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POST TRAUMATIC DIPLOPIA : A CASE REPORT

Madhushri V D*	Department of Emergency Medicine, SS Institute of Medical Sciences and Research Centre. *Corresponding Author
Narendra S S	Professor and Head, Department of Emergency Medicine, SS Institute of Medical Sciences and Research Centre.
Shahnawaz Makandar	Junior Resident, Department of Emergency Medicine, SS Institute of Medical Sciences and Research Centre.

ABSTRACT Internuclear ophthalmoparesis (INO) or internuclear ophthalmoplegia, is a distinct gaze abnormality characterized by impaired horizontal eye movements with weak adduction of the affected eye, and abduction nystagmus of the contralateral eye presenting as diplopia. A 34-year-old female patient was brought to the Emergency department following a history of road traffic accident where she fell backwards from the bike. She had a history of loss of consciousness for a duration of 1 hour. On arrival to the emergency department she complained of headache and diplopia. Initial non-contrast Computed tomography of the brain was normal. However, a diffusion weighted MRI of the Brain revealed focal acute infarcts involving the medial longitudinal fasciculus. The patient was diagnosed with Acute ischemic cerebrovascular accident, likely embolic etiology and was started on antiplatelet medication.

KEYWORDS : Diplopia, head trauma, opthalmoperesis.

INTRODUCTION

Internuclear ophthalmoplegia (INO), is caused by lesions in the medial longitudinal fasciculus (MLF), and is characterized by paresis of adduction of the lateral gaze with horizontal jerk nystagmus in the contralateral abducting eye. The medial longitudinal fasciculus, a fiber tract that rises from the abducens nucleus in the pons to the contralateral oculomotor nucleus in the midbrain. Lesions in the medial longitudinal fasciculus result in the failure of adduction on attempted lateral gaze. Any brain-stem syndrome can interrupt the medial longitudinal fasciculus and result in impaired horizontal eye movement, but the most frequent underlying cause is multiple sclerosis. This patient had internuclear ophthalmoplegia in both eyes following a history of head injury.

Case report

A 34 year old female patient presented to the Emergency department with a history of road traffic accident. She was a pillion rider and fell backwards. She had loss of consciousness for a period of 1 hour after the fall and also had seizure like activity characterised by stiffness of all four limbs. On arrival to the emergency room she was drowsy and a GCS of E3V4M6 was documented. She was disoriented towards time, place and person for 6 hours post the accident. On physical examination she was afebrile, with respiratory rate of 16 breaths/min, blood pressure of 130/80 mm hg, Heart rate of 90 bpm, and oxygen saturation of 98% on room air. Initial ocular examination (Fig. 1) revealed bilateral pupils were 3 mm and equally reactive to light. An ocular exam showed disconjugate gaze at rest with right eye and left eye laterally deviated with right eye deviated more laterally than the left eye. Horizontal eye movements were impaired with weakness of medial rectus muscles of both eyes. The remaining extraocular muscles (superior rectus, inferior rectus, lateral rectus, superior oblique, and inferior oblique) and other cranial nerves were normal. She had vertical nystagmus on superior, inferior gaze. Motor examination, Sensation, proprioception, joint and vibratory sense were normal.

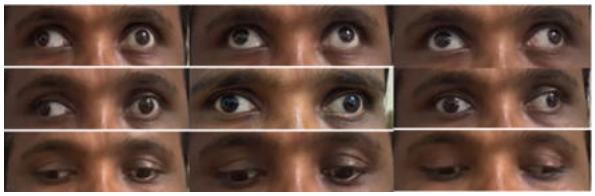

Fig 1: Ocular examination of the patient

Investigations

The initial Non- Contrast Computed tomography of the brain done from the emergency department did not reveal any significant abnormality. However, 18 hours after the injury the patient persisted to have diplopia and a diffusion weighted MRI of the brain was done which revealed a hyperintense lesion at the junction of midbrain and pons (Fig 2) involving the medial longitudinal fasciculus- likely embolic aetiology.

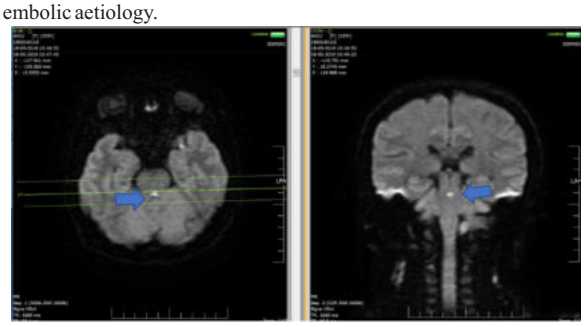

Fig 2: Axial and Coronal section of diffusion weighted MRI reveals diffusion restriction (blue arrows) in the median longitudinal fasciculus.

DISCUSSION

Diplopia is the common presentation in patients with internuclear ophthalmoplegia (INO). Bilateral internuclear ophthalmoplegia is most commonly seen in patients with multiple sclerosis[1]. The other causes of INO includes stroke and head injury[2]. Most of the times head injury is associated with unilateral INO due to haemorrhage, but this patient developed a bilateral INO secondary to an ischemic etiology.

The pathogenesis of the isolated damage to the MLF associated with head trauma is unclear, but several mechanisms have been suggested. Common hypotheses on the anatomical origin of post-traumatic MLF injury include differential displacement of the brainstem with resultant stretching of the MLF fibers[3], shearing forces in the brainstem caused by a blow to the head[4], and shearing forces caused by the angular acceleration or deceleration of the head upon impact[5]. The shearing forces generated by angular movement exert the maximal effect where the difference between the densities of the cerebrospinal fluid and the adjacent neural tissue is the greatest. Because the MLF is located near the aqueduct and the floor of the fourth ventricle, it is vulnerable to these shearing forces. The posterior portion of the brainstem can be displaced downwardly than the anterior portion because the latter is tethered by small penetrating arteries of the basilar artery.12) Therefore, the shearing forces can create a temporary downward displacement of the posterior brainstem and cause shear injury to the MLF.

In this case report, we found that although the patient presented with a history of head trauma, the patient had Internuclear ophthalmoplegia due to an embolic etiology. Hence it is important to identify the etiology for specific management.

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