Abducens–Oculomotor Synkinesis Following Acquired Sixth Nerve Palsy

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INTRODUCTION

Paradoxical patterns of pupillary, eyelid, and eye movement following third nerve paralysis have been referred to as aberrant regeneration of the third nerve, oculomotor misdirection, or acquired oculomotor synkinesis.1 Aberrant regeneration following paralysis of the oculomotor and abducens nerves has been reported.2 We describe a patient with abducens–oculomotor synkinesis following isolated sixth nerve palsy.

CASE REPORT

A 30-year-old man with no history of a preexisting eye movement disorder had had diplopia since an episode of brainstem hemorrhage from a ruptured arteriovenous malformation at the vertebrobasilar junction. Bilateral sixth and right seventh nerve palsies were noted immediately. There was 40 PD of esotropia in the primary position. No synkinesis was noted. A computed tomography scan showed pontine and intraventricular hemorrhage. An angiogram demonstrated minute arteriovenous malformations near the vertebrobasilar junction and the incidental finding of a left internal carotid artery aneurysm. A left frontotemporal craniotomy was performed to clip the aneurysm. After the operation, there was no change in esotropia and eye movements.

One year after the episode of brainstem hemorrhage, the ophthalmologic examination revealed right sixth nerve palsy, causing 40 PD of esotropia in the primary position. On attempted dextroversion, both eyes adducted simultaneously (Fig. 1). Vertical ocular movements were normal. Bell phenomenon was intact. With a synoptophore, he had fusion with stereopsis when the 40 PD of esotropia was corrected. Forced duction testing showed slight resistance to the abduction of the right eye. On forced generation testing, the right medial rectus muscle contracted on attempted dextroversion. Electromyography showed the firing of the right medial rectus muscle on the right and left gaze, and there was minimal contraction of the right lateral rectus muscle on the right gaze. Right seventh nerve palsy was persistent, whereas left sixth nerve palsy was resolved.

Full tendon transposition of the vertical rectus muscles to the insertion of the right lateral rectus muscle was performed, along with a 9-mm recession of the right medial rectus muscle on adjustable sutures. Postoperatively, the patient had 2 PD of esophoria in the primary position. On attempted right gaze, the right eye adducted. There was limitation of adduction of the right eye on attempted left gaze. There was no limitation of vertical movements.

Three years postoperatively, he had orthophoria in the primary position but still displayed paradoxical adduction of the right eye on attempted right gaze (Fig. 2).

DISCUSSION

Various patterns of synkinesis have been observed following both congenital and acquired third nerve palsies. Four hypotheses that have been proposed to explain oculomotor synkinesis are the misdirection of regenerating axons, ephaptic transmission (side-to-side interaxonal cross-stimulation),
central reorganization, and denervation supersensitivity.¹

Misdirection seems to be the most widely accepted mechanism that explains the synkinetic movements observed in acquired third nerve palsy. When misdirection occurs in peripheral nerves that innervate more than one muscle, the regenerating sprouts from axons that previously innervated one muscle group may ultimately innervate a different muscle group with a different function.³

For most congenital aberrant innervations, the two possible mechanisms are peripheral miswiring and brainstem miswiring.⁴ Oculomotor synkinesis in congenital third nerve palsies is believed to be due to a brainstem lesion more likely than a peripheral lesion. Although the oculomotor–abducens synkinesis, such as Duane syndrome, is the most common type of congenital ocular aberrant innervation,⁵ the synergistic innervation between the sixth and the third nerves is not common in an acquired group. One case of abduction defect associated with aberrant regeneration of the third nerve after intracranial aneurysm, suggesting co-contraction of the medial recti and lateral recti muscles on attempted abduction, has been reported.⁶ The same paradoxical movement was also noted in a 22-year-old man with traumatic ophthalmoplegia. Aberrant regeneration involving the abducens and the oculomotor nerves is believed to be the underlying mechanism.² These two cases showed that abducens–oculomotor synkinesis can be acquired following third nerve palsy. To the best of our knowledge, abducens–oculomotor synkinesis following acquired sixth nerve palsy without third nerve palsy has not been reported.

Our patient displayed paradoxical adduction of the right eye on attempted right gaze. Failure of relaxation of the right medial rectus muscle on attempted abduction demonstrated by electromyography suggested co-contraction of horizontal recti muscles as the origin of this paradoxical movement. Forceps testing and ocular electromyography demonstrated that limitation of abduction of the right eye resulted from sixth nerve paralysis and anomalous innervation of the antagonistic right medial rectus muscle. Weakening surgery on the medial rectus muscle did not eliminate the synergistic movement.

The hypothesis of misdirection seems to be the possible rationalization. It may be that some of the innervation intended for the lateral rectus muscle aberrantly reached the medial rectus muscle, producing adduction on attempted abduction. However, morphologic documentation of aberrant regenerating fibers from the oculomotor subnucleus is rare.⁵ Because ephaptic transmission is a transient phenomenon,⁷ it seems an unlikely explanation for our patient, who had had synergistic movement for 3 years. Denervation supersensitivity develops early and is frequently transient,¹ whereas the synkinesis of our patient developed later and persisted. The central reorganization hypothesis proposes that peripheral nerve injury induces retrograde changes in the nerve cell body and its connections that may result in synkinesis by unmasking previously encoded connections.¹ It is possible that brainstem injury in our case produced central reorganization between the abducens and oculomotor nuclei in the brainstem. Electromyography measures only end-organ activity, and this can be changed by either peripheral or central mechanisms. Electromyography alone cannot differentiate between these two mechanisms.
To our knowledge, the mechanism responsible for the synkinetic movement in this patient is unknown. Synkinetic movement may occur as the result of misdirection or of central synaptic alterations, or of a combined mechanism. Other mechanisms (ephaptic transmission or denervation supersensitivity) are unlikely.

REFERENCES