COMBINED INTERNUCLEAR OPHTHALMOPLEGIA AND CONTRALATERAL SUPERIOR OBLIQUE PALSY: A CASE REPORT

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Abstract

Although the fourth cranial (trochlear) nerve nucleus and the medial longitudinal fasciculus are anatomically closely associated, discrete lesions affecting both of these structures would appear to be rare. A case is presented of a man with a left internuclear ophthalmoplegia and a right fourth nerve palsy following a mild CVA.

Key words: *Internuclear ophthalmoplegia, fourth cranial nerve palsy, brain stem lesion.*

The fourth cranial (trochlear) nerve nucleus lies ventral to the Sylvian aqueduct and just dorsal to the medial longitudinal fasciculus at the level of the inferior colliculus.

The fascicular portion of the nerve passes laterally and backwards around the aqueduct and crosses the midline behind the aqueduct to emerge on the surface of the midbrain below the inferior colliculus (see Figure 1). Thus a lesion of the nucleus or its fasciculus would result in a paresis of the contralateral superior oblique.

The medial longitudinal fasciculus (MLF) is located dorsally on either side of the median line in the brain stem and consists of fibres extending from the cervical cord to the rostral midbrain. Excitatory impulses from the paramedian pontine reticular formation (horizontal gaze centre) cross and ascend in the contralateral MLF, so that the lateral rectus of one eye contracts at the same time as the medial rectus of the other eye (see Figure 2). A lesion of the MLF between the midpons and the oculomotor nucleus typically results in the classic signs of an internuclear ophthalmoplegia i.e. defective adduction of the ipsilateral eye, normal convergence and abducting nystagmus of the contralateral eye.

Although the fourth cranial nerve and the MLF are closely associated, discrete lesions affecting these structures together would appear to be rare. The following case, however, appears to illustrate such a lesion.

S.T., a 68 year old man, presented at the Casualty Department of Concord Hospital, with a history of an onset of diplopia looking forward or to the right. It was associated with veering to the right, a temporary loss of consciousness and collapse. He had a dull headache but no other neurological signs. He had a previous history of hypertension and had been on aldomet.

He was admitted to hospital for observation, but no other neurological signs, other than his diplopia, were found. He was discharged two weeks later with his diplopia improved, but still present to the right. When reviewed in the Eye Clinic one month later he had a poorly controlled

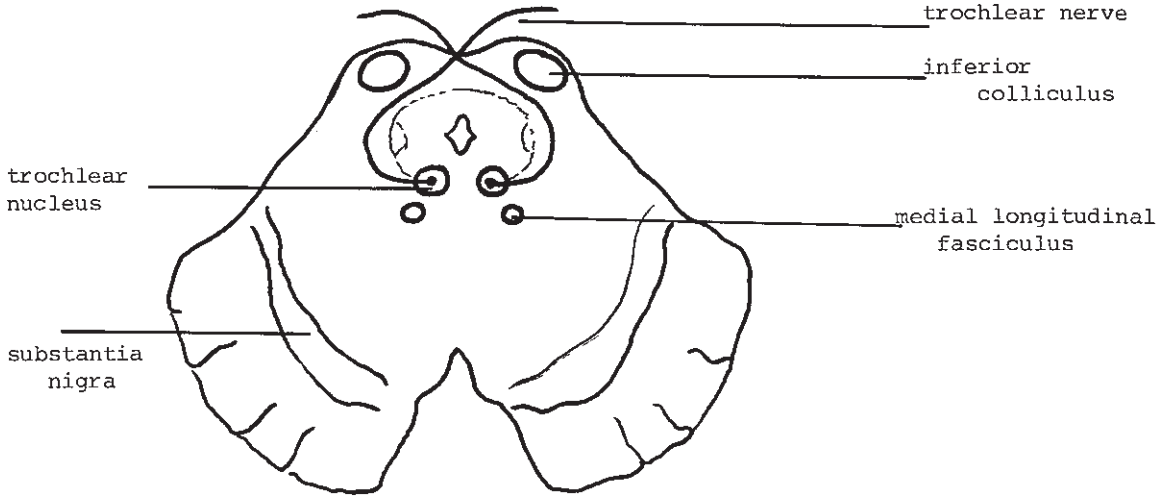

Figure 1: Cross section of the midbrain at the level of the inferior colliculus to show the nucleus and course of the fourth (trochlear) nerve and its relationship to the medial longitudinal fasciculus.

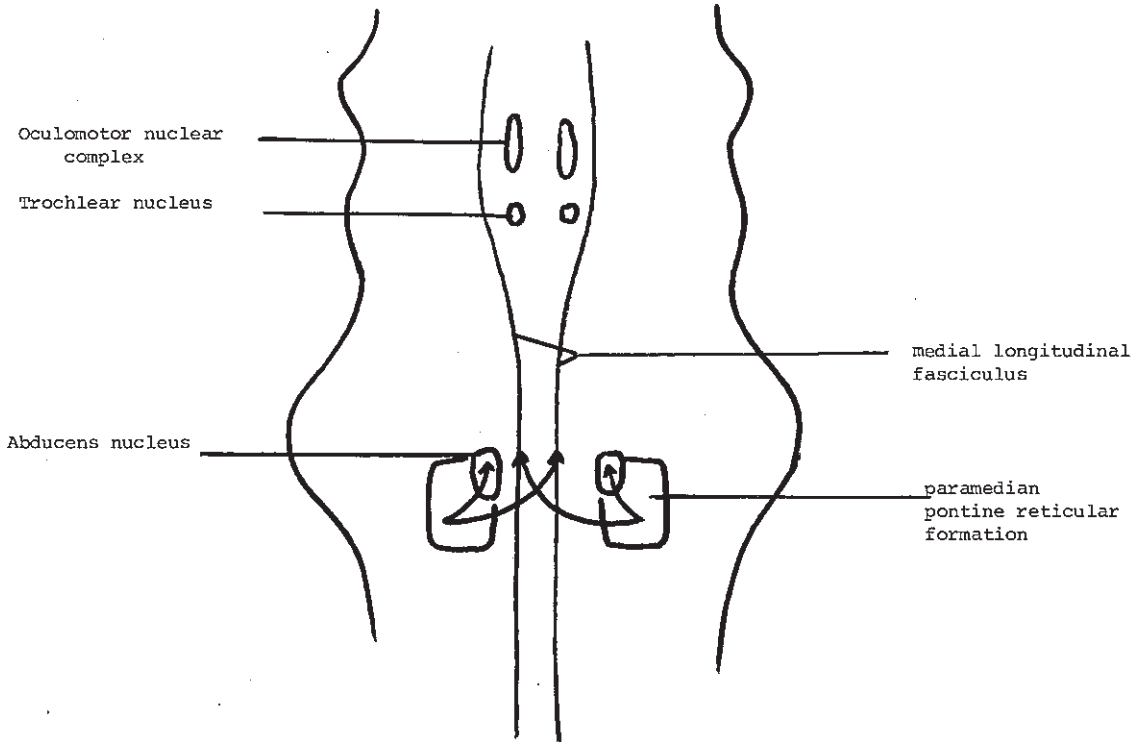

Figure 2: Diagrammatic representation of the ocular motor nuclei in the brain stem and the MLF. Impulses to initiate horizontal gaze arise in the PPRF and pass to the ipsilateral abducens nucleus, and also cross to the contralateral MLF to travel to the oculomotor nucleus.

exophoria in the primary position at distance and near which readily decompensated to a right exotropia with diplopia. He reported horizontal diplopia looking to the right, and vertical diplopia looking to the left. Examination of his extraocular muscle balance revealed slightly defective adduction of the left eye, abducting nystagmus of the right eye, normal convergence and a mild underaction of his right superior oblique. This, and the following measurements are consistent with a diagnosis of a lesion involving the left MLF and the left fourth nerve nucleus.

Neurological investigation included a CAT scan which revealed mild cerebral atrophy with no evidence of intracerebral or intracranial abnormality. However, brain stem auditory evoked potentials indicated a bilateral brain stem conduction disturbance. The patient's diplopia resolved sufficiently to be of little trouble to him, although clinical evidence of it was still present when he was last seen four months after its onset.

Prism cover test		Dextro version	Primary position	Laevo version
33 cm	FR	$-6^{\Delta}\ \frac{R}{L}\ 3^{\Delta}$	$-3^{\Delta}\ \frac{R}{L}\ 4^{\Delta}$	$\phi\ \frac{R}{L}\ 7^{\Delta}$
	FL	$-10^{\Delta}\ \frac{R}{L}\ 3^{\Delta}$	$-4^{\Delta}\ \frac{R}{L}\ 3^{\Delta}$	$\phi\ \frac{R}{L}\ 4^{\Delta}$
6 m	FR	$\phi\ \frac{R}{L}\ 2^{\Delta}$	$\phi\ \frac{R}{L}\ 3^{\Delta}$	$\phi\ \frac{R}{L}\ 3^{\Delta}$
	FL	$\phi\ \frac{R}{L}\ 1^{\Delta}$	$\phi\ \frac{R}{L}\ 2^{\Delta}$	$\phi\ \frac{R}{L}\ 2^{\Delta}$

Head Tilting Test		Tilt R	Tilt L
(6 m)	FR	$\frac{R}{L}\ 8^{\Delta}$	$\frac{R}{L}\ 1^{\Delta}$
	FL	$\frac{R}{L}\ 5^{\Delta}$	0

Synoptophore

$0\ \frac{R}{L}\ 1^{\Delta}$		$0\ \frac{R}{L}\ 1^{\Delta}$	$\oplus$		$+2^{\circ}\ \theta$
to right←					→to left
$0\ \frac{R}{L}\ 2^{\Delta}$		$+2^{\circ}\ \frac{R}{L}\ 5^{\Delta}$	$+3\theta$		$+6^{\circ}\ \frac{R}{L}\ 4^{\Delta}$

LE Fixing RE Fixing

This case is presented to alert examiners to the possibility of a contralateral fourth nerve paresis in the presence of an internuclear ophthalmoplegia. In fact, the presence of a superior oblique palsy was not initially diagnosed in this case until a full examination of ocular motility was carried out. Mild underactions of the superior oblique are not always revealed unless they are specifically looked for, especially if there is a more obvious clinical sign as there was in this case.