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Epstein–Barr Virus–Associated Dacryoadenitis Leading to Keratoconjunctivitis Sicca in a Pediatric Patient: A Case Report

Arash Maleki, MD; C. Stephen Foster, MD, FACS, FACR

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Introduction

Primary infection with the Epstein–Barr virus (EBV), a common pathogen, typically results in infectious mononucleosis (IM) in younger individuals. IM most often presents with fever, pharyngitis, anterior and posterior cervical lymphadenopathy, and hepatosplenomegaly. Acute EBV infection can lead to complications such as hepatitis, airway obstruction, and splenic rupture, ocular involvement.

Ophthalmic manifestations including conjunctivitis, dacryoadenitis, episcleritis, keratitis, and iritis have been described in reported cases. Upper eyelid edema is the most common ocular symptom and sign and may present even before systemic manifestation of the disease. Acute dacryoadenitis is a rare ocular complication of EBV infection and can be challenging to differentiate from upper eyelid edema. Acute dacryoadenitis is caused by inflammation of the lacrimal gland, which is located lateral to the extraconal orbital fat.

Case Report

We evaluated a 10-year-old boy with severe bilateral dry eye associated with significant pain and photophobia despite aggressive conservative therapy. On examination, he was extremely photophobic and had been largely housebound and socially limited since symptom onset. He was nearly unexaminable in the clinic; however, a detailed examination was performed under anesthesia. Best-corrected visual acuity in a darkened room was 20/100 OD and 20/70 OS. Examination revealed 2+ conjunctival injection bilaterally, abundant mucus discharge, and 2+ superficial punctate keratopathy. The palpebral lobes of the lacrimal glands were enlarged bilaterally, and the remainder of the examination was unremarkable.

In his past medical history, six months prior to evaluation, the patient had been swimming in a river in Arkansas with friends, after which all developed conjunctivitis. However, his ocular symptoms, including bilateral eyelid swelling, conjunctival injection, and ocular discomfort persisted. A diagnosis of viral conjunctivitis was made, and he was treated with topical tobramycin and dexamethasone four times daily for two weeks without improvement. Subsequently, increasingly severe superficial punctate keratopathy developed, and treatment was escalated to prednisolone acetate every two hours, dexamethasone ointment at bedtime, ciprofloxacin drops every four hours, and lubricants. After three weeks, there was no improvement in signs or symptoms.

Schirmer testing demonstrated zero values bilaterally. Corneal sensation was normal in both eyes. All conjunctival cultures were negative. Serologic evaluations revealed elevated alkaline phosphatase and aspartate aminotransferase levels, as well as an elevated serum angiotensin-converting enzyme level. Gallium scanning demonstrated increased uptake of gallium citrate in the parotid and lacrimal glands. All other laboratory studies were negative or within normal range, and HLA typing was noncontributory. A diagnosis of sarcoidosis was made, and the patient was treated with systemic prednisone (100 mg orally daily) without noticeable improvement in ocular signs or symptoms. The prednisone was subsequently tapered, and the patient was referred to our service.

Our serologic studies revealed elevated antibody titers to the Epstein–Barr virus viral capsid antigen, nuclear antigen, and early antigen. Lacrimal gland biopsy demonstrated no evidence of granulomatous inflammation, with preservation of a significant amount of normal-appearing glandular tissue, along with focal dacryoadenitis characterized by lymphocytic infiltration and focal neutrophilic infiltration within the tubules; a small number of acini showed scarring.

Immunohistochemical analysis disclosed almost no secretory IgA, indicating marked dysfunction of even the normal-appearing acini. EBV viral proteins EBNA-2 and VCA were identified by immunohistochemistry in the areas of lymphoproliferation, and EBV early restricted antigen was present in the tubular epithelial cells. No such staining was seen in control lacrimal gland tissue.

We considered acute keratoconjunctivitis sicca secondary to Epstein–Barr virus infection to be the most likely diagnosis. Intravenous acyclovir (640 mg four times daily for two weeks) was administered, followed by systemic cyclosporine (50 mg orally twice daily) to suppress the presumed immune-mediated lacrimal gland inflammation.

At 12-month follow-up, the patient was asymptomatic, with best-corrected visual acuity of 20/20 OU, an adequate tear meniscus, and Schirmer values of 15 mm OD and 10 mm OS.

The EBV early antigen titer normalized, and liver enzyme and serum angiotensin-converting enzyme levels returned to within normal limits.

We believe that this patient had Epstein-Barr virus induced severe keratoconjunctivitis sicca. Six months of immunomodulatory therapy was required, in addition to high dose intravenous Acyclovir therapy, to abort both the chronic infection and the infection-induced (presumed) autoimmune dacryoadenitis.

Discussion

Acute dacryoadenitis might be observed early in the course of EBV infection and sometimes precedes the typical symptoms of infectious mononucleosis. Several studies reported that fever and lacrimal gland swelling were the only symptoms of primary EBV infection in patients with pharyngitis or other symptoms**.** However, EBV-associated dacryoadenitis is mostly overlooked. In most cases, acute dacryoadenitis improves spontaneously, although some studies have reported that symptoms can persist for 3–6 weeks.

This case is unique in that the patient developed chronic dry eye and secondary keratoconjunctivitis sicca, which responded to intravenous acyclovir.

Conclusion: Chronic Epstein–Barr virus infection may cause chronic dacryoadenitis and secondary keratoconjunctivitis sicca, which may respond to antiviral therapy.

References

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