



A RARE CASE OF BILATERAL INTERNUCLEAR OPHTHALMOPLÉGIA AS A PRESENTATION OF STROKE

Ophthalmology

**Dr.Sai Sneha
Yadlapalli**

Dr. U Vivekanand

**Dr. Sai Sreya
Yadlapalli**

**Dr.Sarat
Mallipeddi**

ABSTRACT

We report a 36-year-old woman who presented with bilateral Internuclear ophthalmoplegia with diplopia, left-sided cerebellar ataxia, and horizontal coarse nystagmus caused by a stroke in the posterior cerebral artery territory.

KEYWORDS

Internuclear ophthalmoplegia; medial longitudinal fasciculus,vertebrobasilar artery stroke

INTRODUCTION:

Internuclear ophthalmoplegia (INO) or the syndrome of the medial longitudinal is caused by lesions in the medial longitudinal fasciculus (MLF). It is classically outlined by paresis of adduction on the lateral gaze associated with nystagmus on the abduction of the contralateral eye. It is presented as dissociated, or ataxic nystagmus and it is caused by the interruption of the impulses between the centre for lateral gaze in the pons and the oculomotor nuclei¹. The unilateral form is more common than bilateral. Multiple sclerosis in young adults can present with Bilateral (INO) It can also be caused due to head injury with or without clivus fracture, vascular lesions of brainstem², neuropsychiatric lupus erythematosus, and Herpes zoster vasculitis. The motive behind this particular rare case report is to document bilateral internuclear ophthalmoplegia which is caused by a stroke in the posterior cerebral artery territory which is resolved gradually with treatment.

Case History:

A 36-year-old hypertensive female was referred to the ophthalmology outpatient department with chief complaints of sudden onset of double vision of 3 days duration. She presented with an unsteady gait, giddiness, abnormal and impaired sensations, headache which was associated with vomiting of 15 days duration. She had similar complaints and was diagnosed as ischemic stroke secondary to hypertension in the past. She has not been using the treatment for the past two months, after which she presented with these symptoms. She had no history of fever, seizures, loss of consciousness, slurring speech, tinnitus, or urinary incontinence. She had no history of tuberculosis in the past. No history of contact in the family.

General physical examination was routine. Blood pressure was 150/90 mm Hg at the time of the presentation. Neurological examination revealed the bilateral weakness of the adducting eye on lateral gaze, as shown in fig 1 and abducting eye showing coarse, horizontal nystagmus. There was preserved adduction during convergence. All other cranial nerves were healthy. Ataxic gait swaying to the left is present. Finger nose test, finger nose test, knee heel test was impaired on the left side. Past pointing was present to the left.



Fig 1: On attempted right lateral gaze, the left eye failed to adduct across the midline. When the patient looked to the left, the right eye did not cross the midline.

All systemic investigations were unremarkable. Carotid Doppler was normal. MRI brain showed acute infarcts in left cerebellar hemisphere, pons and midbrain. Chronic infarcts in the bilateral caudate nucleus and right lentiform nucleus, as shown in fig2.



Fig 2: MRI brain showed diffusion restriction in left midbrain, pons and left cerebellar hemisphere suggestive of acute infarcts.

Following as neurology opinion, she was started on injection heparin 5000 IU SC two times a day, antihypertensive medication- Tab. Metoprolol 25mg + Tab. Telmisartan 40 mg, Tab. Aspirin 150 mg once a day and Tab. Atorvastatin 40 mg once a day.

DISCUSSION:

In conclusion, the most common cause of Bilateral internuclear ophthalmoplegia is Multiple sclerosis. In contrast, stroke in the posterior cerebral artery territory is a rare causing factor of bilateral internuclear in young adults.

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