Orbital Inflammation Developing from Epidemic Keratoconjunctivitis in an Adult

Sung In Kim  Kyeong Wook Lee

Department of Ophthalmology, Saevit Eye Hospital, Goyang-si, Republic of Korea

Key Words
Epidemic keratoconjunctivitis · Orbital inflammation · Orbital cellulitis · Idiopathic orbital inflammation

Abstract

Introduction: We report a rare case of epidemic keratoconjunctivitis (EKC) that developed into an orbital inflammation in an adult. Case Presentation: A 67-year-old Korean man, who had been diagnosed with EKC and treated for conjunctival injection and chemosis in the right eye for 4 days, was referred to Oculoplastics as orbital cellulitis was suspected. At the point of referral, clinical features such as decreased visual acuity, severe eyelid swelling, chemosis, follicles, corneal edema, limitations in lateral eye movement, and diplopia were observed in the right eye. Orbital cellulitis was suspected according to orbital computed tomography scan images, but there was no response to systemic antibiotics. Systemic steroid was administered instead, and then his symptoms and signs started to improve. The final diagnosis of this patient was orbital inflammation related to EKC based on the facts that there was no response to antibiotics, that he presented with contralateral symptoms and signs, that pseudomembrane formation occurred in both eyes, and that the symptoms resolved completely after 2 weeks. Conclusion: Clinicians need to consider the possibility of orbital inflammation developing from EKC, even in an adult patient, and treat the patient properly if the EKC symptoms and signs, such as conjunctival injection and follicles, are accompanied with symptoms and signs similar to orbital cellulitis.
Introduction

Symptoms of orbital inflammation include a variety of pain, periorbital swelling, visual disturbance, limitation of eyeball movement, and proptosis [1–5]. Causes of orbital inflammation may be related to infection or immune reaction. Infectious orbital inflammation may be caused by bacteria, fungi, parasites, and viruses. For immunologic orbital inflammation, the most common cause is idiopathic orbital inflammation, but Graves’ orbitopathy was found to be a common cause as well in systemic diseases [4, 5]. There are also very few cases of orbital inflammation associated with viral infection such as herpes zoster virus, herpes simplex virus, and influenza A virus [6–8]

An extensive review of the literature revealed a few cases of orbital inflammation developing from adenovirus conjunctivitis in children, yet no such case in an adult has been reported [9–11]. Therefore, here we report a case of orbital inflammation developing from epidemic keratoconjunctivitis (EKC) in a 67-year-old man.

Case Presentation

A 67-year-old man was referred to Oculoplastics after suffering from conjunctival injection and chemosis in the right eye for 4 days. Previously being diagnosed with EKC by another ophthalmologist, the patient had been treated with levofloxacin eye drops (Cravit®, Santen Pharmaceutical Co. Ltd., Osaka, Japan) and fluorometholone 0.1% eye drops (Flumetholon 0.1®, Taejoon Pharm Co. Ltd., Seoul, Korea). However, his symptoms were worsening even on medication, so orbital cellulitis was suspected and he was referred to our clinic. The patient had an ocular history of cataract operation – phacoemulsification and posterior chamber intraocular lens implantation in both eyes 9 months ago. When he had a check-up 1 month ago, his corrected visual acuities were 20/20 in both eyes. He had a medical history of diabetes, but there was no other clinical evidence of any other systemic disease or a trauma.

At the point of referral, the patient’s corrected visual acuities were 20/50 in the right eye and 20/20 in the left eye. His right eye was almost closed due to severe erythematous lid swelling, and he had marked conjunctival injection and chemosis. He complained of limited extraocular motility in horizontal gaze and diplopia in lateral gaze in the right eye. There was no relative afferent pupillary defect in the right eye. Intraocular pressure (IOP) was 22 mm Hg in the right eye and 9 mm Hg in the left eye by Goldmann tonometry. Slit lamp examination of the right eye showed diffuse bullous conjunctival chemosis, hyperemia, corneal edema, and mild anterior chamber reaction. Examination of the left eye was unremarkable (fig. 1). A non-contrast orbital computed tomography (CT) scan displayed right periorbital infiltration and swelling, thickening of the right lateral rectus muscle, and mucoperiosteal soft tissue attenuation in the left maxillary sinus, leading to a diagnosis of right orbital cellulitis (fig. 2). The patient was given intravenous cefmetazole (Cefmetazole®, SCD Pharm Co. Ltd., Seoul, Korea) 500 mg twice a day; oral amoxicillin 500 mg/clavulanate 125 mg (Augmentin®, Ilsung Pharmaceuticals Co. Ltd., Seoul, Korea) 3 times a day; levofloxacin (Cravit) eye drops, and fluorometholone 0.1% (Flumetholon 0.1) eye drops. His body temperature was 36.6°C, i.e. he was afebrile.

Five days after the onset of symptoms, and 1 day after the systemic antibiotic treatment, anterior segment conditions and extraocular motility in the right eye did not improve. IOP increased to 32 mm Hg in the right eye, but it was normalized after using a topical IOP-lowering agent. Six days after the onset, and 2 days after the treatment with systemic
antibiotics, chemosis and conjunctival injection were seen in the contralateral, left eye, while symptoms in the right eye did not improve.

Three days after the treatment with systemic antibiotics, there was no improvement in the right eye. We started to consider the possibility of idiopathic orbital inflammation. As infection could not be excluded completely, the patient was given 1 dose of intravenous methylprednisolone 250 mg (solu-Medrol®, Pharmacia and Upjohn, USA) per day with systemic antibiotics. Eight days after the onset, and 1 day after the treatment with systemic steroid, there was improvement in lid edema, conjunctival injection, and chemosis in the right eye. Yet, no changes were seen in the left eye.

Systemic antibiotics were discontinued because the possibility of an infection now seemed low. The patient continued on intravenous methylprednisolone 250 mg (solu-Medrol) per day and started on 20 mg oral prednisolone (Nisolone®, Kukje Pharm, Korea) twice a day. Nine days after the onset, 2 days after the treatment with systemic steroid, there was significant improvement in the lid swelling of the right eye, and conjunctival injection and chemosis in both eyes. His corrected visual acuities went up to 20/25 in the right eye and pseudomembranes were seen in both eyes. Oral prednisolone was continuously tapered by 10 mg per 2 days.

Two weeks after the onset, lid swelling of the right eye, conjunctival injection, chemosis, and corneal edema in both eyes were all gone. The patient's corrected visual acuities recovered to 20/20 in the right eye. The patient's limited extraocular motility in the right eye and diplopia also resolved (fig. 3).

Discussion

EKC is a common eye infection that is caused by adenovirus types 8 and 19. Patients chiefly complain of tearing, foreign body sensation, photophobia, and blurred vision. Slit lamp microscopy reveals chemosis, conjunctival injection, follicles, lid swelling, and pseudomembranes may be seen in acute stages [12, 13]. EKC usually occurs in both eyes. However, usually milder manifestations appear in the other eye within 2–7 days after one eye is infected. It tends to resolve in about 14 days in the first eye, and 18–21 days in the second eye [12, 13]. The severity of EKC ranges from subclinical conjunctivitis to severe diseases with bacterial superinfection. Lid swelling and associated inflammatory ptosis are usually seen only in the primarily affected eye [14, 15].

Generally, when a patient presents to an ophthalmologist with complaints of severe painful lid swelling and conjunctival chemosis, a clinician considers all diseases that induce periorbital inflammation and swelling. Orbital CT scan can help to diagnose and differentiate the diseases. The present patient was suspected to have acute orbital cellulitis initially, so an orbital CT scan was performed and systemic antibiotics were administered. Complete blood count and other blood tests were not conducted in this case, because the possibility of an acute bacterial or fungal infection seemed low since the patient was afebrile, and we could assume that there was no acute bacterial or fungal inflammatory reaction.

However, the patient's symptoms and signs aggravated despite the systemic antibiotic treatment. Next, he was given systemic steroid, as idiopathic orbital inflammation syndrome was suspected. The symptoms and signs started to improve on the next day, and systemic antibiotics were discontinued.

Orbital involvement in viral infection is extremely rare, and only a few cases of ophthalmic zoster that involved vitreous and retina have been reported [6–8]. A few cases of children with adenoviral conjunctivitis that mimics preseptal and orbital cellulitis were
reported by Ruttum and Ogawa [11] in 1996. In Korea, Kim et al. [13] reported 507 EKC cases out of their 9,147 patients from 1967 to 1968; conjunctival injection, chemosis, and follicles were present in their cases, but there was no orbital involvement.

The clinical manifestations of orbital inflammation are characterized by lid swelling and redness, chemosis, abrupt onset of pain, limited extraocular motility, proptosis, and decreased vision. The causes of decreased vision include compression of optic nerves resulting from increased intraocular pressure, anoxia of vessels, and degenerative changes caused by optic neuritis. If the inflammation gets worse and starts to compress the central retinal artery or arterioles, papilledema or optic neuritis may occur and lead to progressive optic atrophy, which can cause significant damages to the patient’s vision. Our patient also presented with decreased vision and limited extraocular motility in the right eye. Thus, it is very important for clinicians to be precise in the diagnosis and treatment of orbital inflammation [16].

Corticosteroids are used primarily to treat orbital inflammation [17]. They work by reducing inflammatory cytokines in organs and by inhibiting capillary permeability, thus having anti-inflammatory effects [18, 19]. In general, any orbital inflammation may respond well to intravenous or oral steroids including orbital cellulitis, since the orbit is a closed compartment and steroids may decrease intraorbital pressure.

In the present case, based on the facts that there was no response to antibiotics, that he newly developed chemosis and conjunctival injection in the contralateral eye 5 days after the first eye was affected, that pseudomembrane formation occurred in both eyes 1 week after manifestation, and that the symptoms completely resolved after 2 weeks, we concluded that it is more probable that the patient had orbital inflammation developing from EKC than acute orbital cellulitis or idiopathic orbital inflammatory syndrome.

Clinical symptoms and signs provide the majority of clues to make a diagnosis of EKC, but laboratory tests such as antigen detection, nucleic acid detection, electron microscopy, and cell culture can confirm the diagnosis. Virus antigen detection and nucleic acid detection tests are widely used in clinics because they are rapid and easy to perform [13]. However, this case was diagnosed from clinical symptoms and signs and was not confirmed by laboratory tests.

In conclusion, clinicians need to consider the possibility of orbital inflammation developing from EKC, even in an adult patient, and treat the patient properly if the EKC symptoms and signs, e.g. conjunctival injection, are accompanied with erythematous eyelid swelling and limitation of eye movement.

References

1. Reddy SC, Sharma HS, Mazidah AS, et al: Orbital abscess due to acute ethmoiditis in a neonate. Int J Pediatr Otorhinolaryngol 1999;49:81–86.
2. Choe CH, Eckstein LA, Vageli MR: Orbital inflammation after dental procedures. Ophthal Plast Reconstr Surg 2012;28:e113–e115.
3. Gordon LK: Orbital inflammatory disease: a diagnostic and therapeutic challenge. Eye (Lond) 2006;20:1196–1206.
4. Gunalp I, Gunduz K, Yazar Z: Idiopathic orbital inflammatory disease. Acta Ophthalmol Scand 1996;74:191–193.
5. Shovlin JP: Orbital infections and inflammations. Curr Opin Ophthalmol 1998;9:41–48.
6. Vardy SJ, Rose GE: Orbital disease in herpes zoster ophthalmicus. Eye 1994;8:577–579.
7. Tornerup NR, Fomsgaard A, Nielsen NV: HSV-1-induced acute retinal necrosis syndrome presenting with severe inflammatory orbitopathy, proptosis, and optic nerve involvement. Ophthalmology 2000;107:397–401.
8. Harley MJ, Guerier TH: Orbital cellulitis related to an influenza A virus epidemic. Br Med J 1978;2:13–14.
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Fig. 1. Right periorbital swelling and ptosis, and severe chemosis were observed in the initial presentation.
Fig. 2. Non-contrast orbital CT scan revealed periorbital tissue infiltration and right orbital edema (arrows). a Axial view shows muscular thickening of the right lateral rectus muscle. b Coronal view shows right periorbital swelling and mucoperiosteal soft tissue attenuation in the left maxillary sinus.

Fig. 3. Right lid swelling, chemosis, corneal edema, and orbital inflammation resolved 2 weeks after the onset.