Double Trouble- Double Elevator Palsy With Pseudoptosis A Case Report

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ABSTRACT
A monocular elevation deficiency is also known as Double elevator palsy. It suggests paralysis of both elevator muscles (the superior rectus and the inferior oblique) of same eye; resulting in clinical condition characterized by defective elevation in the entire range of upward gaze, (i.e. in primary gaze, in adduction and abduction) and a hypotropia in the primary gaze. It can be caused by paretic, restrictive or combined etiology. It may be either congenital or acquired. 50% presents with true ptosis due to associated hypotropia.

40% of the cases present with pseudoptosis. Pseudoptosis disappears when patient fixates with the paretic eye associated with hypertropia in uninvolved eye.

We present a case of 6 years old male patient having right eye congenital monocular elevation deficiency with pseudoptosis.

Keywords- Double elevator palsy, hypotropia, Pseudoptosis

Introduction
The term ‘Double elevator palsy’ currently known as Monocular elevation deficiency was first described by White in 1942 [1] and later by Dunlap.[2] Though attributed to paralysis of both elevators in some cases, superior rectus palsy can alone account for MED.[3] MED can also be caused by inferior rectus restriction and supranuclear etiologies.[4] It may be congenital or acquired. It is characterized by unilateral defective elevation of involved eye in both adduction and abduction also associated with hypotropia in the same eye. Patient may fixate with the hypotropic eye (Involved eye) and presents with hypertropia of normal eye which is greater due to secondary deviation. It is also associated with pseudoptosis which resolves after taking fixation. [5]

Case History
A 6 yrs old male presented in the ophthalmology Department in M.G.M Medical college & MYH hospital Indore, India with his parents with the complaints of drooping of the right eyelid and downward displacement of the right eye since birth. (Fig-1)
There was no associated history of trauma, pain, headache, fever and diplopia. His birth history was normal. He was currently meeting his developmental milestones. There was no history of any systemic diseases. There was no significant past, family or personal history. His general and systemic examinations were normal including neurological also. Clinically, he was well oriented to time place and person. On ophthalmic evaluation of right eye vision 6/18 with pinhole 6/12. Mild chin elevation was present. Extraocular muscles movement revealed a restriction of the movement of right eye in up gaze (supraduction, dextroelevation, levoelevation) (Fig-2) indicating towards elevator weakness (right superior rectus and right inferior oblique). All other ocular movements were normal. Bells phenomenon was weak. Jaw winking phenomenon was absent. On worth’s four-dot test-no diplopia or suppression noted. His visual fields were normal by confrontation test. FDT (Forced duction test) was negative on elevation. Direct cover test of left eye revealed right took fixation and there was improvement in right eye upper lid ptosis. (Fig-3) When right eye took fixation, left eye showed hypertropia. Hirschberg test revealed 30 degree hypotropia. On prism bar cover test there was 16 Dioptres deviation noted. Krimsky corneal reflex test showed central corneal reflex in squinting eye with 16 dioptres prism. There were no restrictions of the left eye movements. Anterior segment were normal with normal fundus evaluation. Cycloplegic refraction revealed 6/12 with -0.25 cylinder at 100 degree in right eye and 0.00 D in the left eye.

When the patient fixates with the nonparetic uninvolved eye (Left), the paretic eye (right) will take a hypotropic position and the upper lid may be remain ptotic. Fixation with the paretic eye (right) will cause a hypertropia of the nonparetic eye (left), and ptosis may disappear, due to some fascial attachment between superior rectus and levator palpebral superioris provided the levator palpebral is not involved unlike true ptosis. FDT is done to confirm any mechanical restriction of movement and was negative.

**Discussion**

In Monocular elevation deficiency, there is unilateral defective elevation in the entire range of upgaze. Patients prefer to keep their head tipped backward with chin up position to maintain binocular vision. Congenital elevator weakness may be caused by a long standing superior rectus palsy with later spread of comitance leading to involvement of field of action of inferior oblique.

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**Differential diagnosis of DEP include**

Blow-out fracture of the orbital floor with incarceration of inferior rectus muscle, congenital or acquired fibrosis of inferior rectus muscle, thyroid ophthalmopathy with inferior rectus myopathy, myasthenia gravis, Brown syndrome and 3rd nerve palsy.

Surgical treatment is generally based on findings of the Forced duction test (FDT). If FDT is positive, inferior rectus recession is recommended. In the absence of IR restriction, (forced duction test negative) surgical treatment comprises of the KNAPP PROCEDURE. In this procedure the entire tendon of both the medial and lateral rectus muscle is transferred to the side of superior muscle insertion. If vertical deviation in primary position is larger, then Inferior Rectus recession with Superior Rectus resection is done.

**Conclusion**

Thus proper selection of surgical technique gives ultimate good outcomes in such patients.
Fig no 2: Right eye elevation palsy showing restriction in supraduction, levoelevation & dextroelevation.

Fig no 3: Pseudoptosis is relieved after taking fixation with same eye and hypertropia in normal eye.

References


