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Unilateral upper eyelid eversion and kink associated with epidemic keratoconjunctivitis

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ABSTRACT

Purpose: Upper lid eversion in adults from non-cicatricial causes is rare. We report a case of upper eyelid eversion secondary to epidemic keratoconjunctivitis (EKC).

Observations: A 37 year-old female presented with unilateral upper lid eversion. Known for left upper lid ptosis repair in childhood, the patient presented with seven-day history of severe bilateral conjunctivitis and eversion of her left upper lid three days prior. On exam, she had follicular conjunctivitis, punctate epithelial keratopathy with subepithelial infiltrates and membranes bilaterally, with an everted upper lid tarsus, and swollen and ulcerated palpebral conjunctiva. She received topical and oral prednisone to quickly reduce the inflammation, as well as moxifloxacin drops and lubrication. When the swelling subsided, the tarsus adopted a kinked and everted configuration, and was managed successfully with reversion, pressure patching, shielding and close follow-up.

Conclusions and Importamce: This is the first reported case of upper lid eversion secondary to EKC, likely due to sudden marked inflammation and edema of the posterior lamella caused by the adenoviral infection. This case was successfully managed with conservative therapy.

1. Introduction

Ectropion, the outward rotation of the eyelid margin, is a common yet potentially complex problem encountered by ophthalmologists. It can lead to ocular irritation, conjunctival inflammation, and significant ocular morbidity. Etiologies are usually involutional or cicatricial, but can also be congenital.[1] Congenital upper eyelid eversion can occur in newborns, in which the entire tarsus is completely everted at birth. The cause remains unknown though certain conditions such as Down's syndrome and some anatomical abnormalities such as the absence of a tarsal plate or a short anterior lamella seem to predispose the eyelid to eversion.[2] People of African American heritage are also at higher risk of congenital ectropion.[2] In most congenital cases, the lid eversion is usually temporary and responds to conservative measures, which include pressure patching and taping.[1] In adults, upper lid ectropion is most usually cicatricial in etiology, commonly developing after surgery, trauma, and skin disease such as Herpes Zoster.[3,4] In all instances, the condition must be managed to revert the eyelid and minimize corneal exposure.

Another common ophthalmological condition is epidemic keratoconjunctivitis (EKC).[5] In the United States, there are over 6 million cases of viral conjunctivitis reported annually[6] and viral conjunctivitis is said to be the cause of one third of ophthalmic emergency room visits.[7] Adenovirus (serotypes 8, 11, 19 and 37) is the most common etiologic agent of EKC, which is often associated with discharge, lacrimation, membrane formation, and multiple subepithelial corneal infiltrates.[8] One study found that adenovirus was positive by PCR of conjunctival swabs in 78% of patients with clinical signs of acute bilateral keratoconjunctivitis.[9] Other common agents for viral conjunctivitis include Coxsackievirus A16 and A24, and Enterovirus 70 and 71[8].

EKC is highly contagious and often has a very characteristic presentation. One study investigated characteristics of EKC by recruiting 62 patients and studying their respective symptoms, which include hyperemia, lacrimation, burning, and foreign body sensation, as well as characteristic swelling and pruritis.[8] Table 1 lists common symptoms and signs of EKC. In addition, patients may also complain of extraocular symptoms such as headache and fever. The mean symptomatic duration was about twenty-five days, which did not significantly differ between patients who received topical antibiotics and those who did not. Another study reports an average duration of fourteen days, with some cases lasting up to five weeks.[10] The standard treatment is mainly supportive, such as lubrication and cold compresses, seeing as it commonly resolves

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Table 1
Common symptoms and signs of keratoconjunctivitis.[10,11,19]

Ocular symptoms and signs
•Hyperemia of bulbar/palpebral conjunctiva
•Ocular irritation
•Photophobia
•Tearing
•Foreign body sensation
•Chemosis
•Loss of visual acuity
•Eyelid edema
•Follicular reaction
•Clear or yellow discharge
•Epithelial keratitis
•Petechial hemorrhages in conjunctiva
•Subepithelial infiltrates
•Membranes and pseudomembranes
•Symblepharon

on its own.[11]

Another characteristic feature of EKC is its association with epidemics in work environments or physician offices. Diagnosis is usually based on history and a slit lamp examination. While viral conjunctivitis typically presents with its usual signs and symptoms as mentioned previously, rare associations with diseases such as Kawasaki disease and Stevens-Johnson syndrome have been reported.[12–14]

We report the clinical course and pathology of a patient with upper lid eversion secondary to adenoviral EKC.

2. Case report

A 37-year-old female presented to the emergency room complaining of bilateral red eyes, blurred vision, ocular pain, and unilateral left upper lid eversion. Her husband at home also had bilateral red eyes. Her past medical history was unremarkable other than a history of left upper lid ptosis repair in childhood and a hysterectomy a few years prior for a non-malignant etiology.

History revealed a seven-day history of severe bilateral conjunctivitis and eversion of her left upper lid three days prior (Fig. 1A). She had been to an outpatient clinic at the beginning of her symptoms, but her infection did not resolve with the oral amoxicillin and topical erythromycin that she had been prescribed.

Her main complaints were left-sided blurred vision, eye pain, and photophobia. On exam, her visual acuity was 20/20 OD and 20/30 OS. Her pupils were equal and reactive with no afferent pupillary defect, and measurement of her intraocular pressures was deferred due to the high suspicion of viral conjunctivitis and to avoid contamination. Her extraocular movements were normal and non-painful. On slit-lamp exam, she had lower lid follicles on her palpebral conjunctiva, punctate epithelial keratopathy with subepithelial infiltrates and mucosal membranes bilaterally. There was no symblepharon. She had clear, non-purulent discharge, and an everted, swollen and ulcerated left upper lid tarsus. Fig. 2 illustrates the left upper lid tarsal configuration.

She was diagnosed with adenoviral EKC, and a resultant upper lid ectropion. Due to the severity of the lid swelling, the tarsus was unable to be reverted and thus, conservative management was initiated. Given the appearance of membranes, her treatment included prednisolone drops QID. Moreover, given the extent of upper lid swelling and the concern for possible cicatricial changes developing, a short course of oral prednisone 60 mg (0.65 mg/kg of body weight) was initiated to reduce inflammation. Moxifloxacin drops were also prescribed to prevent superinfection and lubricating ointment and an eye shield were used to protect the cornea.

Within the week, the swelling had subsided enough for the tarsus to be manually reverted, but her tarsus had adopted a kinked configuration (Fig. 2) and spontaneously everted back into an ectropic position. This was managed conservatively with reversion, pressure patching and close follow up. She was seen the day following her initial presentation, q2 days for the following week, and then four weeks later. The duration of her course of oral prednisone was one week and her topical corticosteroids were tapered over one month. On her final follow up, having just stopped using the pressure patch, the patient had complete resolution of her lid ectropion (Fig. 1C). Her visual acuity was 20/20 and her subepithelial infiltrates had resolved.

Fig. 1. The patient on presentation (A), 2 days post start of treatment (B) and 40 days after presentation (C).

3. Discussion

This case represents the first reporting of upper eyelid eversion secondary to EKC, likely due to sudden marked inflammation and edema of the posterior lamella caused by the adenoviral infection. Lid ectropion is usually associated with aging, congenital malformations, or cicatricial changes after trauma or surgery.[1] Some conditions have also been reported to cause ectropion, such as ichthyoses, where characteristic skin hyper keratinization causes the anterior lamella to shorten, and the ectropion to form.[3] The anterior lamella may also be shortened from cicatricial changes seen in Herpes Zoster and may also lead to ectropion. Lid eversion after trauma, acid burns or surgery usually require surgical management in order to regain adequate lid apposition to the globe. Conservative measures involve repositioning the eyelid with a pressure bandage or adhesive tapes; however these are often not sufficient,[2] as the lid tends to revert back to its everted position. This case

Fig. 2. Representation of the left upper lid tarsal configuration.

demonstrates that lid eversion may occur after local inflammation and can be managed conservatively with favorable outcomes. Ectropion has been reported after recurrent chlamydial infection or years after an initial infectious conjunctivitis that left conjunctival scarring,[15] but to our knowledge, there are no publications reporting acute eyelid eversion following EKC.

Ectropion must be managed due to the potential for exposure of cornea and conjunctiva. The exposure may render the eye susceptible to complications such as ulceration or infection, which can lead to vision loss.[1] The etiology of the ectropion usually guides treatment. In our patient, her ectropion was secondary to acute inflammatory changes, which suggested that conservative measures could be attempted before surgical repair.

Corticosteroids are powerful agents that are often used for the treatment of ocular surface inflammation. Indications for the use of topical corticosteroids in patients with EKC are generally thought to be the presence of membranes or pseudo-membranes as well as the appearance of symptomatic subepithelial infiltrates.[16] While systemic corticosteroids may be used in systemic conditions that lead to severe ocular surface inflammation such as ocular cicatricial pemphigoid[17] and Stevens-Johnson syndrome,[18] they are generally not used in the treatment of EKC. However, given our patient's presentation with severe ocular surface inflammation with cicatrizing changes that were inducing exposure, it was felt that the risks of this exposure outweighed the risks of a short-term course of oral prednisone. The goal was to induce a rapid decrease of inflammation to allow for conservative management of the lid eversion.

The effect of the patient's previous ptosis surgery is unclear, but due to the infrequency of this presentation after EKC, as well as the same side having been surgically treated, we suspect a possible correlation. During slit lamp examination, there were no obvious signs of conjunctival scarring or lid crease scar, making it challenging to ascertain the exact approach used to repair her ptosis in the past. However, given that the patient did not elevate her brow to open her eye, frontalis suspension was less likely the procedure performed for her repair. A posterior approach using conjunctivomullerectomy is also unlikely as the recessed palpebral conjunctiva could potentially be protective against lid eversion. Similarly, a tarsoconjunctivomuellerectomy may also be protective due to shortening of the posterior lamella, although resection of tarsal tissue could contribute to instability and the possibility of kinking and eversion. Finally, an anterior approach to the ptosis repair with a levator aponeurosis resection seems to most compatible with our patient's condition. Shortening of the aponeurosis, either by resection or plication, would create a relative redundancy of palpebral conjunctiva, predisposing to expansion secondary to edema from EKC and subsequent eversion. Moreover, the reattachment of the levator aponeurosis to the anterior surface of the tarsus may act as a fulcrum, also predisposing to kinking and eversion. As such, although it is difficult to determine the precise method of ptosis repair performed in childhood due to the absence of evident scarring, and anterior approach using a levator aponeurosis resection appears to be the most likely suspect.

4. Conclusion

Upper lid ectropion in adults is most commonly cicatricial in etiology. This case represents the first reporting of upper lid eversion associated with EKC. The acute inflammatory etiology of the ectropion allowed for conservative management to be an appropriate initial approach and led to a favorable outcome.

Patient consent

Consent was obtained from the patient to present her photos for educational and publication purposes.

Disclosure

None of the authors have any financial disclosure or conflicts of interest.

Declaration of competing interest

The authors declare that they have no known competing interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

1. Vallabhanath P, Carter SR. Ectropion and entropion. *Curr Opin Ophthalmol*. 2000;11(5):345–351.
2. Watts MT, Dapling RB. Congenital eversion of the upper eyelid: a case report. *Ophthalmic Plast Reconstr Surg*. 1995;11(4):293–295.
3. Ozgur OR, Akcay L, Tutas N, Ozkurt Y. Cicatricial upper and lower eyelid ectropion in an ichthyosis patient. Surgical correction. *J Dermatol Case Rep*. 2011;5(2):27–29.
4. Elshtewi M, Archer DB. Congenital ichthyosis in a Libyan child with ophthalmic manifestations. *Int Ophthalmol*. 1991;15(5):343–345.
5. Varu DM, Rhee MK, Akpek EK, et al. Conjunctivitis Preferred Practice Pattern®. *Ophthalmology*. 2019;126(1):94–P169.
6. Azari AA, Barney NP. Conjunctivitis: a systematic review of diagnosis and treatment. *Jama*. 2013;310(16):1721–1729.
7. Channa R, Zafar SN, Canner JK, Haring RS, Schneider EB, Friedman DS. Epidemiology of eye-related emergency department visits. *JAMA ophthalmology*. 2016;134(3):312–319.
8. Akcay E, Carhan A, Hondur G, et al. Molecular identification of viral agents associated with acute conjunctivitis: a prospective controlled study. *Braz J Infect Dis : Off. Publ. Braz.Soc. Infect. Dis.* 2017;21(4):391–395.
9. Lee CS, Lee AY, Akileswaran L, et al. Determinants of outcomes of adenoviral keratoconjunctivitis. *Ophthalmology*. 2018;125(9):1344–1353.
10. Molner Jgp LE. Epidemic keratoconjunctivitis. *Am J Nurs*. 1943;43:825–827.
11. Bawazeer A. *Epidemic keratoconjunctivitis*. 2013.
12. Sharma N, Dada T, Dada VK, Vajpayee RB. Acute hemorrhagic keratoconjunctivitis after LASIK. *J Cataract Refract Surg*. 2001;27(3):344–345.
13. Laibson PR, Green WR. Conjunctival membranes in epidemic keratoconjunctivitis. *Arch. Ophthalmol.(Chicago, Ill)*. 1960;83(1):100–102.

14. Hodge WG, Discepola MJ, Deschenes J. Adenoviral keratoconjunctivitis precipitating Stevens-Johnson syndrome. *Can. J. Ophthalmol.Journal canadien d'ophtalmologie*. 1994;29(4):198–200.
15. Trachoma Chidambaram JD LT. A tale of two diseases. *Br J Ophthalmol*. 2005;89.
16. Hood CT. The Wills eye manual: office and emergency room diagnosis and treatment of eye disease. *Br. J. Ophthalmol.* 2009;93:1127–1128.
17. Neff AG, Turner M, Mutasim DF. Treatment strategies in mucous membrane pemphigoid. *Therapeut Clin Risk Manag*. 2008;4(3):617–626.
18. Araki Y, Sotozono C, Inatomi T, et al. Successful treatment of Stevens-Johnson syndrome with steroid pulse therapy at disease onset. *Am J Ophthalmol*. 2009;147 (6):1004–1011, 1011.e1001.
19. Maggio MC, Cimaz R, Alaimo A, Comparato C, Di Lisi D, Corsello G. Kawasaki disease triggered by parvovirus infection: an atypical case report of two siblings. *J Med Case Rep*. 2019;13(1):104.