CASE REPORT

INTERNUCLEAR OPHTHALMOPLEGIA IN MULTIPLE SCLEROSIS PATIENT

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ABSTRACT

Introduction: *Internuclear ophthalmoplegia (INO) characterized by impaired adduction of the affected eye caused by a lesion in the medial longitudinal fasciculus (MLF). The common cause of INO is demyelinating disease including multiple sclerosis. INO arguably the most discrete localizing sign in medicine, has considerable value in predicting multiple sclerosis. Due to its high spatial resolution, MRI has allowed us to depict in vivo the anatomic organization of the human oculomotor nerve complex, the MLF, and related structures in the brainstem.*
Case Report: *A 48-year-old female presented with blurred vision of both eyes for 2 weeks, but then slowly resolved. Best corrected visual acuity presentation on both eyes were 20/20 with normal intraocular pressure (IOP). Adduction deficit observed on the left eye with no nystagmus. Convergence are not impaired in both eyes. Anterior segments was normal except minimal lens cloudiness in both eyes. Funduscopic examination result also within normal limit. Patient had undergone Computerised Tomography (CT-Scan) and the result was normal, no ischemic area or Space occupied lesion (SOL) or intracranial bleeding were found. Brain MRI showed bilateral optic perineuritis and multiple lesion in bilateral frontoparietalis and in the left side of the pons suggested multiple sclerosis.*
Discussion: *Impairment of adduction movement on the patient's left eye caused by demyelinating plaque related multiple sclerosis on the left side of the pons as shown MRI imaging. Management in this patient is based on the underlying condition.*
Conclusion: *Internuclear ophthalmoplegia in this cases maybe caused by lesion in the medial longitudinal fasciculus could be related with multiple sclerosis.*
Keywords: *Internuclear ophthalmoplegia, Medial longitudinal fasciculus, Multiple sclerosis*

INTRODUCTION

Internuclear ophthalmoplegia (INO) is a specific gaze abnormality characterized by impaired adduction of the affected eye with abduction nystagmus of the contralateral eye. It results from a lesion in the MLF in the dorsomedial brainstem, tegmentum of either the pons or the midbrain. The INO can be unilateral or bilateral and can be an isolated finding or may be associated with other brainstem signs and symptoms.[1]

As the MLF is a highly myelinated tract within the brainstem, the most common cause of INO in young people is demyelinating disease secondary to multiple sclerosis (41-54%). Other etiologies can include cerebral/brainstem vascular accidents (23-27%), infection (5-14%), head trauma (6%), brainstem tumour (4-5%), systemic lupus erythematosus (<5%),

nutritional and metabolic disorders, or degenerative disorders.[2]

Internuclear ophthalmoplegia, arguably the most discrete localizing sign in medicine, has considerable value in predicting multiple sclerosis or stroke, depending on whether the INO is bilateral or unilateral and on whether the patient is young or old.[3] Multiple sclerosis is a chronic inflammatory disease characterized by Central nervous system (CNS) plaques composed of inflammatory cells and their products, demyelinated and transected axons, and astrogliosis in both white and gray matter.[4]

We present a case of a patient with left eye Internuclear ophthalmoplegia (INO) in multiple sclerosis patient

CASE ILLUSTRATION

A 48-year-old female referred from Neurology department with blurred vision on both eyes for 2 weeks. The patient have been treated by neurologist with Gamma aminobutyric acid, Calcium channel blockers (CCBs), Angiotensin receptor blockers (ARB), and Biguanide. No history of trauma. The patient has uncontrolled Diabetes Mellitus since 2 years ago and uncontrolled hypertension since 10 years ago.

Best corrected visual acuity on both eyes were 20/20 with normal IOP. Slit lamp examination of both eyes were within normal except minimal lens cloudiness ODS. Eye movement was unremarkable on right eye, while left eye had adduction deficit with no restrain while convergence. there was no nystagmus observed. Other neuro ophtalmology examination including color test dan visual field were normal.

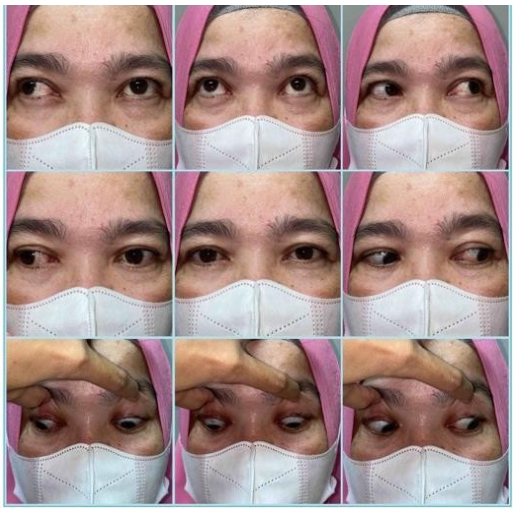

Figure 1. Photographs of eye movement show adduction limitations of the left eye

Patient had undergone Computerised Tomography (CT-Scan) and the result was normal, no ischemic area or Space occupied lesion (SOL) or intracranial bleeding were found.

Thus, we ordered MRI brain and showed bilateral optic perineuritis, multiple lesion in bilateral periventricular lateralis, juxtacortical region, bilateral frontoparietalis and in the left side of the pons suggested multiple sclerosis.

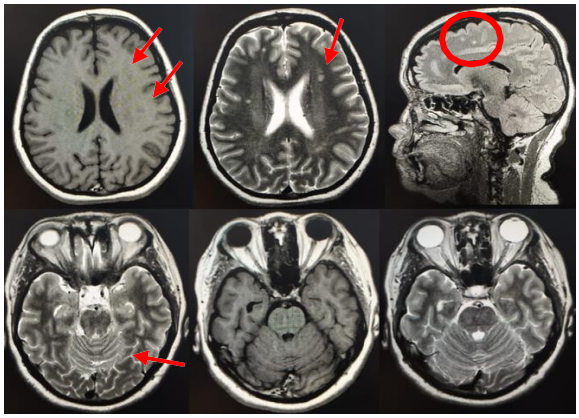

Figure 2. Brain MRI showed multiple lesions suspect multiple sclerosis (red arrows)

The patient then diagnosed OS Internuclear Ophthalmoplegia with multiple sclerosis and we collaborate Neurology department for Multiple sclerosis treatment.

DISCUSSION

The brainstem pathways for horizontal eye movements start from the abducens nucleus as the horizontal gaze center taking signal from the Paramedian Pontine Reticular Formation (PPRF). These pathways continue to the ipsilateral abducens nerve (cranial nerve VI) and the contralateral oculomotor nerve (cranial nerve III) through the MLF and end in conjugate horizontal eye movement in the ipsilateral direction to the side of the abducens nucleus (figure 3).[7]

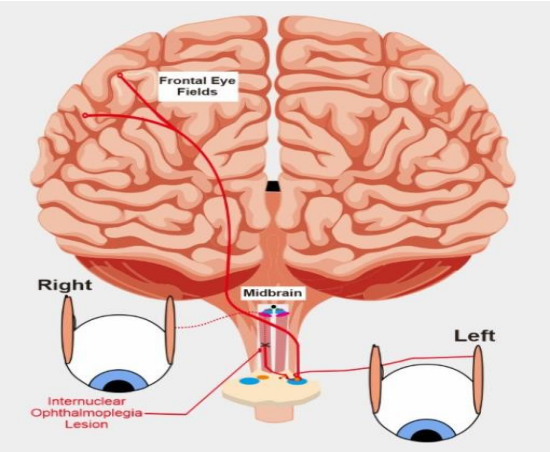

Figure 3. Neurological pathway for INO

INO is the clinical finding most commonly associated with lesions affecting the MLF on brain MRI. Thus, a lesion confined to the MLF disrupts the signal to the contralateral oculomotor nucleus and its activation of the medial rectus muscle.

Clinically, this results in preserved abduction of the contralateral eye with nystagmus, and weak or absent adduction of the ipsilateral eye.[8] However, depending on lesion location within the MLF, several specific neurologic manifestations may result.[9]

Our patient had an adduction under action on the left eye which was seen in eye movement test when she failed to move her eye to nasal. The affected eye adducts normally in convergence eye movement.[9] The ability to converge despite the absence of voluntary adduction indicates a more caudal lesion, with preservation of the medial recti subnuclei in the midbrain.[11] This finding distinguishes INO from the oculomotor nerve palsy.[12]

Patients with unilateral INO may have vertical diplopia due to a non-evident skew deviation, which can be relieved by using a small vertical prism. Skew deviation and Ocular tilt reaction (OTR) can be seen also with lesions independent of the MLF, for example, in the cerebellum or the thalamus. Tilt of the subjective visual vertical, the inner perception of verticality, is very often found in patients with MS.[13] In our patient, there is no vertical diplopia was found during examination.

Several pathologic studies have shown a clear anatomic relationship between the presence of lesions along the ipsilateral MLF and the presence of INO. Due to its high spatial resolution, MRI has allowed us to depict in vivo the anatomic organization of the human oculomotor nerve complex, the MLF, and related structures in the brainstem (typically white matter tracts have low signal intensity and nuclei have higher signal intensity). In patient with INO, MRI shown hyperintense lesions in the origin of the MLF on T-2 weighted images that were not detected using CT.[10]

In our patient, according to brain MRI that showed multiple lesion in bilateral periventricular lateralis, juxtacortical region, bilateral frontoparietalis and in the left side of the pons. This location could indicate damage to the abducens nucleus and the MLF it self. This finding consistent with the found in the patient which is ipsilateral adduction deficit of which the lesion was found (left side of the pons) and the multiple lession suggested to multiple sclerosis.[7]

Multiple sclerosis is a progressive central nervous system inflammatory disorder characterized by the accrual of white and gray matter lesions that exhibit varying degrees of inflammatory cell infiltration, demyelination, gliosis and neuroaxonal loss.[14]

The clinically isolated syndrome (CIS) is one of the most common presentations of MS and is characterised by an acute or subacute neurologic syndrome due to a solitary white matter lesion. INO is a brainstem CIS seen in approximately one-third of MS patients and can lead to a break in binocular fusion, diplopia, loss of depth perception, and reduction in quality of life.[14,15]

Treatment depends on the underlying cause. Acute strokes require hospitalization and neurological evaluation. Other pathologies require management by a physician (MS, infections, SLE). Most patients with demyelination, infectious, and traumatic etiologies show complete recovery. Patients with cerebrovascular disorders had a less favorable recovery. Recovery is said to be more likely if INO is isolated than if other neurological signs accompany it. According to some studies, recovery is also said to be less likely if there was a visible lesion causing internuclear ophthalmoplegia.[18] In our patient, we collaborate neurology departement to treat the multiple sclerosis as the underlying cause and no specific treatment from neuro ophthalmology division.

The majority of patients with persistent INO have minimal symptoms. Those with diplopia may benefit from botulinum toxin injections or Fresnel prisms. Surgical correction of strabismus may be used for patients with wall-eyed bilateral internuclear ophthalmoplegia.[18]

CONCLUSION

Internuclear ophthalmoplegia caused by lesion in the medial longitudinal fasciculus result in an adduction deficit could be related with multiple sclerosis. Management based on neurologist for multiple sclerosis treatment.

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