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BILATERAL INTERNUCLEAR OPHTHALMOPLEGIA AND HORIZONTAL GAZE PALSY SECONDARY TO PONTINE INFARCT IN A YOUNG ADULT WITH HYPERHOMOCYSTEINAEMIA: A RARE CASE REPORT

Ophthalomology

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ABSTRACT

Internuclear ophthalmoplegia (INO) is a rare eye movement disorder caused by lesions of the medial longitudinal fasciculus (MLF). Bilateral INO (BINO) with horizontal gaze palsy is even rarer and indicates an extensive brainstem lesion involving both MLFs and the paramedian pontine reticular formation (PPRF). While multiple sclerosis is the commonest cause of bilateral INO in young adults, vascular causes must always be ruled out. We report the case of a 36-year-old previously healthy man who presented with sudden-onset binocular horizontal diplopia, bilateral adduction deficit with abducting nystagmus, bilateral horizontal gaze palsy, and bilateral lower motor neuron facial palsy. MRI revealed a non-haemorrhagic dorsal pontine infarct involving the MLFs and PPRF. Fundoscopy showed right eye retinal infiltrates, raising suspicion of a microvascular event. Further workup found marked hyperhomocysteinaemia, an under-recognised but reversible risk factor for thrombotic stroke in young patients. The patient was managed with dual antiplatelets, statins, B-vitamin supplementation, ocular surface protection, and orthoptic therapy. He showed partial recovery with normalisation of serum homocysteine at three months. This case highlights the need to consider vascular and metabolic causes, such as hyperhomocysteinaemia, in young adults presenting with bilateral INO. Early detection and correction of modifiable risk factors can improve outcomes and prevent recurrence.

KEYWORDS

Internuclear ophthalmoplegia, bilateral INO, medial longitudinal fasciculus, pontine infarct, hyperhomocysteinaemia, one-and-a-half syndrome, young stroke.

INTRODUCTION

Internuclear ophthalmoplegia (INO) results from interruption of the MLF[1], a compact longitudinal bundle in the brainstem that coordinates horizontal conjugate eye movements by connecting the abducens nucleus of one side to the contralateral oculomotor nucleus. Classically, INO presents with an adduction deficit in the ipsilateral eye and abducting nystagmus in the contralateral eye[1]. Bilateral INO is rare and indicates bilateral involvement of both MLFs.[2]

When bilateral INO coexists with horizontal gaze palsy, it localizes to the dorsal pons involving the MLFs and PPRF—a classic 'one-and-a-half syndrome,' which when bilateral becomes a 'bilateral one-and-a-half syndrome'[2,3]. In young adults, demyelinating disorders such as multiple sclerosis are the commonest cause[4]. However, vascular causes, trauma, infections, neoplasms and metabolic causes like hyperhomocysteinaemia-induced infarction must always be considered.[5]

Hyperhomocysteinaemia causes endothelial dysfunction, increased platelet aggregation and a prothrombotic state[5]. Its association with young stroke is well documented but remains under-recognized, especially in resource-limited settings where screening for homocysteine is not routine[6,7].

Case Study

A 36-year-old male with no significant past medical history presented with sudden-onset binocular horizontal diplopia, facial asymmetry, and mild gait imbalance. He denied headache, fever, trauma, or visual loss.

On examination, best-corrected visual acuity was 6/6 OU. Ocular motility testing revealed:

- Bilateral adduction deficit.
- Abducting nystagmus on attempted gaze.
- Complete bilateral horizontal gaze palsy, indicating PPRF involvement.
- Bilateral lower motor neuron facial weakness with loss of forehead wrinkles and inability to close eyes fully.

Pupils were equal and reactive with intact light reflexes. Vertical eye movements were normal. No signs of cerebellar ataxia were noted except mild unsteadiness on tandem gait. Fundoscopy revealed right eye retinal infiltrates near the inferotemporal arcade suggestive of microvascular occlusion.

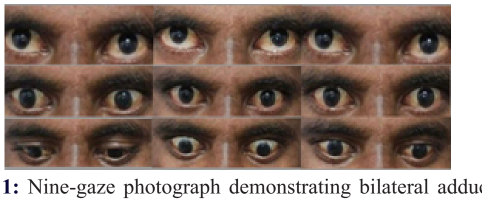

Figure 1: Nine-gaze photograph demonstrating bilateral adduction deficit with abduction nystagmus and preserved vertical gaze, consistent with bilateral internuclear ophthalmoplegia and horizontal gaze palsy.

An urgent MRI brain showed a non-hemorrhagic lacunar infarct in the dorsal pons involving both MLFs and the PPRF. MR angiography was unremarkable. SD-OCT showed mild RNFL thinning consistent with mild axial myopia but no glaucomatous damage.

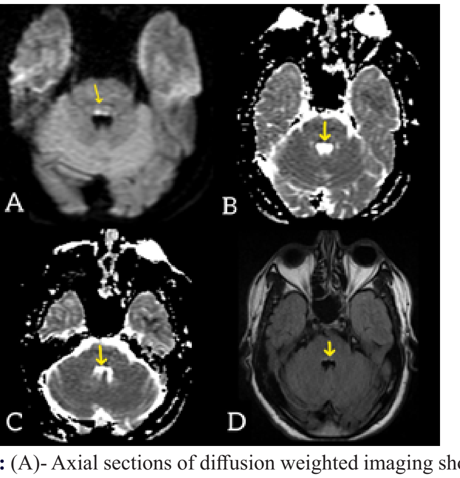

Figure 2: (A)- Axial sections of diffusion weighted imaging showing tiny focal areas of increased signal intensity in the dorsal pons (yellow arrows), (B and C) - Corresponding signal drop in the axial sections of

ADC images (orange arrows) and (D) - Axial sections of T2 FLAIR WI showing subtle hyperintensities (green arrow) in the dorsal pons - Combined features consistent with Acute lacunar non haemorrhagic infarct involving the dorsal pons (pontine tegmentum).

Laboratory Evaluation Showed:

- **Fasting Homocysteine:** 27 µmol/L (normal: 5–15 µmol/L)
- **Serum B12:** low-normal.
- **Lipid Profile:** borderline dyslipidemia.
- **Vasculitis Panel:** negative.
- Cardiac evaluation and echocardiography: normal.
- No evidence of inherited thrombophilia.

Differential Diagnosis:

The Main Differentials Considered Were:

1. **Multiple Sclerosis:** Common cause of bilateral INO in young adults; ruled out due to lack of other demyelinating lesions on MRI and negative CSF studies.
2. **Brainstem Tumour:** Excluded by normal imaging apart from the acute infarct.
3. **Vertebral Artery Dissection:** Unlikely due to normal MRA and absence of neck trauma.
4. **Ischaemic Infarct Due To Hypercoagulability:** Confirmed by elevated homocysteine and absence of other causes.

Management:

The Patient Was Admitted And Started On:

- Aspirin 150 mg and clopidogrel 75 mg daily.
- High-dose atorvastatin 40 mg daily.
- Folic acid 5 mg, vitamin B6 (pyridoxine 25 mg) and vitamin B12 injections.
- Eye care with lubricating drops and eye patching to prevent exposure keratopathy.
- Prism therapy and orthoptic exercises to manage diplopia.

He was counselled regarding dietary modifications, B vitamin-rich food intake and regular follow-up.

Outcome And Follow-up:

At 3 months, the patient reported significant improvement in facial asymmetry and a reduction in diplopia with partial recovery of horizontal gaze. Repeat homocysteine levels normalised to 11 µmol/L. Fundus exam showed resolution of the retinal infiltrate

DISCUSSION

This case highlights an unusual presentation of bilateral INO with horizontal gaze palsy due to an ischaemic pontine infarct in a young patient with hyperhomocysteinaemia.

While multiple sclerosis is a classic cause of bilateral INO in young adults, vascular infarcts account for about one-third of cases. Vertebral artery dissection and small vessel infarcts have been reported as causes of bilateral INO.[4,6,7]

Internuclear ophthalmoplegia caused by demyelination is due to immune-mediated myelin loss in the MLF, typically associated with multiple sclerosis and may present with relapsing-remitting symptoms and additional CNS involvement[2,4]. In contrast, hyperhomocysteinaemia-related INO is the result of a thrombotic infarct in the MLF region due to a prothrombotic metabolic state, presenting acutely and generally with fewer systemic neurological signs. Hyperhomocysteinaemia is an important but underdiagnosed risk factor for premature stroke. Elevated homocysteine promotes endothelial dysfunction, oxidative stress and thrombosis. Early recognition and vitamin supplementation can lower levels and reduce recurrence risk [7,8].

Retinal findings in this patient support an embolic or microvascular phenomenon associated with the same prothrombotic state. This underscores the importance of a full systemic vascular workup in any young patient with stroke-like ocular signs.

Table 1: Previous Reports Of Ocular Manifestation Of Stroke

Parameter	Rafay et al. 2024	Cárdenas et al. 2019	Falla et al. 2024	Present Case
Age	28 y/o male	29 y/o female	79 y/o female	36 y/o male
Aetiology	Hyperhomocysteinaemia	Vertebral artery dissection	Mesencephalic infarction	Hyperhomocysteinaemia
Imaging	ACA infarct	Pontomesencephalic infarct	Mesencephalic infarct	Dorsal pontine infarct
Ocular Features	None reported	Bilateral INO	WEBINO	Bilateral INO + gaze palsy
Risk Factors	High homocysteine	Vascular dissection	Hypertension, diabetes	High homocysteine
Outcome	Good recovery	Recovered	Partial recovery	Partial recovery

CONCLUSIONS

Bilateral INO with gaze palsy localises to the dorsal pons. In young patients, a vascular aetiology including hyperhomocysteinaemia must be considered alongside demyelination. Identifying and treating modifiable risk factors like hyperhomocysteinaemia can prevent recurrence and reduce morbidity.

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