LETTER TO THE EDITOR

To The Editor

Recurrent Abducens Palsy in Relapsing-Remitting Multiple Sclerosis

Keywords: Abducens palsy, Multiple sclerosis, Recurrent ophthalmoplegia, Demyelination

Multiple sclerosis (MS) is an immune-mediated demyelinating and neurodegenerative disease of the central nervous system. The relapsing-remitting phenotype of MS is characterized by episodic relapses with complete or near-complete recovery between events. Oculomotor disorders can present as isolated events in MS relapses, most commonly as internuclear ophthalmoplegia, gaze-evoked nystagmus, and saccadic hypermetria.[1] Isolated cranial nerve palsies, however, are rare. In a study of 1489 individuals with MS, isolated cranial nerve palsies were the presenting symptom in 14 patients, and the only sign of disease relapse in 10 patients.[2] The abducens nerve is the third most commonly affected cranial nerve (1–1.6%), after the trigeminal (4.8%) and facial (3.7%) nerves.[3,4] To our knowledge, there are no accounts of recurrent abducens palsy in the MS population. We report a case of recurrent, isolated left abducens palsy in relapsing-remitting MS.

A 51-year-old woman presented with horizontal binocular diplopia, worse on leftward gaze. She has had 12, stereotyped episodes like this in the past 6 years, with near-complete recovery between episodes. However, there has been an accumulation of deficit after her most recent two episodes. Occasionally, the diplopia is preceded by 1–2 days of ipsilateral or holocephalic headaches that she describes as an aching, 7/10 pain that is accompanied by nausea. On examination, she had a left head turn and left eye esotropia of 18 prism diopters in primary gaze. Left eye abduction was limited to 80% of normal. Pupils were symmetrical to light and accommodation, visual fields were intact to confrontation, visual acuity was 20/20 (corrected with glasses), and fundoscopy revealed normal optic discs and retinas. Facial movement and sensation were symmetrical bilaterally. The tongue was midline and the palate elevated equally. On motor exam, bulk and tone were normal. Power was 5/5 in all major muscle groups. Reflexes were diffusely hyperreflexic (3+). Plantar response was flexor. Sensory examination was intact to pin-prick, vibration, and proprioception. There were no cerebellar signs or involuntary movements. Her gait was narrow-based, with normal velocity and stride length. Tandem gait was intact. Expanded Disability Status Scale (EDSS) score was 2.

She was diagnosed with relapsing-remitting MS 3 years after the onset of the diplopic episodes. The diagnosis was made based on multiple clinical episodes of abducens palsy over 6 years (dissemination in time), a magnetic resonance imaging (MRI) revealing multifocal punctate T2 lesions in the brain (periventricular, juxtacortical, and infratentorial) and cervical spinal cord (fulfilling the criteria for dissemination in space), as well as cerebrospinal fluid positivity for oligoclonal bands (77). Over the course of her care, we had considered the diagnosis of recurrent painful ophthalmoplegic neuropathy (previously known as ophthalmoplegic migraine), but this is a diagnosis of exclusion and was dismissed after disseminated radiological lesions and oligoclonal bands suggested a diagnosis of MS instead. Tolosa–Hunt syndrome was another possibility but pain, when present, preceded ophthalmoplegia, repeated MRI did not reveal pathology in the cavernous sinus, and there was no clinical improvement with corticosteroid therapy offered at initial presentation.

Anti-nuclear antibodies, rheumatoid factor, and anti-neutrophil cytoplasmic antibodies were negative. Inflammatory markers (erythrocyte sedimentation rate and C-reactive protein) were normal. Visual-evoked potentials and somatosensory-evoked potentials were normal. Vascular risk factors (dyslipidemia, diabetes, and hypertension) were absent. She had no other medical conditions. Her mother had had rheumatoid arthritis. There were no MRI lesions corresponding to possible localizations of a left abducens palsy. Despite subclinical radiological progression of demyelination, her EDSS score remained at 2. She was prescribed minocycline, but because of severe headache and recurrence of diplopia, she stopped the medication after 1 week. She declined further disease-modifying therapy.

The abducens nerve is susceptible to injury at several points: within the pons, as it exits the pons anteriorly, traverses the subarachnoid space, passes through Dorello's canal underneath the petroclinoid ligament, enters the cavernous sinus, and enters the orbit. In addition to demyelination, it is susceptible to vascular injury, compression by tumor, changes in intracranial pressure, infection, and neoplastic infiltration.[5] However, recurrent isolated abducens palsy narrows the etiology further. In a retrospective review of 782 patients, aged >50 years, with chronic, recurrent abducens nerve palsy, 57% had structural lesions of the petrous apex and cavernous sinus, 14.29% had recurrent painful ophthalmoplegic neuropathy, 14.29% had an internal carotid artery aneurysm, and 14.29% had microvascular disease.[6] Recurrent abducens palsy has also been reported in chronic inflammatory demyelinating polyradiculoneuropathy.[7] Because of focal demyelination, an MRI brain is a useful tool to detect associated features such as cranial nerve hypertrophy, contrast enhancement, or signal change.[8]

The case discussed here is unique for several reasons. The patient's clinical relapses manifested only as recurrent left abducens nerve palsy, despite radiological progression of MS lesions. She had no features associated with the horizontal binocular diplopia apart from an occasional tension headache that would disappear with the onset of her oculomotor symptoms. Her diplopia would then resolve without intervention over the course of several weeks, albeit longer with each relapse. Multiple MRIs of her brain, with and without gadolinium, did not demonstrate demyelination along the path of the abducens nerve, nor did it reveal any smaller pontine lesions that could otherwise explain her presentation. However, only one MRI scan was obtained during a relapse.

This case demonstrates recurrent isolated abducens nerve palsy as a rare presentation of MS relapse and shows that

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although spontaneous recovery is possible, disability may accumulate over time. In the absence of MRI evidence of demyelination, it is a diagnosis of exclusion and patients need to be worked up for other pathologies.

Conflict of Interest

None.

Statement of Authorship

SS: execution and write-up of first draft of the manuscript; CR-H: reviewing the manuscript; CG: reviewing the manuscript; DR: reviewing the manuscript; FT: conception of research subject and reviewing the manuscript.

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