Think Superior Oblique Palsy

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ABSTRACT

Superior oblique palsy is thought to be the most frequent cause of acquired vertical diplopia. It is often the result of blunt head trauma but may also be caused by brain tumor, meningitis, diabetes, lesions of the cavernous sinus, and superior orbital fissure as well as arteriosclerosis.

The characteristics frequently include a small vertical strabismus in primary gaze, ipsilateral inferior oblique overaction with an increased hypertropia to the contralateral side, a "V" pattern, excyclotorsion, and a positive Bielschowsky head tilt test to the ipsilateral side. In a typical case, it is not difficult to make the proper diagnosis.

Several patients will be described whose superior oblique palsy was marked by an association with another motility disturbance.

Case Reports

A. SIXTH NERVE PALSY

(1) A 41-year-old male developed a right sixth nerve palsy following head trauma from a motor vehicle accident. He complained of horizontal diplopia and had 35 prism diopeters of right esotropia, and an absence of abduction of the right eye beyond the midline. All other versions were full.

One week following injury, $2 \times 10^{-3} \mu g$ (5 units) of botulinum toxin, type A, was injected into the right medial rectus muscle. Three weeks after injection, there were 5 prism diopeters of esotropia in the primary position and the horizontal image separation could be eliminated in two to three degrees of left gaze. Adduction of the right eye was limited to five degrees.

At this time, the patient noted tilting of the image of the right eye and three degrees of excyclotorsion was measured. A four prism diopter right hypertropia was noted in primary gaze. The Bielschowsky head tilt test was positive to the right and a diagnosis of right superior oblique palsy was made. The hypertropia and excyclotorsion has persisted for two years following injury and injection.

(2) A 55-year-old male was seen several months after sustaining severe head trauma in an airplane crash. He had a bilateral sixth nerve paralysis with 100 prism diopeters of esotropia and abduction limited to 20° short of midline in each eye (Figure 1, top). Vertical rotations were full laterally. A head turn was required to fixate in the primary position with either eye.

A medial rectus recession and Jensen transposition was performed bilaterally. This resulted in an esotropia of eight prism diopeters and moderate limitation of abduction (Figure 1, bottom). For the first time, the patient noted excyclotorsion and this was measured to be 15°. Two prism diopeters of left hypertropia was noted and the Bielschowsky head tilt test was positive to both sides, confirming the diagnosis of bilateral superior oblique palsy.

COMMENT: In each case, the esotropia was large enough that the image tilt or small vertical deviation produced by the superior oblique palsy was not recognized. After the deviation was minimized, by occlusion therapy or following surgery, torsion was noted and it became clear that superior oblique palsy co-existed with the sixth nerve paralysis. This should not be unexpected, since blunt head trauma was the cause of abducens weakness in both patients and is also a common etiology of fourth nerve paresis. Especially with a very large angle esotropia (as in the case of bilateral sixth nerve palsy), a small vertical component of the deviation may be difficult to uncover.

It seems unlikely that the occlusion injection to the right medial rectus in Case 1 resulted in sufficient leakage of toxin to affect the right superior oblique muscle with an apparent paresis. The vertical deviations, which have been noted following botulinum injection, have almost all been due to ipsilateral inferior rectus weakness and have been transient. The superior oblique palsy in Case 1 has been present for two years.

In Case 2, 15° of excyclotorsion might have been observed preoperatively, had we looked for it. It has been suggested that the superior oblique tendon may have been caught up or

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166 JULY/AUGUST 1986, VOLUME 23, NUMBER 4
damaged when splitting the superior rectus in the Jensen procedure. The chances seem remote, since the superior oblique palsy was bilateral, the superior rectus was split under direct observation, and this has not been a recognized or reported complication of performing the Jensen procedure.

**B. UNUSUAL HORIZONTAL DIPLOPIA**

(1) A 60-year-old male complained of intermittent horizontal diplopia only when lying on his right side with his head slightly tilted to the right. This was noted while watching television, not when driving, walking, reading and sitting upright while watching television. In primary gaze, at both distance and near fixation, there were 28 prism diopters of intermittent exotropia and 4 prism diopters of intermittent left hypertropia (Figure 2). A moderate left inferior oblique overaction was noted and the left hypertropia increased to 25 prism diopters in right gaze. Versions were otherwise full bilaterally. Double Maddox rod testing indicated two to three degrees of exocyclotorsion. The Bielschowsky head tilt test was positive to the left. A diagnosis of left superior oblique palsy in association with intermittent exotropia was made.

(2) A four-year-old girl was seen with a history of intermittent left esotropia since infancy. No previous treatment had been given. The parents also noted the left eye occasionally drifted upward.

Ten prism diopters of intermittent esotropia and three prism diopters of intermittent left hypertropia was measured at distance and near fixation. The deviation was unaffected by use of the small hyperopic correction in glasses. There was no evidence of dissociated vertical divergence. Rotations revealed a small left inferior oblique overaction while all other versions were full. The Bielschowsky head tilt test was positive to left tilt. Fusion was demonstrated with the Worth 4-dot test at near. The diagnosis of left superior oblique palsy in association with an intermittent, non-accommodative esotropia was made.

**COMMENT:** Both patients presented with an intermittent horizontal deviation (and diplopia in the adult patient old enough to complain of this symptom). In the first patient, fusion could be maintained with an upright position but when reclining, the increased vertical deviation brought about by head tilt did not permit control of the intermittent exotropia and it became manifest with complaints of double vision. In the second patient, the esotropia could generally be controlled but did become noticeable when the superior
SUPERIOR OBLIQUE PALSY

oblique weakness caused a small vertical deviation and loss of horizontal fusion.

C. THIRD NERVE PALSY

(1) A 23-year-old female presented with horizontal diplopia and a vertical component. Three years previously, she had been involved in a motor vehicle accident and suffered a brain stem injury with a diagnosis of right third nerve palsy.

In primary position, 25 prism diopters of right exotropia was manifest with 6 prism diopters of right hypertropia that increased to 12 prism diopters in left gaze. Adduction, right eye, was moderately limited as was depression, more prominently in adduction. A mild right inferior oblique overaction was noted with a "V" pattern. The left pupil was normal while the right pupil had a sluggish light reaction. No right upper lid ptosis was noted. The Bielschowsky head tilt test was positive to the right. The diagnosis was a partial right third nerve palsy along with a right superior oblique palsy.

COMMENT: It is not unexpected to find a superior oblique palsy associated with a traumatic etiology of a third nerve palsy. The first thought concerning the hypertropia in the presence of an exotropia was that the third nerve palsy was responsible for both findings. However, the "V" pattern, more pronounced limitation of depression in adduction, inferior oblique overaction with an increased right hypertropia in left gaze and a positive head tilt test all suggest a coexisting fourth nerve palsy. Aberrant third nerve regeneration might have also accounted for the elevation of the right eye in adduction.

D. POST-RETINAL DETACHMENT SURGERY

(1) A 28-year-old female had a history of two previous retinal detachment operations with successful reattachment. She complained of vertical diplopia following the second detachment repair. Of interest was that vertical prisms were present in the patient's glasses prescribed prior to the first surgery.

At distance and near fixation, there were 16 prism diopters of left hypertropia and 4 prism diopters of exotropia. Mild left inferior oblique overaction was noted along with mild underaction in the field of the left superior oblique resulting in an increased left hypertropia in right gaze. The remainder of the versions were full. The Bielschowsky head tilt test was positive to the left confirming the diagnosis of left superior oblique palsy.

COMMENT: Diplopia presenting after retinal detachment surgery is most frequently due to restrictions caused by the implant material and/or scarring with adhesions. The finding of prism in the glasses points to a pre-existing strabismus. It is likely that prolonged patching associated with the surgery reduced the vertical fusional vergence which apparently controlled the deviation pre-operatively. The prismatic correction was no longer sufficient to eliminate diplopia. Acquired vertical diplopia in this patient was not directly related to mechanical factors in the orbit as is often the case after a detachment procedure.

E. POST-CATARACT SURGERY

A 58-year-old white male complained of diplopia following monocular cataract surgery. Vision in the operated eye was corrected to 20/20 with a contact lens. Interestingly, the patient gave a history of intermittent diplopia prior to cataract surgery. He could fuse in right gaze with a left head turn.

Muscle balance revealed 18 prism diopters of exotropia and four prism diopters of right hypertropia in primary gaze. Versions were full in all directions. The Bielschowsky head tilt test was positive to the right and four degrees of excyclotorsion was measured. The diagnosis of exotropia and right superior oblique palsy was made.

COMMENT: The fourth nerve paresis likely predated the cataract operation. Postoperatively, the small vertical deviation probably prevented control of the exotropia with resulting horizontal diplopia. In right gaze, away from oblique muscle action in adduction, fusion was possible. Exotropia in the postoperative cataract patient may be controlled if the deviation is not too large. In this patient, the exotropia probably could have been controlled except for the presence of a small hypertropia secondary to superior oblique palsy.

Discussion

The thread that holds these cases together is the presence of superior oblique palsy in patients presenting as a different motility disturbance (sixth nerve palsy, third nerve palsy, intermittent horizontal strabismus, post-retinal detachment surgery and post-cataract surgery). The diagnosis of fourth nerve paresis should be suspected in patients with blunt head trauma, horizontal strabismus, or diplopia brought out by head tilt or acquired vertical diplopia in individuals wearing long-standing prisms. In addition, patients we have seen with a large vertical phoria or phoria-tropia have all had compensated superior oblique palsy. It would seem that large, vertical fusional vergences can develop over time utilizing a preferred head position. One patient in a military hospital was found to have built up 30 prism diopters of vertical fusional vergence by working several hours daily on a major amblyoscope for many months (Jampolsky — personal communication).

Other unusual presentations of superior oblique palsy have been noted. Price and Pedezzioli reported two patients with deviations typical of fourth nerve paresis following retinal detachment surgery. Both had encircling bands and exoplants beneath the ipsilateral superior rectus. The possible etiology was an inadvertent superior oblique disinsertion during surgery or that the superior oblique tendon was picked up on the superior rectus muscle hook and anchored to the sclera anterior to the equator. This could displace the functional insertion of the superior oblique anterior to the equator leading to an effective paresis of this muscle.

Wolff noted a patient who developed exotropia, hypertropia and excyclotorsion (with a presumed diagnosis of superior oblique palsy), following a retinal detachment repair. During strabismus surgery, the superior rectus and superior oblique tendon were adherent to one another and to the encircling and radial exoplants that had been applied at the time of the detachment procedure. The adhesion
involved the anterior one-third of the superior rectus muscle and the superior oblique tendon, from the position at which it passed under the superior rectus to its insertion, causing the functional insertion to be anterior to the equator and resulting in the clinical picture of superior oblique palsy.

In contrast, our patient's superior oblique palsy pre-dated the detachment operation and was not caused by mechanical factors related to this surgery.

Two cases of trochlear nerve palsy associated with thyroid ophthalmopathy were described by Knapp. He thought the palsy probably pre-existed, and was not caused by Graves' disease. The paresis seemed to become symptomatic as a result of decreased fusional ability resulting from the thyroid eye disease.

Keane had seen two patients presenting with hypertropia and traumatic lid ecchymosis. They were originally thought to have orbital blowout fractures. Further examination revealed a fourth nerve palsy on the opposite side, as the cause for post-traumatic vertical diplopia.

Burger et al commented on a patient they had seen with myasthenia gravis presenting first as an isolated superior oblique palsy. They believed that the palsy was longstanding (a head tilt was noted in old photographs) and the onset of myasthenia resulted in a decrease in fusional control of the vertical deviation.

Two were associated with sixth nerve paralysis and did not become apparent until treatment reduced or eliminated the esodiversion. In one case, the fourth nerve palsy accompanied a third nerve paresis.

The presentation in two patients was heralded by the onset of intermittent horizontal diplopia. In another case, the superior oblique weakness pre-dated retinal detachment surgery and pre-operative fusional vergence was probably broken down by prolonged patching. The last patient had diplopia following cataract surgery caused by a pre-existing fourth nerve paresis.

Other unusual presentations of fourth nerve paresis are reviewed. The correct diagnosis will be made more often if superior oblique palsy is kept high in the differential diagnosis.

References


Summary

Seven patients are described with unusual presentations of superior oblique palsy.