Head Injury: A Rare Cause of Bilateral Internuclear Ophthalmoplegia

Srikant Kumar Swain[1] Sureswar Mohanty[1] Adya Kinkar Panda[2]

[1]Department of Neurosurgery, Institute of Medical Sciences and SUM Hospital, Siksha 'O' Anusandhan (Deemed to be) University, Bhubaneswar, Odisha, India
[2]Department of Radiology, Institute of Medical Sciences and SUM Hospital, Siksha 'O' Anusandhan (Deemed to be) University, Bhubaneswar, Odisha, India

Address for correspondence Sureswar Mohanty, MS, MCH, Department of Neurosurgery, Institute of Medical Sciences and SUM Hospital, Siksha 'O' Anusandhan (Deemed to be) University, K-8, Kalinga Nagar, Bhubaneswar 751003, Odisha, India (e-mail: sureswar.mohanty@gmail.com).

Indian J Neurotrauma 2018;15:70–73

Abstract

Rare case of bilateral internuclear ophthalmoplegia (INO) following head injury in 25-year-old male is being reported. The site of the lesion in the mid-brain as detected in the magnetic resonance imaging (MRI) correlates well with the clinical presentation. The patient recovered on conservative treatment with a course of dexamethasone (for brainstem contusion).

Keywords
- head injury
- internuclear ophthalmoplegia
- brainstem hemorrhage

Introduction

Internuclear ophthalmoplegia (INO) is caused by lesion in the medial longitudinal fasciculus (MLF) in the brainstem and is classically characterized by limitation of adduction of ipsilateral eye on lateral gaze, associated with nystagmus in the contralateral abducting eye.[1] Bilateral INO among young patients occurs most commonly due to demyelination of the MLF, often secondary to multiple sclerosis (MS).[2] Unilateral INO found in elderly patients is most commonly caused by cerebrovascular disease.[2] INO is rarely associated with minor head trauma. We present a case of traumatic bilateral INO as a sequela of closed head injury.

Case Report

A 25-year-old male presented to the emergency department (ED) after a road traffic accident. The patient had a history of brief loss of consciousness with retrograde amnesia, headache, disorientation, irritability, and unsteady gait. He was not cooperative for complete neurologic examination. There was no evidence of ocular or periorbital trauma. After regaining full consciousness in a couple of days, he complained of dysphonia, dysphagia, visual disturbance, and truncal ataxia. Initially, computed tomography (CT) of the brain performed in the ED revealed acute anterior falcine subdural hemorrhage (SDH) with tiny hemorrhagic contusion in the bilateral parasagittal frontal cortex, left posterior temporal lobe, midbrain, and pontine region—without any mass effect requiring surgical intervention. He was admitted to the Department of Neurosurgery for conservative management with mannitol, dexamethasone (for brainstem contusion), and prophylactic antiepileptic drug. The clinical condition improved after 2 days, and thorough neurologic examination revealed an improvement in dysphonia and ataxic movement, but ocular motility examination revealed restricted horizontal movement during lateral gaze in both eyes. The patient had an adduction deficit in the right eye and nystagmus in the left eye on leftward gaze. He also had an adduction deficit in the left eye and nystagmus in the right eye on rightward gaze (►**Fig. 1**). The patient was referred for complete ophthalmologic evaluation. The intraocular pressure was 17 mm Hg in the right eye and 15 mm Hg in the left eye. The pupillary response, slit lamp, and funduscopy examination were normal. Upward and downward gazes were unremarkable. Magnetic resonance imaging (MRI) of the brain was performed 2 days after injury and was suggestive of tiny hemorrhagic contusion in the bilateral basi frontal paramedian cortex and mid-brain (T2-weighted/fluid-attenuated inversion recovery [T2W/FLAIR]—hyperintense collection in midline of mid-brain and upper pons). Subsequent evaluation with

received
January 21, 2019
accepted
January 23, 2019
published online
August 13, 2019

DOI https://doi.org/10.1055/s-0039-1694696
ISSN 0973-0508.

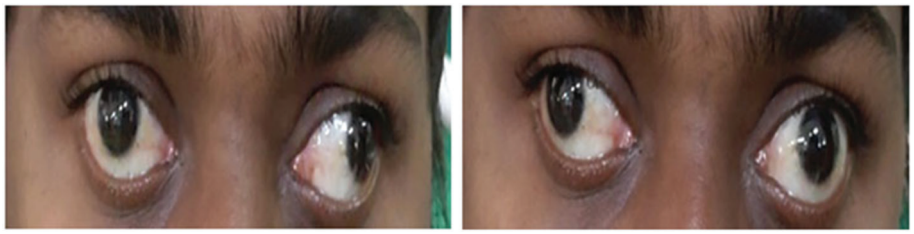

Fig. 1 On attempted right lateral gaze, the left eye failed to adduct across the midline, and on left lateral gaze, the right eye did not cross the midline.

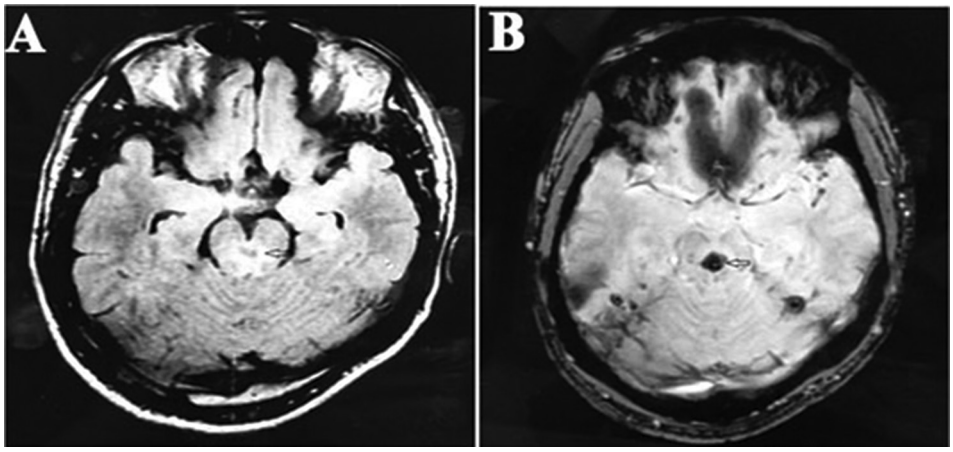

Fig. 2 (**A**) T2WI/FLAIR showing hyperintense collection in midline of the mid-brain. (**B**) SWI (susceptibility-weighted image) showing blooming.

susceptibility-weighted imaging (SWI) definitely revealed a tiny hemorrhage (blooming in that particular area) (►**Fig. 2**).

Discussion

The most common reason for INO in a young patient without head trauma is multiple sclerosis (MS). Bilateral INO was previously described as "pathognomonic" of MS. INO in elderly patients is usually unilateral and secondary to cerebrovascular disease.[2] In a series of 410 patients by Keane, the causes of INO included infarction 38%, MS 34%, tentorial herniation 5%, trauma 5%, infection 4%, tumor 4%, iatrogenic injury 3%, and hemorrhage 3%.[3]

Loss of consciousness following trauma to the head occurs due to stretch effect/ischemia on ascending reticular formation in the upper brainstem. This happens due to relative movement of the cerebrum at its junction with brainstem. However, if the stretch is severe, intraparenchymal capillaries may be disrupted leading to hemorrhage and persistent brainstem dysfunction.

In our patient, such a lesion demonstrated the classic findings of a bilateral internuclear ophthalmoplegia. He had bilateral adduction impairment on lateral gaze. In addition, there was a prominent monocular horizontal nystagmus of the abducting eye. Normal conjugate gaze is based on several distinct neuro-anatomical pathways (►**Fig. 3**).[4] The sixth (VI) nerve nucleus on one side of the brainstem receives supranuclear impulses to direct lateral saccades via the ipsilateral paramedian pontine reticular formation. It also receives pursuit and vestibular input for horizontal eye movements. The VI nerve nucleus contains both motor neurons for the ipsilateral lateral rectus and interneurons that cross the midline of the brainstem and form the contralateral MLF. These excitatory interneurons travel through the MLF and synapse with neurons in the contralateral medial rectus subnucleus of the third nerve nuclear complex, producing contraction of the medial rectus. Unilateral lesions of the MLF allow for normal abduction while impairing ipsilateral adduction. Our patient had a small lesion affecting both MLFs, thus causing bilateral INO. A second pathognomonic sign of an INO is horizontal nystagmus of the abducting eye. Such focal lesion appears benign and recovers in course of time. Use of steroid possibly diminishes secondary phenomenon, that is, edema, and helps in early resolution.

The mechanism by which localized injury to the MLF occurs is somewhat controversial. Until the mid-1970s, shearing forces directly injuring the MLF fibers was the prevailing injury theory.[5,6] More recently, the proposed mechanism involves shear forces disrupting the vascular supply, leading to a localized infarction/hemorrhage of the MLF. During trauma, shear forces stretch these vessels and may cause ischemic or hemorrhagic infarction of the MLF.[7-11]

Conclusion

Our case demonstrated that moderate head injury with focal brainstem hemorrhage can manifest as bilateral INO. Although isolated INO is a rare finding following trauma, it should be considered in the differential diagnosis when one encounters an adduction deficit on horizontal gaze in a

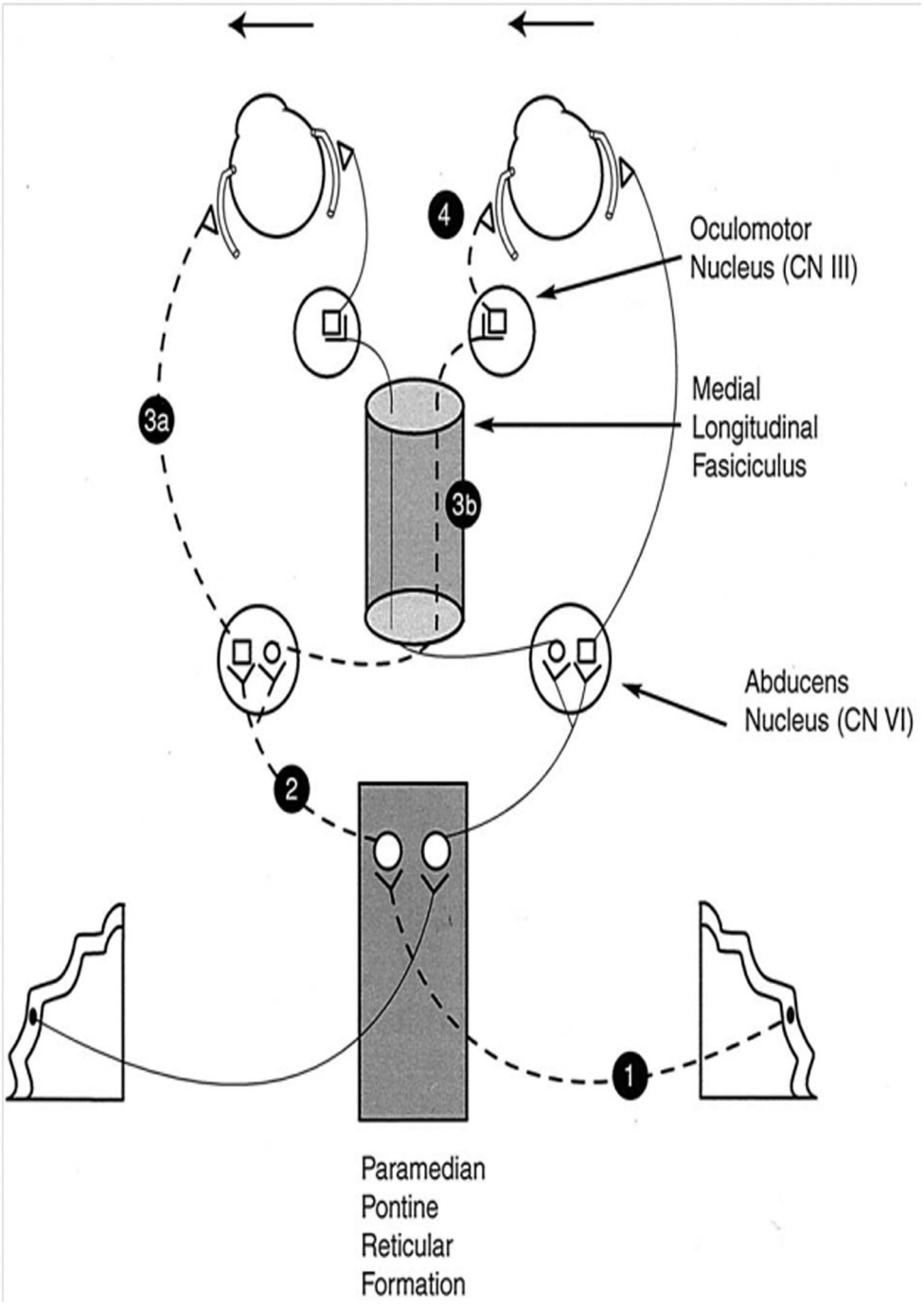

Fig. 3 Schematic diagram depicting anatomical basis of left lateral gaze. Cortical input reaches the sixth nerve nucleus via the paramedian pontine reticular formation. The sixth nerve nucleus contains motor neurons that innervate the ipsilateral lateral rectus (3a) and interneurons that cross the midline and form the contralateral MLF (3b). Interneurons from the MLF synapse with motor neurons in the contralateral oculomotor nucleus, which innervate the contralateral medial rectus. INO occurs in a lesion at (3b).

recently traumatized patient. Prognosis is relatively satisfactory on conservative treatment.

Conflicts of Interest
None.

Acknowledgments
We are thankful to Prof. Dr. M. R. Nayak, Honorable President, Siksha 'O' Anusandhan (Deemed to be) University. We also acknowledge Mr. Somadatta Das, Central Research Laboratory, IMS & SUM Hospital, S 'O' A University, for his technical support in submitting the manuscript.

References

1 Cogan DG, Kubik CS, Smith WL. Unilateral internuclear ophthalmoplegia; report of 8 clinical cases with one postmortem study. AMA Arch Opthalmol 1950;44(6):783–796
2 Smith JW, Cogan DG. Internuclear ophthalmoplegia; a review of fifty-eight cases. AMA Arch Opthalmol 1959;61(5):687–694

3 Keane JR. Internuclear ophthalmoplegia: unusual causes in 114 of 410 patients. Arch Neurol 2005;62(5):714–717
4 Kandel ER, Schwartz JH, Jessell TM, et al. Principles of Neural Science. 3rd ed. New York, NY: Elsevier1991;722
5 Strich SJ. Shearing of nerve fibers as a cause of brain damage due to head injury: a pathological study of twenty cases. Lancet 1961;2:443–448
6 Rich JR, Gregorius FK, Hepler RS. Bilateral internuclear ophthalmoplegia after trauma. Arch Ophthalmol 1974;92(1):66–68
7 Zauel D, Carlow TJ. Internuclear ophthalmoplegia following cervical manipulation. Ann Neurol 1977;1(3):308
8 Baker RS. Internuclear ophthalmoplegia following head injury. Case report. J Neurosurg 1979;51(4):552–555
9 Rosati G, Pinna L, Paolino E, D'Agostini G. Reversible bilateral internuclear ophthalmoplegia due to head trauma: a case report. Eur Neurol 1981;20(2):81–83
10 Devereaux MW, Brust JCM, Keane JR. Internuclear ophthalmoplegia caused by subdural hematoma. Neurology 1979;29(2):251–255
11 Constantoyannis C, Tzortzidis F, Papadakis N. Internuclear ophthalmoplegia following minor head injury: a case report. Br. J Neurosurg 1998;12(4):377–379