Isolated Superior Rectus Palsy Due to Contralateral Midbrain Infarction

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Background: Isolated superior rectus palsy due to a contralateral midbrain lesion has not been reported.

Case Description: A 71-year-old woman suddenly developed diplopia. Examination showed that she had isolated superior rectus paresis. Magnetic resonance imaging showed a tiny infarct at the area of the oculomotor nucleus on the contralateral side.

Conclusion: Isolated superior rectus palsy may be caused by a contralateral midbrain lesion that selectively involves crossing superior rectus nerve fibers.

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Midbrain infarcts may produce ocular motor paresis without other neurological signs. Weakness of a single extraocular muscle has also been reported to be caused by a small midbrain infarction. However, to our knowledge, isolated contralateral superior rectus palsy had not been reported to be caused by midbrain lesions.

Report of a Case

A 71-year-old woman with diabetes mellitus and hypertension suddenly developed vertigo and vertical diplopia. On hospital admission 1 day later, neurologic examination results revealed no extremity weakness, sensory changes, dysmetria, or gait disturbances. There was no anisocoria or anhidrosis. Head tilting was not observed.

On ocular examination, the pupil size was equal and nystagmus was absent in both eyes. On forward gaze, there was slight hypotropia in the right eye. Supraduction of the right eye was significantly limited, while abduction, adduction, and infraduction movements were within normal limits. The limitation of supraduction was also observed during tests of the Bell phenomenon and oculocephalic reflexes. Eyeball movements of the left eye were considered normal. On the red glass test, maximally separated images were present on the right, upward gaze when the red image was present superior to the white one.

Although the palpebral fissure in the right eye appeared slightly narrow as compared with the left one, the patient and her relatives stated that this had been present long before hospital admission. A fundus examination showed extorsion of the right eye without a torsional component in the left eye (Figure 1B). Diplopia test findings with the Hess chart were consistent with the superior rectus palsy in the right eye. Magnetic resonance imaging showed a discrete infarction in the midbrain anterolateral to the cerebral aqueduct at the superior colliculi level (Figure 2). Magnetic resonance angiography findings were normal. Over the next few weeks, the patient’s vertical diplopia gradually resolved.

Comment

Our patient had vertical diplopia and limited supraduction of the right eye due to a tiny infarction in the contralateral midbrain. There were no other neurologic signs except for a slightly narrowed right palpebral fissure, which probably had been present before the onset of stroke.

These findings should be differentiated from ocular tilt reaction, which may result in vertical diplopia in patients with...
brainstem stroke. The tilting in ocular tilt reaction is
counteractive in patients with pontomesencephalic brain-
stem lesions involving the rostral medial longitudinal fasci-
culus or the interstitial nucleus of Cajal, while it is
ipsiversive in patients with medullary lesions.2 Ocular tilt
reaction was ruled out in the patient because of the fol-
lowing observations. First, there was no head tilting in
the patient. Second, ocular torsion due to midbrain le-
sions should be conjugate, but only the torsion of the right
eye was observed in the patient (Figure 1B). Finally, up-
ward eyeball movements were absent during tests of the
Bell phenomenon and oculocephalic reflexes.2 Al-
though extratarsion of the right eye may also be caused
by a fourth nerve palsy, red glass examination and Hess
test findings were not consistent with the superior ob-
lique paresis.

Therefore, we considered that the patient had iso-
lated superior rectus palsy caused by a contralateral mid-
brain lesion, which was probably due to an involvement
of the oculomotor nucleus complex. In the midbrain, the
oculomotor nuclei extend rostrocaudally at the su-
perior colliculi level, ventral to the cerebral aqueduct. The
rostrocaudal length of the complex has been shown to
be 6.1 mm in humans.3 The subnucleus subserving the
superior rectus is located in the caudal two thirds of the
oculomotor nucleus on the contralateral side. Because the
decussation of fibers to the superior rectus takes place
within the oculomotor nuclear complex, the lesions af-
flecting the nucleus may simultaneously involve the ip-
silateral superior rectus subnucleus, as well as the cross-
ing fibers, resulting in bilateral superior rectus palsy.4,5
However, isolated contralateral superior rectus palsy has
been reported with concomitant paralysis of some of the
ipsilateral extraocular muscles, such as the inferior rec-
tus or the medial rectus.6,8

It has been shown that the crossing nerve fibers
subserving the contralateral superior rectus arise mainly
in the dorsoventral area of the caudal one third to one

Figure 1. A, Extraocular movements in 9 cardinal positions. Right eye showed supraduction deficit and hypotropia. B, Dysconjugate ocular torsion in fundus
photography. There was excyclotorsion in the right eye without incyclotorsion of the left eye.

Figure 2. T2-weighted magnetic resonance imaging shows a discrete infarct
in the left midbrain (arrow).
half of the nucleus. On the other hand, no crossing fibers are seen in the rostral half of the nucleus. Therefore, we speculate that a lesion localized at the caudal one third to one half of the oculomotor nucleus may have selectively involved the crossing fibers toward the contralateral superior rectus, causing isolated superior rectus palsy on the side contralateral to the lesion.

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