Bilateral Internuclear Ophthalmoplegia Following Head Injury

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Abstract
A 40-year-old female after a closed head injury presented with bilateral internuclear ophthalmoplegia. Attempted convergence was abnormal and MRI revealed the focal hemorrhage in the medial longitudinal fasciculus region by showing bright signal in the pontomesencephalic region on both T1 and T2 weighted images. The patient regained normal ocular mobility after six months of the injury. The medial longitudinal fasciculus, which is believed to be lesioned in cases of internuclear ophthalmoplegia, is an unusual and rare finding, particularly in patients victims of head injury without further neurological signs. Isolated internuclear ophthalmoplegia should be considered in the differential diagnosis when one encounters an adduction deficit in a patient suffering head injury. ©

INTRODUCTION
Internuclear ophthalmoplegia [INO] is caused by lesion in the medial longitudinal fasciculus [MLF] and clinically this syndrome is characterized by adduction weakness on the side of MLF lesion and monocular nystagmus of the abducting eye. However, unless the lesion is quite high, reaching the midbrain convergence is preserved. Occasionally, there is also skew deviation and vertical nystagmus.1,2 INO may be unilateral or bilateral, multiple sclerosis is the most common cause of bilateral INO while vascular disease is the most common cause of unilateral INO. INO is a rare ocular syndrome after head injury and we report in this communication a patient after closed head injury who presented with isolated bilateral INO and posttraumatic hemorrhagic lesion in the pontomesencephalic region demonstrated by MRI.

CASE REPORT
A 40 year old female presented in the emergency after road traffic accident that produced temporary loss of consciousness. There was no evidence of ocular or periorbital trauma. After regaining clear consciousness, the patient complained of oscillopsia. There was no previous history of ophthalmic, medical, or neurologic disorders. On examination the bilateral visual acuity was 20/20. The pupils were symmetrical and reacted normally to light. Ophthalmoscopy with dilated pupils showed normal findings. Color vision was normal. On attempted right lateral gaze, the left eye failed to adduct across the midline and there was nystagmus of the abducting right eye. When the patient looked to the left, the right eye did not cross the midline, and the left eye had an abducting nystagmus. Attempted convergence was abnormal. Vertical movements showed gaze-evoked nystagmus. Apart from the ocular signs, the patient’s neurologic status was normal. There were no other cranial nerve abnormalities. The muscle power was normal in all extremities. Sensation and the deep tendon reflexes were normal. MRI showed a small bright lesion in the pontomesencephalic junction in the midline on both T1 and T2 weighted images (Figs. 1,2). The patient regained normal ocular mobility after six months of the head injury.

DISCUSSION
Ocular motor disturbance occurs in 3% to 7% of patients with head injuries.3 INO is characterized by acquired paresis of adduction on lateral gaze, in the absence of third nerve palsy. Other associated findings include vertical gaze-evoked nystagmus, impaired vestibular and pursuit vertical movement may occur, impaired convergence, skew deviation, paralysis of horizontal gaze to one side, and involvement of other cranial nerves. Bilateral INO as an isolated finding is considered to be pathognomonic of multiple sclerosis in young patients. Recently, INO has been described to be associated with central nervous system infection and drug-induced INO has also been reported. Traumatic bilateral INO as an isolated sequel is very rare and in most reported cases, INO was a prominent part of the clinical picture, but most patients had other signs of brain stem impairment. The pathophysiology of trauma-induced INO is not known. Three major...
mechanisms leading to MLF syndrome caused by head injury are reported in the literature. They are 1. Primary brain stem injury, 2. Secondary brainstem injury by transtentorial herniation and 3. Circulatory disturbance of perforating branches of the vertebro-basilar artery due to shearing force. Following a blow to the head, a pressure gradient is produced across the skull that results in the creation of shear force, the effect of which is maximum adjacent to the ventricles and at the junction of the white and gray matter. The MLF would be expected to be commonly affected because of its anatomic location posterior in the brain stem. This appears to be the result of relative fixation of the anterior portion of the brain stem by the small penetrating branches of the basilar artery, while the posterior segment moves relatively freely. This differential in movement results in the development of shear force within the brain stem. Damage to the tract could occur by stretching of its nerve fibres, interruption of its blood supply, or focal hemorrhage. However, INO can also be induced by extra-axial brainstem compression. The MRI findings in the present case suggested a true limited focal hemorrhage involving the bilateral MLF.

Isolated internuclear ophthalmoplegia has been scanty reported without cranial nerves or long tracts signs in patient with head trauma. The present case is very unusual because INO was the only abnormality after regaining consciousness in case of head injury, because the unique position of the medial longitudinal fasciculus within the brain stem. Isolated INO should be considered in the differential diagnosis when one encounters an adduction deficit in a patient suffering head injury.

Fig. 1: MRI (axial section, T₁-weighted sequence) showing signal hyperintensity in the pontomesencephalic junction in the midline.

Fig. 2: MRI (axial section, T₂-weighted sequence) showing signal hyperintensity in the pontomesencephalic junction in the midline.

REFERENCES