Anterior and Nasal Transposition of the Inferior Oblique Muscle for Iatrogenic Superior Oblique Palsy

The reflected tendon of the superior oblique (SO) muscle lies in close proximity to the medial horn of the levator palpebrae superioris aponeurosis in the superomedial aspect of the upper eyelid, placing it at risk for inadvertent damage during surgery in that area. However, although levator resection or excision procedures are commonly performed, this complication is rarely reported. Kushner and Jethani reported a series of 7 patients who sustained SO tendon injury during various types of eyelid procedures, which led to SO palsy in 4 cases and Brown syndrome in 3. We describe a case of iatrogenic SO palsy following congenital ptosis surgery that, because of a large torsional component, we elected to treat with anterior and nasal transposition of the ipsilateral inferior oblique (IO) muscle instead of other conventional procedures.

Report of a Case. An 11-year-old girl underwent left levator excision and fascia lata brow suspension for Marcus-Gunn jaw-winking ptosis. The SO tendon was inadvertently cut when the levator palpebrae superioris was severed above the Whitnall ligament, and repair was attempted during the procedure. She visited 1 week later with vertical and torsional diplopia associated with a right head tilt and face turn to the right side, and she was found to have signs consistent with left SO palsy. Strabismus surgery was performed 1 year after the procedure; preoperatively, she had a left hypertropia of 20 prism diopeters (PD), which increased to 25 PD on both right gaze and left tilt (Figure 1). This hypertropia increased significantly from upgaze (5 PD) to downgaze (25 PD), and there was a V-pattern with 8 PD of exotropia on upgaze and 8 PD of esotropia on downgaze. Motility examination showed SO underaction of −3 and IO overaction of +4 in the left eye. Testing of subjective torsion using double Maddox rod showed excyclotropia of 8° in primary position, which increased to 14° on downgaze. The left IO was transposed nasal to the inferior rectus muscle, with its posterior border reattached to the sclera 2 mm below the medial rectus insertion and the anterior border reattached 2 mm nasal to this point (Figure 2C). Postoperatively, the abnormal head posture was corrected. She was orthophoric in both primary position and downgaze, although there was a 6-PD left hypertropia on upgaze with limitation of elevation, more in adduction (Figure 2A and D). She was diplopia free except in upgaze, and the excyclotorsion was eliminated in primary position, with encycloria of 3° only in downgaze (Figure 2B). These results were stable at 11 months.

Comment. Anterior and nasal transposition of the IO is a relatively new procedure devised by Stager et al. It has been reported to be useful in patients with severe or recurrent SO palsies as well as those with missing SO tendons. The IO is transposed not only anteriorly as in standard anterior transposition but also nasal to the inferior rectus insertion. This converts the IO from an extorter and elevator in adduction to an intorter and antielevator in adduction. Although the effects are more pronounced in upgaze owing to neurological activation of the IO in attempted upgaze and inhibition in downgaze,
the procedure is effective in correcting overerelevation and underdepression in adduction as well as excyclotorsion. Our case had profound loss of SO function, and one surgical option was an ipsilateral IO anterior transposition combined with contralateral inferior rectus recession. However, the large amount of excyclotorsion, especially on downgaze, was unlikely to be eliminated even if the IO was placed adjacent or anterior to the inferior rectus insertion.6 Dealing with residual torsion would have been difficult as the SO tendon damage precluded SO transposition surgery. Although horizontal transposition of the vertical recti was an alternative, this technique effectively corrected both the vertical and torsional components in 1 step, with sparing of the ciliary circulation and a more predictable result.

Inez B. Wong, FRCSEd(Ophth)
Vincent Paris, MD
Harold K. Choi, MRCSEd(Ophth)
Sonal Farzavandi, FRCSEd

Author Affiliations: Yong Loo Lin School of Medicine, National University of Singapore (Dr Wong), Department of Ophthalmology, National University Hospital Singapore (Drs Wong, Choi, and Farzavandi), and Singapore National Eye Center (Dr Farzavandi), Singapore; and Department of Strabismology and Pediatric Ophthalmology, University of Liège, Liège, Belgium (Dr Paris).

Correspondence: Dr Wong, Department of Ophthalmology, National University Hospital, NUHS Tower Block Level 7, 1E Kent Ridge Rd, Singapore 119288 (inez_by_wong@nuhs.edu.sg).

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Primary Orbital Melanoma With Poliosis and a Palpable Mass

Poliosis, defined as the depigmentation of hair, has been linked with several inflammatory conditions such as blepharitis, herpes zoster, idiopathic uveitis, Vogt-Koyanagi-Harada syndrome, sarcoidosis, vitiligo, Marfan syndrome, and tuberous sclerosis.1 To our knowledge, we report the first case in the English-language ophthalmic literature of eyelash poliosis as one of the manifesting signs of primary orbital melanoma.

Report of a Case. A healthy 60-year-old white man had swelling above the left eyeball and whitening of the left upper eyelid for 3 months. Visual acuity was 20/20 OD and 20/20 OS. No afferent pupillary defect was detected. He had eyelash poliosis of the left upper eyelid with a mobile, nontender, firm mass in the left anterior orbit superonasally (Figure 1). There was no proptosis, and he had normal ocular surface examination, anterior segment examination, and fundus examination results. Examination results of his right eye were unremarkable. Magnetic resonance imaging with contrast revealed a well-circumscribed, diffusely enhancing mass in the superonasal region of the left anterior orbit (Figure 2). Excisional biopsy of the mass was achieved through an eyelid crease incision. A histopathologic report revealed solid sheets of spindle cell neoplasm. Melan-A immunohistochemical staining showed cytoplasmic immunoreactivity in the neoplastic cells. The histopathologic findings were consistent with the diagnosis of orbital melanoma. No inflammatory cells were seen in the mass.

An extensive systemic evaluation showed no evidence of distant metastasis or primary tumor elsewhere. The mass was diagnosed as primary orbital melanoma, although primary tumor elsewhere cannot be totally ex-