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Chlamydia trachomatis presenting as preseptal cellulitis in a 3-year-old girl

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Inclusion conjunctivitis usually presents with lid swelling, red eye, foreign body sensation, and a mucopurulent discharge in association with a follicular reaction involving the palpebral and bulbar conjunctiva and semilunar fold. Similar to epidemic keratoconjunctivitis, it may present with preauricular lymphadenopathy, superficial punctate keratitis, and subepithelial corneal infiltrates, which tend to be more peripheral. We present the case of preseptal cellulitis in a 3-year-old child, caused by nonconsensual sexual contact with chlamydia trachomatis.

Case Report

A healthy 3-year-old girl presented emergently at BronxCare Health System with worsening left periorbital swelling, redness, and mucopurulent discharge, which had been worsening for 3 days. Review of systems was negative. The child lived at home with her mother, father, and 2 siblings and attended daycare. Visual acuity was not obtainable because of poor cooperation. External ophthalmologic examination revealed a normal right eye; the left eye had marked edema and erythema of the eyelids and a follicular reaction on the inferior tarsal conjunctiva. Because complete ophthalmologic examination was precluded by poor patient cooperation, computed tomography of the face and orbits was obtained to rule out orbital involvement. It showed preseptal swelling on the left, without orbital or sinus inflammation.

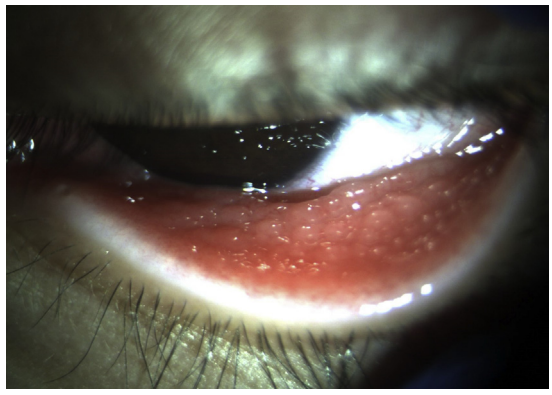

FIG 1. External photograph of the left lower palpebral conjunctiva showing follicular inflammation.

A diagnosis of bacterial preseptal cellulitis in association with epidemic keratoconjunctivitis (EKC) was made, and the patient was treated with systemic clindamycin. The following day, the patient was more cooperative, allowing for more complete examination. Uncorrected visual acuity measured 20/20 in each eye, and the eyelid and conjunctival appearance remained unchanged. The rest of the eye examination was unremarkable. Three days later, the child was discharged on a 10-day course of oral cephalexin and topical erythromycin. Two weeks later, the follicular conjunctivitis (Figure 1), lid edema, and erythema were still present, but two midperipheral subepithelial corneal infiltrates were newly noted. As a result, swabs of the conjunctiva were taken and were positive for *Chlamydia trachomatis*, serotype E, as identified by immunofluorescence. Urine C. trachomatis ribonucleic acid transcription–mediated amplification testing was also positive. Testing was negative for *Neisseria gonorrhoeae*. Child protection services was notified, and the child was readmitted to the hospital for protection and treatment with oral azithromycin. Her ocular symptoms completely resolved within 1 week.

Discussion

Preseptal cellulitis is common in the pediatric population, and sinus disease has been found to be the most common predisposing factor.[1] *Staphylococcus aureus* and *Streptococcus sp.* are the most common bacterial pathogens causing preseptal cellulitis.[1,2] Additionally, periorbital changes have been noted to occur with EKC[3] and may be misdiagnosed as preseptal cellulitis, which we believe also occurred in our patient with chlamydial conjunctivitis. To our knowledge,

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there is only one other case of *C. trachomatis* presenting as conjunctivitis and preseptal cellulitis.[4] In that case, as in ours, it cannot be definitively stated whether the periorbital changes were reactive[3] to the conjunctivitis or were caused by a secondary bacterial soft-tissue infection. However, the persistence of the periorbital symptoms after administration of non-chlamydia-covering antibiotics, and their complete resolution after azithromycin, suggests that chlamydia was in fact causative of the cellulitis.

Chronic conjunctivitis in children is frequently misdiagnosed as viral in etiology.[5] The ophthalmologist and the pediatrician should maintain a high index of suspicion of chlamydial infection in cases of chronic conjunctivitis, which, if confirmed in a child incapable of consenting to sexual activity, raises the suspicion of child abuse.

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COL4A1 mutations in two infants with congenital cataracts and porencephaly: an ophthalmologic perspective

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***COL4A1* mutations present with a spectrum of clinical phenotypes often involving the cerebrovascular and ophthalmic systems. We report 2 cases of *COL4A1* mutations that presented with congenital cataracts and porencephaly. Both patients had posterior cortical cataracts and radiographically defined bilateral posterior lenticonus. Considering the long-term clinical implications of these mutations, posterior cortical cataracts, bilateral posterior lenticonus, and porencephaly should raise clinical suspicion for *COL4A1* mutations.**

Type IV collagen is a physiologically important component of basement membranes in blood vessels of the brain, eye, kidneys, and muscle.[1] The α1 chain of the *COL4A1* gene contains many highly conserved Gly residue repeats (Gly-X-Y) that are essential to the triple helix structure. Any mutation that leads to the replacement of a Gly residue with a bulkier residue weakens the structural integrity of the triple helix and alters the function of the entire collagen strand.[2] *COL4A1* mutations are associated with a wide spectrum of clinical phenotypes, including intracerebral hemorrhage, porencephaly, cerebral calcification, and hereditary angiopathy, nephropathy, aneurysms, muscle cramping syndrome.[2-5] Congenital cataracts are frequent in patients with *COL4A1* mutations, present in 35 of 157 (22%) patients in one review.[2] In the ophthalmic literature, cataracts and associated eye findings in *COL4A1* patients have not been described in detail. To better characterize the ophthalmic phenotype, we present 2 cases referred to University of Colorado School of Medicine for evaluation.

Case 1

A 9-month-old boy was referred for an absent red reflex and exotropia. After a normal pregnancy, he was born full term without complications. After receiving no early pediatric care, he presented at age 8 months to a pediatrician with right-sided weakness. Family history was negative for ophthalmic problems, stroke, and kidney disease. Physical examination demonstrated right-sided spastic hemiparesis. Magnetic resonance imaging (MRI) of the brain revealed left frontal porencephaly and dysmorphic lenses (Figures 1B and 2A-B). On ophthalmological examination, he exhibited left-eye preference. Slit-lamp examination revealed bilateral central and posteriorly located opacities: 4 mm in the right lens and a <1 mm focal opacity in the left lens. He had a right exotropia of 35$^{\Delta}$, normal pupils, and a normal-appearing fundus. Cataract workup, including testing for TORCH (toxoplasmosis–other agents–rubella–cytomegalovirus–herpes simplex) infections, was negative. *COL4A1* and *COL4A2* genes were sequenced, revealing a c.2969G>T (p.Gly990Val) mutation in the *COL4A1* gene.

The Gly990 residue is completely conserved across species. The missense variant was evaluated by in silico