Acute Esotropia During an Adenoviral Keratoconjunctivitis

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Case report

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Abstract

Background

Acute acquired comitant esotropia (AACE) is a very infrequent subtype of esotropia that has been used to describe a sudden onset of concomitant esotropia with diplopia in adults. Despite the multiple reports carried out its cause is still controversial.

Case presentation:

We describe a case of a young patient who presented an AACE during the course of an adenoviral keratoconjunctivitis. The patient had no history of previous strabismus. As a result of surgery the patient recovered orthotropy.

Conclusion

AACE is a very infrequent strabismus that has been related with many etiologies and that it occurs during a viral infection is extremely rare. We report a case of an acute acquired comitant esotropia during an adenoviral keratoconjunctivitis that has not been published before.

Background

Acute acquired comitant esotropia (AACE) is a very infrequent subtype of esotropia that has been used to describe a sudden onset of concomitant esotropia with diplopia. AACE often occurs in older children and adults who have potential for normal binocular vision and highly favorable surgical outcomes.

Historically, AACE has been classified into 3 types which have been accepted as the pivotal work in this area: (I) the Swan type, which result from temporary or permanent monocular occlusion or visual loss (1), (II) the Franceschetti type, which is associated with physical or psychological stress with low hyperopia and (III) the Bielschowsky type related to different degrees of uncorrected myopia (2).

We describe the case of a young patient who presented an acute acquired comitant esotropia during the course of an adenoviral keratoconjunctivitis. Patient presented an intense conjunctival inflammatory reaction, subepithelial corneal infiltrates and eyelid swelling that prevented the complete opening of his right eye. During the process, the patient developed acute esotropia and he had no history of previous strabismus.

We consider that the cause of the strabismus in this case was the prolonged occlusion during several days due to edema and palpebral ptosis which interrupted the normal fusion and disrupted the corrective fusional movements, hence the criteria for Swan type.

Case Presentation

A 26-year-old male patient was seen by his general practitioner for a traumatic corneal ulcer of the right eye that was treated with topical antibiotics that resolved without any sequelae. One week later, he presented an epidemic bilateral adenoviral keratoconjunctivitis with an intense conjunctival inflammatory reaction and eyelid swelling that prevented the complete opening of the right eye. During the process, the patient was referred to the Ophthalmology Department in order to review a deviation of the right eye and diplopia. Regarding acute esotropia the patient referred that it started a few days after the initial ocular symptoms and he had no history of previous strabismus.

At ocular examination, his best corrected visual acuity was 20/30 in the right eye and 20/20 in the left eye. Slit lamp examination of right eye revealed adenoviral conjunctivitis with eyelid swelling that lead ptosis, conjunctival injection, follicular hypertrophy, conjunctival pseudomembranes, subepithelial infiltrates and preauricular lymphadenopathy. The involvement of the left eye was milder. He had comitant esotropia. Ductions and versions were full with no evidence of sixth cranial nerve palsy. Funduscopy examination revealed no abnormalities. We prescribed topical dexamethasone 0.1% and monthly follow up.

Two weeks later deviation could be measured, presenting comitant 40 Δ esotropia at distance and at near by alternating prism and cover test, with no A or V pattern. Cycloplegic refraction revealed hyperopia of + 2.50–0.50 x 180° right eye, + 1.75 left eye. Glasses did not reduce the strabismus angle. Stereoacuity assessed with TNO test at distance of 40 cm was unable to demonstrate any random-dot stereopsis. Patient was examined by a neurologist and underwent a blood test, magnetic resonance imaging, single fiber EMG and acetylcholine receptor antibody. All results were normal. At slit lamp examination, subepithelial corneal infiltrates persisted but were fewer (Fig. 1).

Six months later, his esodeviation continued and the strabismus was essentially unchanged. Subepithelial corneal infiltrates disappeared and surgery was performed. Medial rectus of right eye was recessed 4 mm and lateral rectus of right eye was resected 6 mm.

As a result of surgery the patient recovered orthotropy (Fig. 2). Ductions and versions were normal and he had no diplopia on Worth 4-Dot testing. Visual acuity was 20/20 in both eyes and stereoacuity with the TNO test showed a result of 480 s. Postoperative fusional amplitudes were measured with prisms. Fusional divergence amplitude for distance was 6 PD and for near was 10 PD. Fusional convergence amplitude for distance was 18 PD and for near was 25 PD.

Discussion

Historically, AACE has been classified into three types depending on clinical features and apparent etiology: a) Type I (Swan): refers to an esotropia following the interruption of fusion by monocular occlusion or vision loss (1); b) Type 2 (Burian-Francesschetti): esotropia associated with physical or

psychological stress and mild hyperopia with no other known cause; and c) Type 3 (Bielschowsky): esotropia that occurs in adolescents and adults, related to different degrees of uncorrected myopia.

Since then, findings from a number of reports suggested other etiologies of AACE such as accommodative spasm, decompensating esophoria, age-related distance esotropia, decompensated monofixation ocular syndrome, myasthenia gravis and neurological disorders (3).

It is important to be conscious about the infrequent, but possible, presence of central nervous system pathologies in these patients. Comitancy in AACE did not exclude the possibility of an underlying serious neurological disorder (4). In our patient neurological examination and neuroimagen findings were negative.

Epidemic keratoconjunctivitis (EKC) is a relatively common eye infection caused by human adenoviruses (5). Clinical signs of EKC include lid swelling, conjunctival injection, follicles, chemosis, pseudomembranes and subepithelial infiltrates. Most patients completely recover without any complications in a few weeks, however in some cases subepithelial infiltrates may persist for months (6).

Orbital involvement in viral infection is extremely rare. There are some reports of adenoviral conjunctivitis that caused orbital inflammation and caused extraocular motility restriction and diplopia (7). Our case did not present proptosis nor limited extraocular motility.

Our patient also developed subepithelial infiltrates but these did not cause a severe loss of vision; therefore, we consider that the cause of the strabismus was the prolonged occlusion of the eye due to edema and palpebral ptosis during several days, which disrupted the corrective fusional movements and lead to esotropia. Besides, the patient probably had to make an effort to use exclusively the eye of + 1.75 D of hyperopia without the aid of glasses, which probably caused abnormal convergence impulses that resulted in an acquired comitant esotropia. Therefore, considering all signs mentioned above this case could be classified as a Swan type (1).

Patient had no history of previous strabismus, however a latent deviation could manifest as an esotropia as a result of an interruption of the fusion mechanism that previously worked correctly and maintained the alignment of the eyes (8).

It is known that patients with esophoria initially use divergence fusional reserves in order to compensate it, but sometimes stress situations, physical illnesses, close working distance or even aging can reduce these reserves provoking an unmasked large angle AACE (9). In this case, fusional divergence amplitude at distance and at near were slightly lower than normal. So we cannot discard that the esotropia could have been triggered by a decompensated esophoria that had not been previously diagnosed (9).

In patients with AACE surgical treatments are important because spontaneous recovery is uncommon. The prognosis for binocular recovery is relatively good because given the late-onset of esotropia, and most patients have already established binocular single vision and stereopsis before the onset of

strabismus (10, 11). The surgery performed in our patient allowed us to achieve orthotropy and stereoacuity.

Conclusion

Adenoviral keratoconjunctivitis produced eyelid edema and mechanical ptosis that occluded the eye for several days, causing disruption of the fusion mechanisms in a patient with impaired fusional reserves.

Declarations

Ethics approval and consent to participate: ethical approval is not required at our institution for publishing a case report in a medical journal.

Consent for publication: written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editors-in-Chief of this journal

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Authors' contributions: JMR: corresponding author, major contributor in writing the manuscript. MLR: conceived the idea, provided the framework and edited the manuscript. ECM: reviewer and designer of manuscript. CPP: performed the extensive literature search and wrote the initial drafts. ENH: took the pictures and also searched the literature. MLC: reviewer and designer of manuscript.

All authors read and approved the final manuscript.

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References

1. Swan KC. Esotropia following occlusion. Arch Ophthal. 1947;37(4):444-51.
2. Bielschowsky A. Das Einwartsschielen der myopen. Ber Deutsche Ophth Gesell. 1922;43:245–8.
3. Chen J, Deng D, Sun Y, Shen T, Cao G, Yan J, et al. Acute Acquired Concomitant Esotropia: Clinical features, Classification, and Etiology. Medicine (Baltimore). 2015;94(51):e2273.
4. Williams AS, Hoyt CS. Acute comitant esotropia in children with brain tumors. Arch Ophthalmol. 1989;107(3):376-8.
5. Garcia-Zalisnak D, Rapuano C, Sheppard JD, Davis AR. Adenovirus Ocular Infections: Prevalence, Pathology, Pitfalls, and Practical Pointers. Eye Contact Lens. 2018;44 Suppl 1:S1-s7.

6. Gottsch JD. Surveillance and control of epidemic keratoconjunctivitis. Trans Am Ophthalmol Soc. 1996;94:539-87.
7. Kim SI, Lee KW. Orbital inflammation developing from epidemic keratoconjunctivitis in an adult. Case Rep Ophthalmol. 2013;4(2):93-8.
8. von Noorden GK. In: Burian HM, von Noorden GK. eds. Binocular vision and ocular motility. 2nd ed. St Louis: Mosby, 1980.
9. Godts D, Mathysen DG. Distance esotropia in the elderly. Br J Ophthalmol. 2013;97(11):1415-9.
10. Ali MH, Berry S, Qureshi A, Rattanalert N, Demer JL. Decompensated Esophoria as a Benign Cause of Acquired Esotropia. Am J Ophthalmol. 2018;194:95-100.
11. Kassem RR, Elhilali HM. Factors affecting sensory functions after successful postoperative ocular alignment of acquired esotropia. J aapos. 2006;10(2):112-6.

Figures

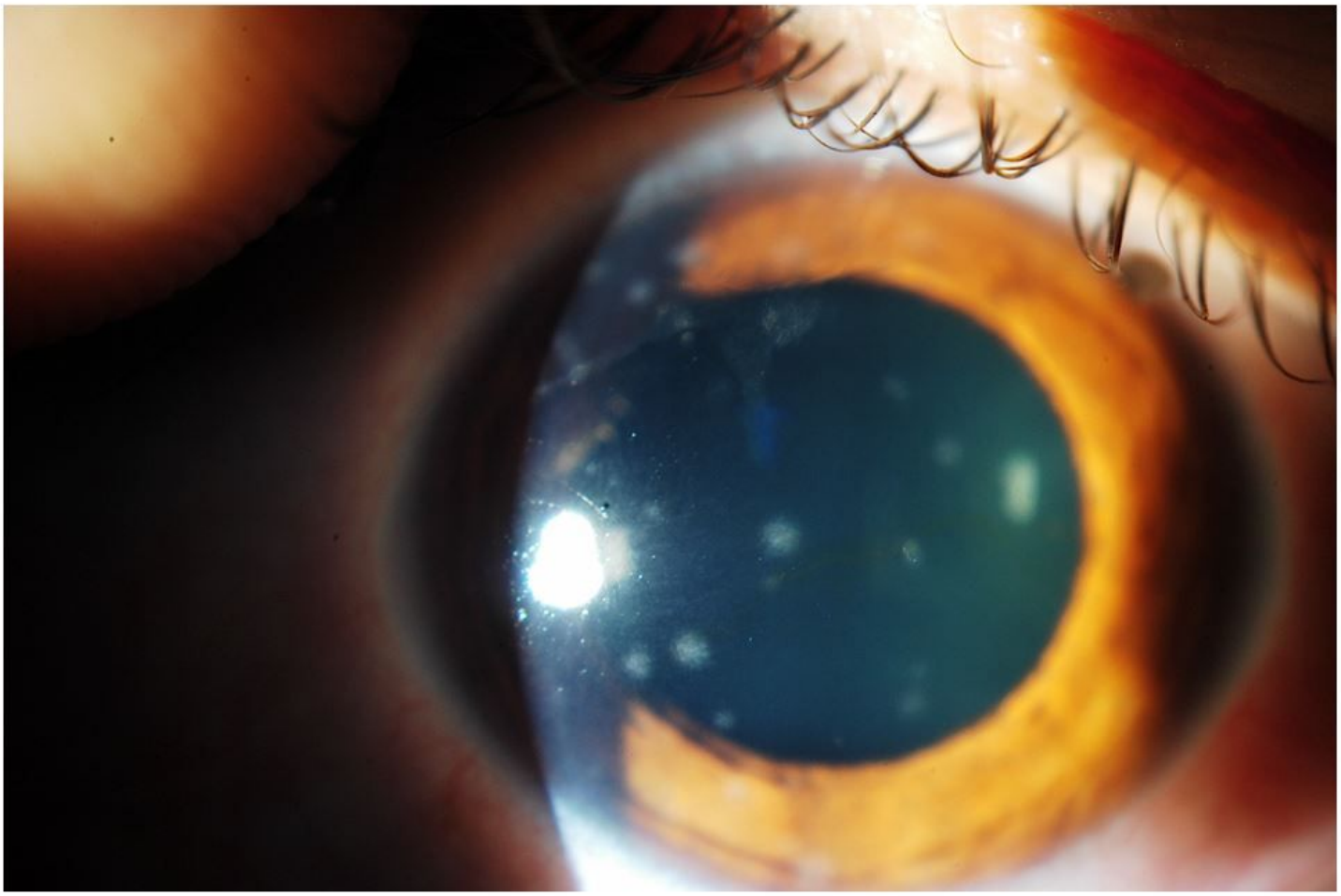

Figure 1

The photography shows the subepithelial infiltrates.

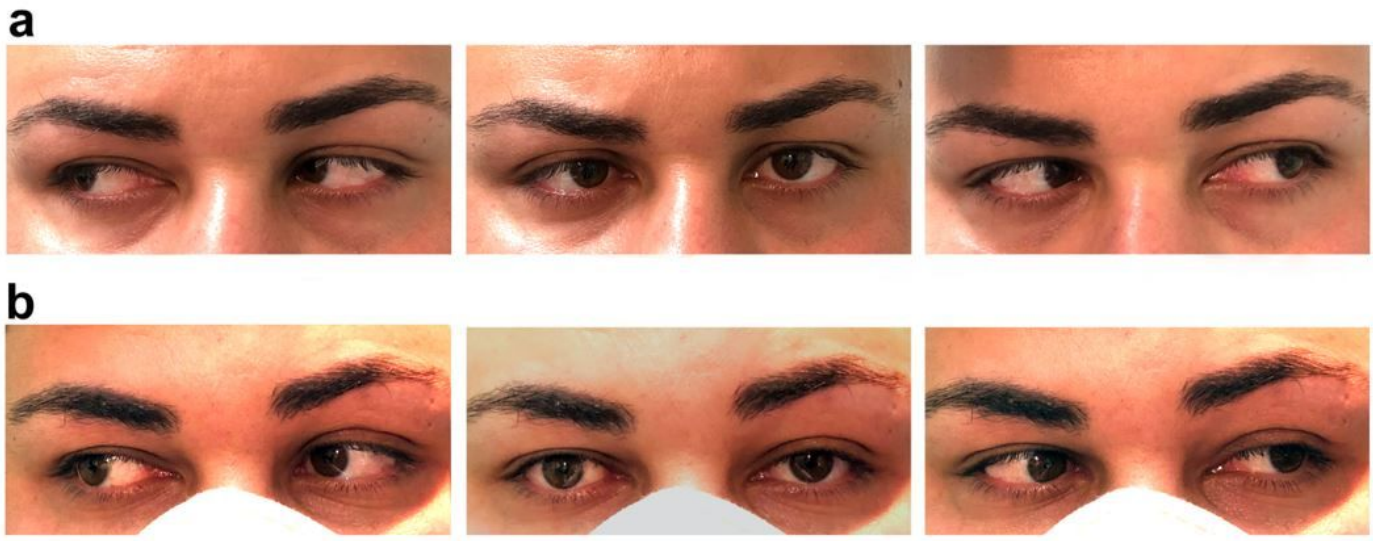

Figure 2

a) The three superior photographs show the preoperative gaze. b) The three bottom photographs show the result after alignment surgery.