A Case of Bilateral Oculomotor Nuclear Palsy

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To the best of our knowledge, isolated bilateral oculomotor nuclear palsy has not yet been reported in the literature, while bilateral oculomotor nuclear palsy with more widespread rostral brainstem infarction has often been reported. We present a patient having ‘top of the basilar syndrome’ with midbrain infarction selectively involving the bilateral oculomotor nucleus. A 61-year-old woman with two episodes of vertebrobasilar infarction presented with sudden onset of bilateral ptosis. Examination revealed pronounced bilateral ptosis. In the primary position, fixation of either eye produced an approximately 50 prism diopter exotropia. Adduction of the right eye was restricted to the midline. There was moderately decreased adduction of the left eye, severe limitation of depression, and moderately decreased elevation of both eyes. Abduction of both eyes was normal. The pupils were equal, round, and reactive to light. Bilateral ptosis is suggestive of oculomotor nuclear palsy. On the basis of clinical findings alone, we could not establish whether the precise location of the lesion was all the subdivisions of the oculomotor nucleus except the Edinger-Westphal nucleus or the central caudal nucleus and bilateral fascicles. However, because axial MRI showed a small midbrain infarct in the oculomotor nucleus region, we concluded that she had an isolated, pupil-sparing, bilateral oculomotor nuclear palsy caused by midbrain infarct.

Key words: bilateral oculomotor nuclear palsy, midbrain infarct

INTRODUCTION

The major cause of oculomotor nuclear palsy is chiefly ischemia, and rarely hemorrhage, infiltration by tumor, inflammation, and brainstem compression. Isolated bilateral oculomotor nuclear infarction is theoretically possible, because small perforating branches of the basilar artery selectively supply it. The thrombotic or embolic occlusion of these small perforating branches or, less often, from occlusion of the distal portion of the basilar artery itself leads to oculomotor nuclear palsy (top of the basilar syndrome).

To the best of our knowledge, isolated bilateral oculomotor nuclear palsy has not yet been reported in the literature, while bilateral oculomotor nuclear palsy with more widespread rostral brainstem infarction has often been reported. We present a patient with midbrain infarction selectively involving the bilateral oculomotor nucleus excluding the Edinger-Westphal nucleus.

CASE REPORT

A 61-year-old woman with a previous history of
vertebrobasilar infarction was admitted at the neurology department via the emergency room because she developed sudden onset of vertigo and altered level of consciousness. She was deeply drowsy. Two days later, she recovered alertness, but complained that she could not open her eyes. She was therefore consulted to the ophthalmology department. Her medical history was diabetes mellitus, arterial hypertension, and hyperlipoproteinemia.

Examination revealed pronounced bilateral ptosis, with eyelid fissures measuring 2 mm on the right and 2 mm on the left. She could open her eyes only by lifting her lids manually. Visual acuity was 20/200 OD and 20/100 OS. The pupils were equal, round, and reactive to light. No afferent pupillary defect was noted.

In the primary position, alternate exodeviation was observed. Fixation of either eye produced an approximately 50 prism diopter exotropia. There was markedly decreased adduction of the right eye, slightly decreased adduction of the left eye, severe limitation of depression, and moderate decreased elevation of both eyes. Abduction of both eyes was normal (Fig. 1).

Doll’s eye maneuver (oculocephalic maneuver) and Bell’s phenomenon (vestibulo-ocular reflex) were all negative (Fig. 2). Two splinter hemor-

rhages were noted in the inferior portion of the optic disc in the right fundus.

Axial T2-weighted MRI revealed a small focus of increased T2 signal in the anterior portion of the median tegmental area presumably involving the caudal oculomotor nucleus bilaterally and possibly involving the utmost proximal portion of oculomotor fasciculi of both sides (Fig. 3).

She had ataxia and suffered left side limb weakness because of the separate lesions in the cerebellum and right cerebral peduncle. Six months after the onset of symptoms, bilateral ptosis was much improved. Adduction and elevation were slightly improved.

**DISCUSSION**

The oculomotor nuclear complex extends in the midbrain from the trochlear nucleus at the pontomesencephalic junction up to the posterior commissure rostrally. The sylvian aqueduct lies dorsal to the complex, and the medial longitudinal fasciculus (MLF) runs rostrocaudally just ventral to the oculomotor nucleus complex.\(^5\)

According to Warwick’s studies\(^6\) of retrograde chromatolysis in the cells of the oculomotor nuclear complex of rhesus monkeys, the third nucleus is a
REFERENCES

Several papers have suggested that the small lesion of the parietal cortex with the survival of the superior parietal lobule may be involved with the spatially selective involvement of callosal function since it was the subject of clinical findings supporting the idea of bilateral callosal involvement. In these cases, the lesion to the callosal complex and its relationship with the parietal regions may be implicated as the cause of the disorder. In recent cases, bilateral callosal lesions with the preservation of the callosal function in the hemisphere have shown a small callosal lesion in the region of the callosal nuclei, as reported by Finnish et al. (1997).

other individual extraocular muscles are spared.

Deep inspection of the left superior rectus muscle shows a midline symmetrical nodule within the muscle, which possessed both crossed and uncrossed axial projections. The superior rectus muscle extends anteriorly to the interciliary muscle, the inferior rectus muscle, and the medial rectus muscle.

**FIG. 2.** Bell's palsy (top) and Bell's eye closed (middle and bottom) are all negative.

**FIG. 3.** Axial T2-weighted MRI (TR = 4000 ms, TE = 120 milliseconds) of the midbrain 1 month ago. Axial T2-weighted MRI (TR = 120 milliseconds, TE = 120 milliseconds) of the midbrain 1 month ago.