Acute Comitant Esotropia Following Penetrating Keratoplasty

Abbas Bagheri, MD; Farid Karimian, MD; Mohammad Abrishami, MD; Hamidreza Hasani, MD; Shahin Yazdani, MD

Shaheed Beheshti Medical University

Purpose: To report a case of acute comitant esotropia in a patient with bilateral keratoconus presenting after penetrating keratoplasty (PKP) in the fellow eye.

Case Report: A 17-year-old male patient with bilateral keratoconus underwent PKP in his right eye. He experienced diplopia after removal of the patch from the operated eye 12 hour postoperatively due to esotropia in his left (unoperated) eye. Diplopia was controlled using prism glasses and botulinum toxin A injections until PKP was performed in his left eye which resulted in restoration of stable fusion up to 3 years of follow up.

Conclusion: Acute comitant esotropia may occur after loss of fusion due to ocular patching in a vulnerable patient. In this patient fusion was preserved with non-surgical methods until it was restored by surgical means.

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Correspondence to: Abbas Bagheri, MD. Associate Professor of Ophthalmology; Ophthalmic Research Center, Labbafinejad Medical Center, Boostan 9 St., Pasdaran Ave., Tehran 16666, Iran; Tel: +98 21 22585952, Fax: +98 21 22590607, e-mail: abbasbagheri@yahoo.com

INTRODUCTION

Acute comitant esotropia is an alarming condition. The patient can remember its exact onset due to the abrupt diplopia. Although comitant strabismus often presents during infancy and childhood, it may occur suddenly in a person with previously normal vision.[1,2] The approach to a patient with acute strabismus should first start by differentiating incomitant from comitant deviations. The cause of acute incomitant esotropia is often lateral rectus paralysis and entails a poorer prognosis than that of the comitant type. Nevertheless, the underlying cause of the latter condition is not always benign in nature and certain neurologic conditions must be ruled out.[3-7]

Acute comitant esotropia not associated with neurological disorders may occur under three different circumstances:

1) Following unilateral patching or after unilateral or asymmetric loss of vision (Swan type). This type of acute comitant esotropia has been reported during patch therapy for amblyopia,[8] following chalazion excision[9] and after post-traumatic eyelid swelling.[10] Although most eyes with this type of deviation are hyperopic, a few cases have been reported in eyes without any refractive error.[1,2] It is believed that occluding one eye results in disruption of fusion. If compensatory mechanisms such as fusion range cannot overcome this disruption, the deviation becomes manifest. This type of deviation is temporary in nature and often eliminated by correcting the refractive error and surgical management, if needed.[1,2]

2) Severe systemic conditions or a major psychological stress may also lead to acute comitant esotropia (Franceschetti type). These cases usually have pre-existing asymptomatic esophoria and the deviation may occur intermittently at the onset before becoming constant shortly thereafter. Mild hyperopia exists in these cases. Although surgical management is most often needed to restore binocular conjugate state, the prognosis for achieving binocular vision is also good in this category.[11-17]

3) Acute comitant esotropia may occur in myopic eyes (Bielschowsky type). Most of these cases have mild to moderate myopia (less than 5 diopters) and the deviation usually occurs following shock or exhaustion. It is thought that myopia and accommodative convergence for focusing on near objects results in this type of deviation. This condition must be differentiated from esotropia resulting from high myopia due to medial rectus fibrosis and lateral rectus atrophy from mechanical pressure from adjacent bones in a large globe. The latter is characterized by restriction in eye movement and high myopia, usually over 10 diopters.[12-21]

Herein, we report a patient with bilateral keratoconus who presented with acute comitant esotropia in one eye following penetrating keratoplasty (PKP) in the fellow eye.

CASE REPORT

A 17-year-old male patient with bilateral keratoconus underwent PKP in his right eye due to low vision (counting fingers at 2 meters) uncorrectable with contact lenses. Uncorrected and best-corrected visual acuity in the left (unoperated) eye were 1/10 and 9/10, respectively. Immediately before PKP, the contact lens was removed from the left eye and the operated eye was patched for 12 hours postoperatively. The patient developed diplopia following removal of the patch the day after PKP (Fig. 1). External ocular examination revealed intermittent esotropia of 40 prism diopters (PD) for near and far in the left eye regardless of wearing a contact lens. The corneal graft was clear in the right eye and other ocular examinations were unremarkable. Brain CT Scan and Tensilon test were also normal.

Ground-in base-out prism of 20 PD was prescribed for both eyes which controlled the diplopia (Fig. 2). After five months the patient requested a more desirable treatment, meanwhile the esotropia had increased to 50 PD. He received a 10 unit injection of botulinum toxin type A (Dysport) in the medial rectus of the left eye. Four days after this injection he developed exotropia in the same eye (Fig. 3) which resolved after two months and the eyes remained orthophoric for 6.5 months. Thereafter, the diplopia recurred and examination revealed 30 PD esotropia in the left eye. He received a second injection of Dysport in the same muscle which controlled the deviation and diplopia. The patient was then recommended to undergo PKP in the left eye because he stated that whenever he delayed contact lens wear, he experienced transient diplopia.

The patient underwent PKP in his left eye two months after the second injection, while the eyes were orthophoric preoperatively. The eyes retained the orthophoric condition which remained stable for more than three years (Fig. 4). He achieved best spectacle-corrected visual acuity of 10/10 with OD: +1.5 -5.00@40° and OS: Plano -1.5@180° at final follow up. Titmus test revealed stereopsis of 800 seconds of arc and worth 4-dot test showed normal fusion for near and far.

DISCUSSION

Acute comitant esotropia is an unpleasant condition and may be associated with serious conditions such as brain tumors, Arnold-Chiari syndrome, hydrocephalus, thalamic lesions, myasthenia gravis and certain types of seizures.[1-7] The neurologic mechanisms for comitant esotropia include hydrocephalus, bilateral dysfunction of the sixth cranial nerves and damage to the vergence center in the brainstem. Nystagmus is the characteristic sign in these patients[3] which did not exist in our case.

Despite the presence of myopia due to keratoconus, we believe that our patient de-

veloped acute comitant esotropia through the Swan type mechanism, because he experienced sudden asymmetric loss of vision due to patching of the operated eye and contact lens discontinuation in the fellow eye which broke the probably labile fusion resulting in overt strabismus. The clue supporting this view is that final stereopsis was no more than 800 seconds of arc despite the long-term orthotropic status and good visual acuity in both eyes. Other possible factors which may have played a role include decompensated preexisting esophoria, postoperative anisometropia and aniseikonia hindering fusion as development of new axes of astigmatism, changes in the eye dominancy and finally the major psychological stress of surgery in a young subject.

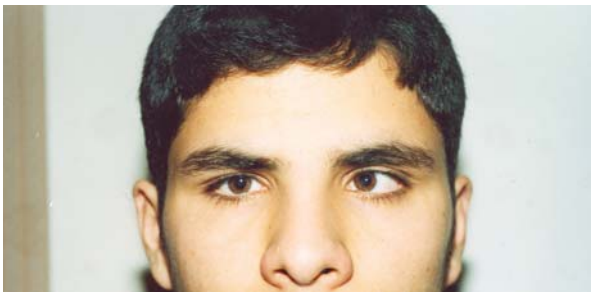

Figure 1 Acute esotropia of the left eye following penetrating keratoplasty in the right eye and patching for 12 hours.

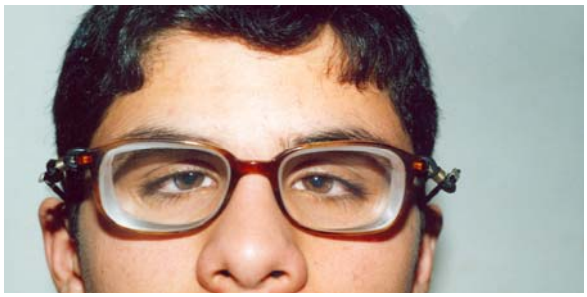

Figure 2 Diplopia was temporarily controlled using prism glasses.

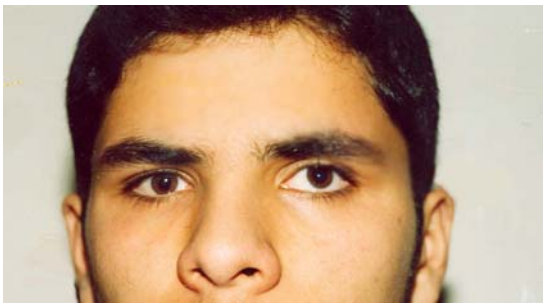

Figure 3 Exotropia of the left eye four days after injection of botulinum toxin in the left medial rectus.

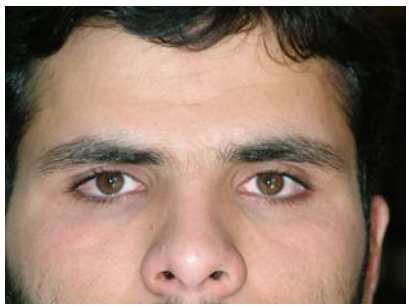

Figure 4 Final orthophoric state after performing penetrating keratoplasty in the left eye

According to most studies, in non-neurologic cases of acute comitant esotropia (Swan and Franceschetti types), binocular vision is so good that patients regain good stereoscopic vision.[8-17] The low final stereopsis in our patient may be due to the long-standing keratoconus and significant refractive errors for several years in both eyes which could have hindered good fusion and stereopsis.

Our success in restoring orthophoria without strabismus surgery may have been due to the prompt use of prisms for maintaining binocular vision which has been reported to have an important role on the success of subsequent treatments in acquired esotropias[22] and the use of botulinum toxin injections which has been established as an effective treatment for eliminating the need for strabismus surgery in acute acquired esotropias.[23] However, some studies have revealed no relationship between early initiation of treatment and final results in terms of fusion and stereoscopic vision.[24]

In conclusion, in patients with acute acquired esotropia, recognizing and eliminating the underlying cause is the basis of treatment, however if the condition persists, non-surgical

modalities such as prism glasses and botulinum toxin injections, may eliminate the need for surgery. In case of treatment failure, surgical intervention is usually successful in restoring fusion and stereopsis.

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