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Isolated sixth nerve palsy from pontine infarct

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Abstract

Isolated sixth nerve palsy associated with pontine infarct is very rare due to close anatomic organization of the structures. A 62-year-old woman, who complained of diplopia, had a diagnosis of sixth nerve palsy. Ophthalmological examination revealed 30 PD left esotropia in primary position with limited abduction of the left eye. Neurologic examination was normal. MR showed a lacunar infarct in the pons consistent with a fascicular lesion. Cerebral angiography was normal. Pontine infarcts causing fascicular lesions should be kept in mind in isolated sixth nerve palsies.

Key words : Sixth nerve palsy ; fascicular sixth nerve palsy ; pontine infarct.

Sixth cranial nerve palsies are the most frequently reported ocular motor palsies. The most common cause of isolated sixth nerve palsy in patients over 40 years of age is infarction of the nerve trunk as it is in isolated pupil-sparing third nerve palsy and nontraumatic fourth nerve palsy (Burde *et al.*, 1992). The site of nerve injury is thought to be within the subarachnoid space, superior orbital fissure or cavernous sinus (Johnson & Hepler, 1989, Burde *et al.*, 1992). Sixth nerve palsies due to a lesion of the pontine fascicle are usually associated with other neurologic findings. Isolated sixth nerve palsy from a pontine lesion is very rare due to close spatial relationship with other pontine structures. We report a patient with sixth nerve palsy due to a lacunar infarct in the pons without any associated neurological deficit. Awareness of this rare, isolated form of sixth nerve palsy may facilitate the differential diagnosis and treatment.

Case report

A 62-year-old woman was seen with a 6 month history of diplopia. Visual acuity was 20/20 and visual fields were normal. Pupils were equal in diameter and briskly reactive to light wih no afferent pupillary defect. Motility examination revealed 30 PD left esotropia in primary position with limited abduction of the left eye. The left esotropia increased to 40 PD in left gaze and decreased in right gaze. Her smooth pursuit and saccades were normal, except limited abduction in the left eye, and the forced duction test revealed minimal contraction of the left medial rectus muscle. Her neurological examination was evaluated as normal and her medical history was unremarkable except for hypertension. MRI was done due to delay in recovery and showed a lacunar infarct in the pons, consistent with isolated left sided fascicular lesion and without any associated cerebral pathology (Fig. 1A, B). Cerebral digital subtraction angiography was normal and any vascular pathology or malformation were ruled out. In the follow-up, she showed improvement in 10 months with a residual esotropia of 10-12 PD left ET however without any complaint of diplopia. The results of her laboratory tests and systemic evaluation were normal.

Discussion

Our case shows that an isolated sixth nerve palsy may result from a small pontine infarct that affects the abducens nerve fascicle without affecting any of the surrounding structures. Lateral rectus weakness from a pontine lesion is usually associated with other neurological signs such as ipsilateral Horner syndrome, ipsilateral conjugate gaze palsy, ipsilateral internuclear ophthalmoplegia and contralateral hemiparesis. In nuclear lesions ipsilateral conjugate gaze palsy (due to involvement of the internuclear neurons between 6th nerve nucleus and contralateral medial longitudinal fasciculus) is seen and this helps for differential diagnosis of nuclear lesions and isolated 6th nerve palsy. Millard-Gubler syndrome (6th nerve palsy, ipsilateral 7th nerve paresis and contralateral hemiparesis), Raymond syndrome (6th nerve palsy, contralateral hemiparesis) and Foville syndrome (horizontal conjugate gaze palsy, ipsilateral 5,7, and 8th nerve palsies and ipsilateral Horner syndrome) are the syndromes associated with lesions in brainstem and pons. However, with developments in imaging techniques, it was shown that selective lesions within the PPRF might affect horizontal gaze in

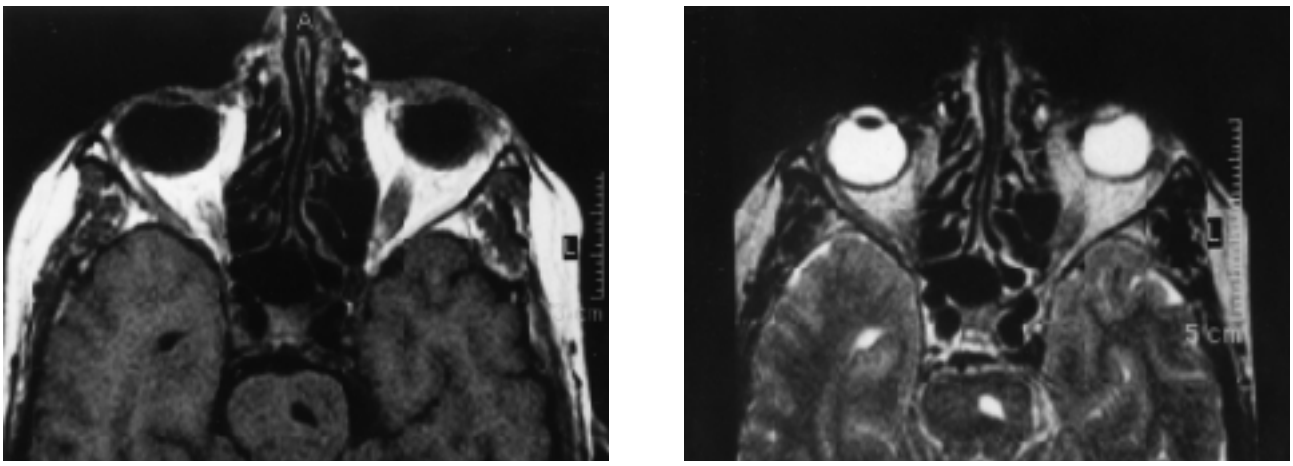

FIG. 1 A-B. — Signal change due to an infarct in the left central part of the pons is seen as a well-defined hypointensity on the T1-weighted (A) and hyperintensity on the T2-weighted (B) MR images.

different ways, including sixth nerve palsies (Lopez *et al.*, 1996). The possible locations of the lesions causing horizontal gaze abnormalities were clearly shown by the study conducted by Bronstein *et al.* (Bronstein *et al.*, 1990). In their study, most abnormal MRI signals from different patients with central 6th nerve palsy were in the area of the posterior half of the abducens fasciculus, and lesions of the 6th nerve nucleus resulted in paresis of abduction of the ipsilateral eye and the adduction of the contralateral eye with electrooculographic recordings, that is an ipsilateral gaze palsy . Even though we were unable to perform a saccadic velocity recording, the adduction of the other eye was normal with clinical evaluation in our patient. As our patient had no clinical or subclinical gaze palsy we can assume that there was no 6th nerve nucleus involvement and there was involvement of the abducens fasciculus. Also, as the patient had no signs of 7th nerve palsy or ataxia, we could suggest that the lesion was not affecting neighbouring structures, such as 7th nerve nucleus or the central tegmental tract.

In the literature, isolated third and forth nerve palsies due to midbrain hemorhage or infarction were reported and isolated sixth nerve palsy due to pontine hemorrhage is very rare and reported only few times previously (Donaldson and Rosenberg, 1988, Johnson and Hepler, 1989, Fukutake and Hirayama, 1992, Galetta and Balcer, 1998, Keane and Ahmadi 1998). Our case differed from previous cases with late improvement as all of the previous cases recovered spontaneously in a few months.

The developments in neuroimaging techniques revealed new etiological factors. In the differential diagnosis of isolated sixth nerve palsies, pontine infarcts should be kept in mind, especially in the presence of ischemic etiologic factors such as hypertension. The follow-up of these cases should be similar to the ischemic cases and radiological examination should be performed when recovery is not seen after 3 months even in the absence of associated neurological findings. Our patient was different from previous cases as she improved over 6 months and surgery can be delayed more than 6 months in such cases.

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