midbrain tegmentum, inferior to the left red nucleus, and a chronic infarction in the left cerebellum (Figure 2). Magnetic resonance angiography of the brain and neck was unremarkable. Trans-thoracic echocardiography did not reveal any cardiac origin for cerebral embolism. Steroid treatment for gout disease and antihypertensive treatment with clopidogrel for the secondary prevention of ischaemic stroke were started. Ptosis of the left eyelid and upgaze palsy remained during his stay in our clinic, and it was unchanged at the follow-up examination, which was held 2 months after his discharge.

Discussion

Several cases with specific patterns of third nerve palsy due to fascicular involvement have been

Figure 1. Neuro-ophthalmologic examination shows complete blepharoptosis and minimal impairment of the upgaze on the left side. (A) Primary gaze, (B) primary gaze with left eyelid elevated, (C) right gaze, (D) left gaze, (E) upward gaze, (F) downward gaze.

reported previously.^{2,4–10} Hriso et al. described a patient who was presented with isolated monocular elevation paresis and ptosis, suggesting that the fibres destined to the elevators of the eye and eyelid course laterally in the midbrain.² Another case with isolated inferior oblique paresis due to lateral midbrain infarction was reported by Castro et al.⁶ Pupillary function and inferior rectus muscle were selectively spared in two cases documented by Schwartz et al. and Purvin,^{7,8} whereas they were selectively impaired in another two cases reported by Ksiazek et al.⁹

These cases suggest that a topographical arrangement of oculomotor fibres exist in the midbrain.¹⁰ Castro et al. described a two-dimensional model for

the organisation of oculomotor fibres on the horizontal plane and revised it afterwards, based on the reported cases.⁶ According to this model, the fibres for pupil, inferior rectus, medial rectus, levator palpebrae superioris, superior rectus, and inferior oblique muscles lie from medial to lateral.³ Thereafter, Ksiazek et al. proposed a rostrocaudal organisation of the oculomotor fibres in a three-dimensional model, in which the fibres for pupil are the most rostral, followed by inferior rectus, inferior oblique, medial rectus, superior rectus, and levator palpebrae superioris.⁹

The clinical findings and magnetic resonance imaging (MRI) demonstrating an acute infarction in the midbrain tegmentum were consistent with a

Figure 2. Diffusion-weighted (A), T₂ coronal (B), and T₂ axial (C) images showing an acute infarction in the middle of the left midbrain tegmentum, and T₂ axial image showing chronic infarction in the left cerebellum (D).

Figure 3. A modified illustration from Schwartz et al.⁷ showing the arrangement of the third nerve fascicle in the coronal plane. The proposed lesion location affecting the fibres for levator palpebrae and superior rectus is shown with a grey oval. IO = inferior oblique; IR = inferior rectus; SR = superior rectus; MR = medial rectus; P = pupil; LP = levator palpebrae; SCP = superior cerebellar peduncle.

partial damage of the oculomotor fascicle in this case. Based on the topographic models of the oculomotor fibres in the midbrain, we suggest that the most caudal fibres destined to levator palpebrae and by the side of it superior rectus were affected (Figure 3). Bilateral dysmetria and ataxia were consistent with the involvement of superior cerebellar peduncle, which is situated inferior to the oculomotor fascicles in the lower midbrain tegmentum. We think that chronic infarction in the left cerebellum also contributed to the development of bilateral cerebellar findings in our patient.

This case is important in terms of better understanding of the topographic model of the oculomotor fibres in the midbrain.

Declaration of interest

The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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