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Hypertropia in unilateral isolated abducens palsy

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Abstract

Purpose—To evaluate the incidence and features of hypertropia in abducens nerve palsy.

Methods—The records of consecutive patients with unilateral, isolated, previously unoperated abducens nerve palsy were reviewed for binocular alignment on cover testing, Krimsky measurement, or Hess screen testing. Patients with associated cranial nerve palsy (including bilateral abducens palsies), orbital disease, myasthenia gravis, Horner syndrome, hemiplegia, cerebellar signs, arteritis, or previous strabismus surgery were excluded. Control subjects underwent complete examination to confirm normality.

Results—A total of 79 patients were included (40 males; mean age 49.2 years). Hypertropia in lateral or central gazes was present in 15 of 79 cases (19%) on alternate cover or Krimsky testing, in 32 of 56 cases (57%) on Hess screen testing, and absent in all 30 normal controls. Of cases with hypertropia, the mean of the greatest hypertropia in lateral or central gaze on was $5.0^{\Delta} \pm 2.3^{\Delta}$ (standard deviation; range, 1^{Δ}–8^{Δ}) routine clinical examination, and $5.8^{\Delta} \pm 4.2^{\Delta}$ (range, 2^{Δ}–24^{Δ}) on Hess screen testing. Of 39 cases with partial abducens palsy evaluated by Hess screen testing, the ipsilesional eye was hypertropic in 24 (61%) and hypotropic in 15 cases (39%).

Conclusions—Small-angle hypertropia is common in isolated, unilateral abducens and does not necessarily imply existence of multiple cranial neuropathies or skew deviation.

The abducens nerve is the most susceptible to neuropathy of the ocular motor cranial nerves. The estimated annual incidence of abducens palsies is 11.3/100,000.[1] Richards and colleagues[2] reported on 4,278 cases of extraocular muscle palsy, of which abducens palsy comprised 44%; oculomotor palsy, 28%; trochlear palsy, 15%; and combined cranial nerve palsies, 13%. The most common etiology of abducens palsy in that series was idiopathic

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(26%), followed by neoplasm (22%), trauma (15%), and vascular pathology (13%). The incidence and etiologies of extraocular muscle palsies vary based on the population studied and investigations performed.

There is little data concerning hypertropia associated with isolated abducens palsy. Slavin[3] described 16 patients, all exhibiting hypertropia ranging from 4^{Δ} to 16^{Δ} (mean, 8.2^{Δ}). Wong and colleagues[4] detected small hypertropia in 22% of 27 patients by alternate prism-cover testing, but in 74% using Maddox rod testing. Holmes and Leske[5] reported that 27% of 56 patients with chronic abducens palsy had hypertropia ranging from 1^{Δ} to 8^{Δ} (median, 3^{Δ}). If hypertropia is observed with abducens palsy, clinicians may consider multiple cranial neuropathies or a skew deviation[6] and initiate further neurological investigation. The purpose of the present study was to ascertain in a larger series of patients whether hypertropia is a typical concomitant of abducens nerve or a coexisting lesion.

Subjects and Methods

This study was approved by the UCLA Institutional Review Board and conformed to guidelines of the US Health Insurance Privacy and Accountability Act of 1996. The medical records of consecutive patients seen between July 1988 and October 2012 by the senior author (JLD) were retrospectively reviewed to identify patients diagnosed with abducens palsy. Records were reviewed for history, including neurologic signs and symptoms, and detailed ophthalmic examinations performed, including ductions, saccades, binocular alignment by alternate cover testing or Krimsky testing in cardinal gaze positions at distance and near, and Hess screen testing if appropriate. Hess screen testing is not reliable or indicated in patients, including most children, who have strabismic suppression or anomalous retinal correspondence. Only cases of unilateral, isolated, previously unoperated abducens palsy were included. Cases likely to be associated with intrinsic brain lesions that might result in skew devision as well as cases where muscular disease, neuromuscular disease, or involvement of additional cranial nerves might account for hypertropia were excluded, as were bilateral cases and cases with previous strabismus surgery. Isolated, unilateral abducens palsy by definition excluded cases with associated cranial nerve palsies (including bilateral abducens palsy), orbital disease, myasthenia gravis, Horner's syndrome, hemiplegia, cerebellar signs (nystagmus, ataxia, or dysmetria), or giant cell arteritis. Complete abducens palsy was defined as nonrestrictive inability to abduct past midline, and incomplete palsy as a lesser limitation.

Hess screen testing was also performed in a paid control group of adults (age range, 18–70 years) who were recruited by advertising and who underwent complete ophthalmic and motility examinations to verify that they had no ocular pathologies (Pseudophakia was considered normal in older adults).

The examination data were taken from the first visit to the senior author (JLD). Hess screen testing was peformed as previously described.[7] Primary rather than secondary deviation measurements were analyzed. For specificity, hypertropia was recorded for this study by either cover testing, Krimsky or Hess screen testing in central gaze and/or 30° dextro- or levoversion along the horizontal meridian; hypertropia present only in tertiary gaze positions

was disregarded. Krimsky testing was performed in central gaze only. Hess screen angles were doubled to convert to prism diopters. Results are reported as mean plus or minus standard deviation.

Results

Of a total of 155 patients with abducens palsy, 79 met inclusion criteria (40 males; mean age, 49.2 years [range, 3 weeks to 93 years]). Patients with any of the following were excluded: multiple cranial nerve involvement, prior strabismus surgery; resolved on presentation, orbital surgery, cerebellar signs; Brown syndrome; blowout fracture, giant cell arteritis, hemiplegia; one and half syndrome, Horner syndrome. Some cases were excluded for multiple, concurrent reasons. Only 8 cases of abducens palsy with trochlear palsy but without oculomotor palsy were excluded. Alignment measurements were obtained by alternative cover testing in 73 included patients and by Krimsky testing in 6 patients. Hess screen testing was performed in 56 patients.

Symptom duration at presentation ranged from 1 day to 12 years (mean, 14.2 months). Etiologies are listed in Table 1. Magnetic resonance imaging (MRI) or computed X-ray tomography (CT) of the brain and in some cases of the orbits was performed in 65 of 79 patients (82%), with most (54/65) undergoing MRI. Patients considered to have microvascular abducens palsy underwent MRI or CT in 20 of 28 cases (71%); imaging was considered clinically unnecessary in the remaining cases.

Alternate cover or Krimsky testing demonstrated hypertropia in central and/or lateral gaze in 15 of 79 cases (19%) and on Hess screen testing in central and/or 30° lateral gazes in 32 of 56 cases (57%). Only 1 patient exhibited hypertropia in lateral gaze by alternative cover testing that was not also detected in central gaze. Central gaze hypertropia was present on alternate cover or Krimsky testing in 14 of 79 (18%) and on Hess screen testing in 19 of 56 cases (34%). Hess screen testing demonstratred hypertropia in 11 of 17 cases (65%) with complete and 21 of 39 (54%) with partial abducens palsy. By comparison, Hess screen testing did not demonstate any hypertropia in any of the 30 control subjects in central or lateral gazes. Only a small exodeviation was seen in a few controls, compatible with physiologic exophoria. No heterophoria was noted by alternate cover testing in any control subject.

Of cases of abducens palsy with hypertropia, the mean maximum hypertropia in lateral or central gaze on clinical examination was $5.0^{\Delta} \pm 2.3^{\Delta}$ (range, 1^{Δ} –8^{Δ}); on Hess screen testing, $5.8^{\Delta} \pm 4.2^{\Delta}$ (range, 2^{Δ}–24^{Δ}). The ipsilesional eye was hypertropic in 24 of 39 cases (62%) and hypotropic in 15 cases (39%). In 16 of 20 cases of ipsilateral hypertropia (80%), the hypertropia on Hess screen testing was significanatly greater in abduction than in central gaze ($5.2^{\Delta} \pm 2.8^{\Delta}$ vs $2.4^{\Delta} \pm 3.0^{\Delta}$; $P = 0.0000076$; Table 2). In the 7 of 12 cases of contralateral hypertropia (58%), the hypertropia on Hess screen testing was greater in abduction than in central gaze ($5.1^{\Delta} \pm 6.4^{\Delta}$ vs $2.9^{\Delta} \pm 2.9^{\Delta}$), although this difference was not statistically significant ($P = 0.17$; Table 3). In 23 of 32 cases (72%) there was hypertropia greater in abduction than in adduction on Hess screen testing. Of 28 cases attributed to microvascular disease, 16 (57%) exhibited hypertropia.

Discussion

The present study confirms and extends findings of smaller, earlier studies reporting that hypertropia is commonly associated with isolated, unilateral abducens nerve palsy.[3–5] Etiology of this hypertropia has been controversial but has been hypothesized to include trochlear palsy, skew deviation, and physiologic hyperphoria unmasked by horizontal strabismus. Earlier investigators recognized the association of hypertropia and abducens palsy and allowed existence of small hypertropia before implicating other etiologies. Kestenbaum[8] supposed that the lateral rectus muscle weakly inhibited vertical duction, stating, “In abducens paresis a vertical component is sometimes found in the distance between the images.” He elaborated that hypertropia is often seen in the field of action of the paretic lateral rectus muscle in sursumversion and deorsumversionup- and downgaze. Commenting on Kestenbaum’s description of hypertropia in abducens palsy, Smith[9] opined that hypertropia $<3^{\Delta}$ might be attributable to slip of the weakened lateral rectus muscle relative to the globe. O’Donnell and colleagues[6] stated that a small hypertropia may accompany abducens palsy but that hypertropia $>4^{\Delta}$ should suggest additional pathology, such as trochlear palsy or skew deviation. Wong and colleagues[4] found small hypertropia in 22% of patients with abducens palsy but further reported that 80% of normal subjects have vertical hyperphoria: in 10 normal subjects Wong and colleagues found no hypertropia on prism and cover testing in any gaze position and hypertropia of $1.52^{\Delta} \pm 1.49^{\Delta}$ on Maddox rod and prism testing in central gaze. They suggested that the small hyperphoria is unmasked by paralytic esotropia and that diagnosis of multiple cranial nerve palsies or skew deviation not be entertained in patients with abducens palsy unless the hypertropia exceeds 5^{Δ}. The high prevalence of hyperphoria reported by Wong and colleagues[4] in normal subjects contrasts with Hess screen test results of the present study, which demonstrated no detectable hypertropia in 30 control subjects who had been carefully confirmed to be free of ocular pathology. This raises the question of whether Maddox rod and prism testing, employed by Wong and colleagues,[4] might induce an artifact interpreted as hypertropia or whether Hess screen testing might be insensitive to very small vertical heterophoria.

The current study included 17 of 79 patients with hypertropia exceeding 5^{Δ} on either alternate cover testing or Hess screen testing not attributable to additional cranial nerve palsies or clinically plausible skew deviation. Slavin[3] described 16 patients with abducens palsy and a mean hypertropia ranging from 4^{Δ} to 16^{Δ} (8.2^{Δ}), and present in central gaze in 63%[3]; we found a mean central gaze hypertropia of 5.8^{Δ} in 34 of cases on Hess screen testing, less than that reported by Slavin but greater than the 3^{Δ} reported by Holmes and Leske.[5] Slavin and colleagues[10] found vertical heterophoria in 77% of normal subjects in any field of gaze but in only 1 of 61 patients with vertical heterophoria in central gaze. These variations could of course be attributable at least in part to different testing techniques. The range of primary gaze hypertropia reported in association with abducens palsy exceeds normal vertical fusional amplitudes of 3^{Δ}–4^{Δ},[11] and at least in many cases would be unlikely to represent the unmasking of normal vertical heterophoria suggested by Wong and colleagues.[4]

Although trochlear palsy seems an obvious potential explanation for associated hypertropia in abducens palsy, this association has in fact been rarely demonstrated. The series of 3,000

consective cases of extraocular muscle palsies from the Mayo Clinic included only one abducens palsy with a contralateral trochlear palsy.[12–14] One case of abducens palsy associated with trochlear palsy due to herpes zoster was reported in a series of 165 cases.[15] In a series of 193 patients with extraocular muscle palsy and an intracranial neoplasm, only 5 cases included both abducens and trochlear palsies.[2] Walsh and colleagues[16] described 5 patients with abducens palsy and contralateral trochlear palsy. These reports suggest that abducens palsy with hypertropia secondary to a trochlear palsy is actually a rarity. We encountered 8 cases of abducens palsy and trochlear palsy without oculomotor nerve palsy in 155 cases of abducens palsy, which is a higher incidence than the other series; however, we excluded such cases from the current analysis.

In some patients with abducens palsy the large-angle esotropia could cause coexisting trochlear palsy to go unrecognized. Holmes and colleagues[5] speculated that small-angle hypertropia due to fusion loss secondary to the large esotropia cannot be easily distinguished from a hypertropia secondary to a trochlear palsy in patients with abducens palsy. However, fusion loss in other settings is not often associated with hypertropia. For example, a prospective, observational study of 44 children with infantile esotropia in whom 19 also underwent full-time alternate occlusion to preclude binocular experience, only 6 (14%) exhibited hypertropia.[17] A 6-month, multicenter, observational study of infantile and acquired esotropia in 167 children under age 6 years employed alternate cover testing, yetdid not note any cases of hypertropia.[18] If fusion loss were sufficient to unmask ubiquitous hyperphorias, hypertropia should be a nearly universal accompaniment to all forms of constant horizontal strabismus. Obviously such is not the case.

Skew deviation is another possible cause of hypertropia with associated with abducens palsy. Some patients in the current series had intracranial neoplasms external to the brain substance, but many were attributed to other etiologies. The majority of our 28 patients with microvascular abducens palsy (57%) exhibited hypertropia, and MRI or CT scans were performed to rule out causative lesions in 71% of these cases. The remaining patients were diagnosed with microvascular abducens palsy based on age, past medical history, and clinical course. Skew deviation could not have plausibly existed in such a large number of patients exhibiting abducens palsy without other neurological signs or imaging evidence of brain pathology.

We suggest another possible etiology for hypertropia in setting of isolated abducens palsy: compartmental paralysis or paresis of the superior or inferior zones of the affected lateral rectus muscle.[19] The horizontal rectus muscles of humans and monkeys have been demonstrated by three-dimensional histological reconstruction to consist of distinct superior and inferior muscle fiber compartments, separately innervated by either a superior or inferior abducens nerve division.[20,21] This anatomical division of the lateral rectus muscle into separate compartments has been correlated with multipositional MRI demonstrating differential contractility in the superior and inferior lateral rectus muscle compartments during ocular counter-rolling in a manner that would contribute to ocular torsion.[22] Computational modeling suggests that lateral rectus compartmentalization could impart significant torsional and vertical effects to the lateral rectus muscle.[22] The possibility of transmission of these differential forces to different locations on the globe was demonstrated

by Shin and colleagues,[23] whose dual-channel biomechanical measurements in bovine specimens showed substantial mechanical independence of forces in the superior and inferior compartments of both extraocular muscles and tendons.

Further evidence that the lateral rectus muscle could have differentially compartmentalized function is provided by the observation that in 8% to 15% of human cadavers, the abducens nerve is grossly divided into two nerves paralleling one another.[24–27] The lateral rectus muscle itself can also exhibit splitting, as seen congenital fibrosis of the extraocular muscles type 1,[28] congenital oculomotor palsy,[29] congenital trochlear nerve palsy,[29] and Duane syndrome.[30,31] Taken together, these observations imply that the lateral rectus muscle functions as if two separately controlled muscle bellies coursing in parallel proximity but inserting on different scleral sites. Each of these lateral rectus bellies is innervated by a separate division of the abducens nerve.

If a patient were to have a abducens palsy lesion affecting one lateral rectus compartment more than the other, the imbalanced force could have significant cyclovertical effects greater in abduction and less in adduction during lateral rectus contraction and relaxation, respectively. This effect was observed in 23 of 32 of the current cases (72%) of abducens palsy. The hypertropia could be ipsilateral or contralateral to the abducens palsy, presumably depending on whether the superior or the inferior compartment was weaker. In the current study patients had a higher probability of a hypertropia ipsilateral to the abducens palsy (62%). This is consistent with Slavin's results of 56% of patients having hypertropia ipsilateral to abducens palsy.[3]

Wong and colleagues[4] found no correlation between laterality of abducens palsy and hypertropia and no variation in hypertropia consistent with associated vertical rectus or oblique muscle paresis in their patients with abducens palsy, but in contrast to the current report, they found the hypertropia to be comitant.[4] Absence of significant incomitance could be due to Wong and colleagues' small sample size.[4] The present, larger study also found no pattern of hypertropia consistent with associated vertical or oblique muscle paresis. Supporting a mechanism of hypertropia related to the involved lateral rectus muscle itself, the present study observed incomitent hypertropia significantly greater in abduction than central gaze in 20 cases of hypertropia ipsilateral to abducens nerve palsy. In 12 cases of hypertropia greater contralateral to abducens palsy, the difference did not reach statistical significance, perhaps due to the smaller sample size of such cases. However, both trends are consistent with the expection that ipsilateral hypertropia would increase in abduction during partial contraction of a relatively preserved superior lateral rectus compartment, and contralateral hypertropia would increase during partial contraction of a relatively preserved inferior lateral rectus compartment. We also anticipated more frequent hypertropia in patients with partial versus complete abducens palsy, but our finding of roughly similar prevalence is inconsistent with this assumption (54% partial versus 68% complete). However, it is unknown whether the clinical distinction between "partial" and "complete" abducens palsy accurately reflects residulal function in putative lateral rectus compartments.

Slavin[3] found the maximal hypertropia was ipsilateral to the abducens palsy in 14 of 16 patients (2/16 in central gaze) and undetectable in gaze contralateral to the abducens palsy.

We found 72% of 32 patients to have the maximal hypertropia to be ipsilateral to the abducens palsy, 16% to be the same, and only 13% to be in gaze contralateral to the abducens palsy. These observations strongly implicate abnormal lateral rectus function as the cause of the hypertropia.

Recommendations vary for clinical investigation of isolated abducens palsy, and recognition of hypertropia might motivate more extensive neurological evaluation. Palsies involving multiple cranial nerves or associated with other neurological signs or symptoms require etiologic investigation, as do palsies in patients <50 years of age.[32,33] Published algorithms sought to reduce extensive investigations and imaging, especially for patients >50 years of age, who have vascular risk factors such as diabetes and hypertension.[1,6,34–37] Some authors have argued that all patients should undergo brain MRI, even those with vasculopathic risk factors and isolated abducens palsy. A prospective imaging study in 23 patients with acute ocular motor mononeuropathies (4 with abducens palsy) demonstrated significant pathology in 14% of patients >50 years of age.[38] Another prospective study of abducens palsy identified a causative lesion in 63% of 43 patients; 15% of patients with vasculopathic risk factors had significant lesions on imaging.[39] Warwar and colleagues[40] reported a patient with a abducens palsy and vasculopathic risk factors who nevertheless died of pituitary apoplexy.[40] Authors of the foregoing studies have recommended cranial imaging all patients with abducens palsy, regardless of other considerations.

Since clinical recommendations vary, clinicians might consider tailoring evaluation to the specific patient population, mindful that hypertropia in the setting of isolated nerve palsy seldom implies additional pathological process. However, since the current series was ascertained in a tertiary academic referral practice, the results may not be generalizable to all settings.

The present report has the limitations of a retrospective, referral center–based study for which data collection was not initiated with the major hypothesis in mind. Not all alignment data was obtained using both prism cover and Hess screen testing, and not all patients underwent brain imaging. However, since data collection was by a single academic practice prior to the development of any particular hypothesis, there is little likelihood of data collection bias. Strengths of the study include a large number of consecutive patients in whom detailed evaluations were performed using uniform procedure, with Hess screen testing in the majority.

Our results have demonstrated that cases of unilateral, isolated abducens palsy commonly have associated incomitant hypertropia. This hypertropia does not typically imply trochlear palsy or a skew deviation but seems to be consistent with selective compartmental paralysis or paresis of the superior or inferior zones of the affected lateral rectus muscle. This knowledge should help refine clinical evaluation of abducens palsy and with further study might influence surgical therapy.

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Table 1

Etiology of abducens palsy

Etiology	Prior treatment	N (%)
Microvascular		28 (35.4)
Tumor		10 (12.7)
Meningioma	Resection (2); gamma knife (2)	4
Nasopharyngeal carcinoma	Gamma knife; chemotherapy radiation (2)	2
Abducens schwanomma		2[a]
Sarcoma		1[a]
Multiple myeloma		1
Trauma		10 (12.7)
Idiopathic		10 (12.7)
Other		21 (26.6)
Migraine		4
Post-viral		3
Aneurysm	Embolization; clipping	2
Arteriovenous malformation	Embolization	2
Cerebrovascular accident		2
Neurovascular compression		2
Arnold-Chiari malformation		2
Meningitis		1
Retrobulbar block		1
Carotid-cavernous fistula	Embolization	1
Idiopathic intracranial hypertension		1

Table 2

Ipsilateral hypertropia demonstrated by Hess screen test in abducens palsy

	Sursumversion	
	ET 15.0 ± 14.1, HT 0.7 ± 2.2	
Abduction	Central:	Adduction
ET 47.9 ± 23.8, HT 5.2 ± 2.8	ET 24.6 ± 14.7, HT 2.4 ± 3.0	ET 7.4 ± 9.6, HT 2.5 ± 2.8
	Deorsumversion	
	ET 23.9 ± 15.4, HT 4.5 ± 5.1	

ET, esotropia; *HT*, hypertropia.

Means ± standard deviations in prism diopters for 20 patients for the primary deviation in central and 30° secondary gazes.

Table 3

Contralateral hypertropia demonstrated by Hess screen test in abducens nerve palsy

	Sursumversion	
	ET 19.0 ± 13.2, HT 1.25 ± 5.0	
Abduction	Central	Adduction
ET 36.0 ± 19.0, HT 5.1 ± 6.4	ET 20.6 ± 14.3, HT 2.9 ± 2.9	ET 8.0 ± 8.2, HT 1.2 ± 2.6
	Deorsumversion	
	ET16.0 ± 14.4, HT 1.0 ± 2.0	

ET, esotropia; *HT*, hypertropia.

Means ± standard deviations in for 12 patients for the primary deviation in central and 30° secondary gazes.