



MIDBRAIN INFARCTION WITH INTERNUCLEAR OPHTHALMOPLEGIA: A RARE PRESENTATION

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ABSTRACT Internuclear ophthalmoplegia (INO) is a distinctive ocular motility disorder caused by damage to the medial longitudinal fasciculus (MLF), often due to ischemic stroke. We report the case of a 67-year-old male presenting with INO due to an acute midbrain infarction. The patient exhibited adduction deficit in the left eye with abduction nystagmus in the right eye, gait ataxia, slurred speech, and intentional tremors. INO results from MLF disruption and is commonly associated with cerebrovascular accidents. MRI findings confirmed midbrain infarction as the underlying pathology. Midbrain infarction causing Internuclear ophthalmoplegia is usually very rare presentation. Very few cases have been reported in literature. Our case reported below is one such rare presentation. This case underscores the importance of considering midbrain infarction as a cause of INO, particularly in patients with significant vascular risk factors. Detailed clinical examination and neuroimaging are essential for accurate diagnosis and management.

KEYWORDS : Internuclear ophthalmoplegia, Midbrain stroke, Medial longitudinal fasciculus, Ischemic stroke, Gait ataxia

INTRODUCTION

Internuclear ophthalmoplegia (INO) is a rare ocular motility disorder characterized by impaired adduction of the ipsilateral eye and dissociated nystagmus of the contralateral eye during horizontal gaze. This disorder results from damage to the medial longitudinal fasciculus (MLF), a key neural pathway that connects the oculomotor and abducens nuclei, facilitating coordinated eye movements. The most common etiologies of INO include ischemic stroke, multiple sclerosis, and traumatic brain injury^[1,2].

Midbrain infarcts leading to INO are uncommon but can be clinically significant, often presenting with additional neurological deficits depending on the extent of brainstem involvement^[3-4]. The vascular supply of the midbrain originates from perforating branches of the basilar and posterior cerebral arteries, making this region susceptible to ischemic injury due to embolism or small-vessel disease. Patients with predisposing risk factors such as diabetes mellitus, hypertension, and coronary artery disease are at an increased risk of developing such infarcts^[5].

Given the complex presentation of INO, a thorough neurological examination and advanced neuroimaging techniques such as MRI are crucial for accurate localization of the lesion. Early diagnosis and appropriate management, including addressing underlying vascular risk factors, are vital for optimizing patient outcomes and preventing recurrent strokes. This case report highlights an unusual presentation of INO due to midbrain infarction, emphasizing the need for heightened clinical awareness in patients presenting with acute ophthalmological and neurological symptoms.

Case Report

A 67-year-old male who is known diabetic since 8 years, known case of Coronary artery disease on irregular medication, known smoker and alcoholic since 30 years came with complaints of swaying while walking, dysarthria, blurring of vision and double vision since 4 days. On examination, his vitals were stable. Neurological evaluation revealed Dysarthria, adduction deficit in the left eye, abduction nystagmus in the right eye on right gaze, gait ataxia, intentional tremors in the right upper limb. Systemic examinations were unremarkable. Laboratory investigations showed HbA1C of 7.6%. ECG revealed T-wave inversions in V3-V6, lead I, and aVL with left ventricular hypertrophy. Echocardiography showed regional wall motion abnormalities in the LAD territory, moderate left ventricular dysfunction (LVEF: 40%), moderate mitral regurgitation, and mild

tricuspid regurgitation with pulmonary artery hypertension (RVSP: 31 mmHg).

Brain MRI revealed intra-axial T2/FLAIR hyperintensity with diffusion restriction in the midbrain, indicative of an acute infarct. Diffuse cerebral atrophy and small vessel ischemic disease were also noted. MR angiogram showed atheromatous changes in the right anterior cerebral artery (A2 segment), hypoplasia of the right anterior cerebral artery (A1 segment), and right vertebral artery. Carotid Doppler detected fibro-fatty plaques with 15-20% luminal narrowing in the right carotid bulb and 10-15% narrowing in the right common carotid artery.

DISCUSSION

Internuclear ophthalmoplegia (INO) is a well-recognized neurological disorder resulting from lesions affecting the medial longitudinal fasciculus (MLF), a critical brainstem structure responsible for coordinating eye movements. INO is characterized by impaired adduction of the affected eye and nystagmus in the contralateral abducting eye due to disrupted neural communication between the oculomotor and abducens nuclei (Deleu et al., 2005)^[6]. This disorder is commonly associated with cerebrovascular diseases, demyelinating disorders such as multiple sclerosis, and traumatic brain injuries (Chen et al., 2017)^[7].

Although ischemic stroke as a cause of INO is relatively uncommon, it remains clinically significant. Musa et al. (2023) reported that isolated midbrain infarcts leading to INO require a high degree of clinical suspicion for accurate diagnosis^[8]. Haymes et al. (2021) highlighted the vascular architecture of the midbrain, particularly the branches from the posterior cerebral and superior cerebellar arteries, which predispose this region to ischemic events that can result in INO^[9]. These findings are particularly relevant in older individuals with vascular risk factors.

The pathophysiology of INO in ischemic stroke is linked to the disruption of neural pathways involved in horizontal gaze coordination. The MLF transmits signals between the abducens nucleus in the pons and the oculomotor nucleus in the midbrain, ensuring synchronized eye movements. When the MLF is compromised due to an infarct, the affected eye exhibits impaired adduction, while the contralateral eye develops nystagmus during abduction (Deleu et al., 2005)^[6]. The severity of impairment varies depending on the lesion's location and extent, with more extensive

damage potentially leading to additional motor deficits.

In the present case, MRI revealed an acute ischemic lesion in the midbrain, confirming the infarct's role in the pathogenesis of INO. Small-vessel ischemic disease is a primary contributor to such infarcts, particularly in individuals with diabetes, hypertension, and a history of smoking (Sheik et al., 2023)^[10]. Musa et al. (2023) reported that diabetes mellitus increases the risk of brainstem infarctions by 2.6 times, which aligns with our patient's medical history^[8]. Haymes et al. (2021) further observed that paramedian midbrain infarctions often present with associated symptoms such as gait ataxia, dysarthria, and third nerve palsy, all of which were noted in this case^[9].

Neuroimaging plays a crucial role in diagnosing INO and distinguishing it from other ocular motility disorders. Diffusion-weighted MRI is particularly sensitive in detecting acute ischemic lesions (Chen et al., 2017)^[7]. In our case, MR angiography also revealed atherosclerotic changes, including hypoplasia and luminal narrowing in the right anterior cerebral and vertebral arteries, indicating an ischemic etiology (Musa et al., 2023)^[8]. These vascular changes underscore the role of systemic atherosclerosis in midbrain infarctions and highlight the need for aggressive vascular risk factor management.

The management of INO primarily focuses on treating the underlying cause. In ischemic stroke cases, optimizing vascular risk factor control is essential. Stroke prevention strategies include antiplatelet therapy, lipid-lowering agents, and strict glycemic control (Musa et al., 2023)^[8]. In this case, the patient had discontinued antiplatelet medication 20 days before the infarct, which likely contributed to the thromboembolic event. This reinforces the critical need for adherence to secondary prevention measures to reduce the risk of recurrence (Deleu et al., 2005)^[6].

From a therapeutic perspective, patients with INO due to ischemic infarction often experience partial or complete recovery over weeks to months. The extent of recovery largely depends on lesion severity and collateral circulation. Reports indicate that up to 75% of patients with ischemic INO show significant improvement within three months (Chen et al., 2017)^[7].

This case underscores the importance of recognizing midbrain infarction as a potential cause of INO, particularly in patients presenting with acute-onset diplopia, gait disturbances, and a history of vascular comorbidities. Early neuroimaging, prompt intervention, and rigorous vascular risk factor management are crucial for optimizing patient outcomes and preventing recurrence.

CONCLUSION

This case highlights the significance of considering midbrain infarction as an underlying cause of INO which is a rare presentation. Given the patient's history of vascular risk factors, timely diagnosis and management were crucial in guiding appropriate treatment and secondary prevention strategies. Neuroimaging remains the gold standard for diagnosing brainstem infarcts, and adherence to cardiovascular risk reduction measures is essential for preventing further ischemic events. Continued research on the prognosis and rehabilitation strategies for ischemic INO will further enhance our understanding of this rare but impactful neurological condition.

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