CASE REPORT

Myasthenia Gravis Presenting as Lutz Posterior Internuclear Ophthalmoplegia

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ABSTRACT

Myasthenia gravis can mimic central neurological disorders and should be considered in the differential diagnosis of any form of pupil-sparing ophthalmoplegia. We report an unusual manifestation of myasthenia gravis presenting as bilateral internuclear ophthalmoplegia (INO) of abduction, sometimes referred to as Lutz posterior INO (or reverse INO).

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Introduction

Myasthenia gravis (MG) can mimic other neurological conditions. About 85% of patients present with ocular involvement that can resemble an ocular motor nerve palsy, or gaze palsy such as an internuclear ophthalmoplegia (INO)¹ or one-and-a-half syndrome.² Approximately 80% of those patients develop generalized MG, typically within the first year.³ About 15–20% of patients will develop life-threatening bulbar and respiratory muscle weakness.⁴ To our knowledge this is the first report of myasthenia presenting as bilateral pseudo INO of abduction, sometimes referred to as Lutz posterior INO (or reverse INO).^{5,6}

Case report

A 17-year-old mildly myopic, but otherwise healthy male student presented with 10 days of binocular vertical diplopia and 1 year of generalized fatigue. One week before presentation to us, he consulted an outside ophthalmologist who found a 4 prism dioptre right hypertropia that was worse in left gaze and with right head tilt, suggesting an acute right superior oblique palsy. The ophthalmologist obtained a brain MRI that was normal. Four days later, the patient devel-

oped bilateral partial ptosis that worsened throughout the day. He had no bulbar symptoms, no shortness of breath, no overt limb weakness, and no history of head trauma. Family history was significant for his father having Hashimoto's thyroiditis. Examination revealed limitation of abduction by 50% bilaterally (–2 OU) with slow bilateral adducting “nystagmus” (video 1) and impaired convergence. His upper lids fatigued on sustained upgaze and recovered with rest. His orbicularis oculi and orbicularis oris strength were decreased. Snellen visual acuity was 20/20 bilaterally. Anterior segment and dilated fundus examinations were normal.

A diagnosis of ocular MG was made clinically. A myasthenia panel was positive for acetylcholine receptor binding and modulating antibodies. A chest CT did not show a thymoma. He received 30 mg of oral prednisone daily, and his binocular diplopia resolved gradually over 1 month. His examination at that time was notable for orthotropia in primary position, despite mild (–0.5) abduction deficits bilaterally, slow adduction nystagmus of the right eye only, and mild left ptosis. His orbicularis oculi and oris strength returned to normal.

Discussion

Injury to the segment of the medial longitudinal fasciculus between the abducens nucleus and the contralateral oculomotor subnucleus for the medial rectus muscle results in an INO.⁶ INOs may be unilateral or bilateral and may be associated with an ipsilateral hypertropia when unilateral or asymmetric. Cogan classified INO into anterior (rostral) or posterior (caudal); both forms are characterized by adduction deficits and abduction “nystagmus.” Cogan postulated that an anterior INO localized to the midbrain because of impaired convergence, and a posterior INO localized to the pons because of spared convergence; however, the presence of convergence likely is related to effort rather than localization.⁷

In 1923 Lutz used the term posterior INO for an abduction deficit with adduction weakness affecting both saccades and pursuit but not vestibular stimulation, not to be confused with Cogan’s posterior INO. Sometimes Lutz’s posterior INO⁵ is termed a reverse INO, but in 1975 Kommerell suggested the less confusing term, INO of abduction.⁸

The abducting “nystagmus” associated with a typical anterior INO is an adaptive response (overshoot dysmetria) because medial rectus weakness causes increased innervation to both itself and the yoked contralateral lateral rectus (Hering’s law of dual innervation). Patching the eye with the abducting “nystagmus” can decrease the oscillation, supporting this hypothesis⁹; this concept can be extended to our patient, explaining the bilateral gaze-evoked adducting “nystagmus” observed with bilateral lateral rectus weakness due to ocular MG.

In this patient myasthenia mimicked a pseudo INO of abduction (Lutz posterior INO) thus stressing the importance of considering MG in the

differential diagnosis of any form of pupil-sparing ophthalmoplegia.

Disclosure of interest

The authors report no conflict of interest.

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