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Sir,

A Case of Severe Symptomatic Superficial Keratitis Associated with Epstein–Barr Virus

We recently saw a patient who has experienced a fluctuating (chronic) course of severely symptomatic bilateral superficial punctate keratitis with pannus formation and without any conjunctival injection who has had no mononucleosis-like symptoms throughout the duration of her illness and who shows no evidence of immunological dysfunction, but who has associated serological evidence of chronic Epstein–Barr virus (EBV) infection. Other than the pannus, the lesions appeared almost identical to Thygeson's superficial punctate keratitis (TSPK). While EBV infection is frequently subclinical (especially in children), it is commonly associated with several pathological conditions including infectious mononucleosis, African Burkitt's lymphoma, lymphoproliferative disorders in immunocompromised patients, chronic fatigue syndrome and nasopharyngeal carcinoma. Ocular involvement is uncommonly reported in EBV infection. Follicular conjunctivitis is the most frequent ocular finding associated with EBV, but any orbital/ocular tissues may be involved.[1–6] We suggest an EBV panel test on suspected TSPK patients.

Case Report

A 29-year-old white woman presented to our clinic on 21 May 1992 desiring further evaluation of her diagnosis of 'bilateral keratitis'. She first experienced blurred vision, severe photophobia and corneal irritation bilaterally in December 1991. She was debilitated from the photophobia and irritation to the point of not being able to work properly. In January 1992 she sought care from her ophthalmologist who began treating her for 'bilateral keratitis', and by the time we examined the patient, approximately 5 months after the onset of the symptoms, she had tried several courses of therapy including fluoromethalone, prednisolone phosphate 1%, and diclofenac. None of these treatments offered the patient significant relief. As a last resort she had been offered superficial keratectomy for removal of 'blood vessels' from her cornea. She came to us for a second opinion.

On presentation to our clinic the patient complained of continuing blurred vision, severe photophobia and irritation. She reported that her eyes had never turned red throughout the course of her illness, even while her symptoms were at their worst. She denied any systemic complaints, and denied any flu-like symptoms over the past 6–9 months. She also denied previous ocular trauma, significant past medical or ocular history, or medical or environmental allergies. Family history was positive for glaucoma (mother's father). She was not taking any medications, either topical or systemic.

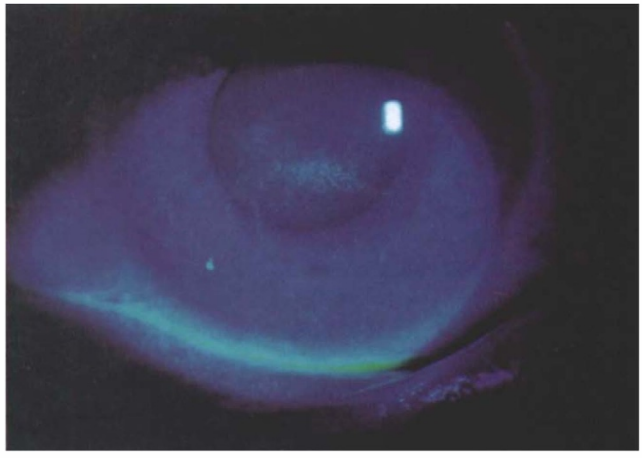

Fig. 1. *Right eye showing active 3 mm inferior pannus and superficial punctate keratopathy.*

Visual acuities were 20/30 in the right eye and 20/40 in the left, and ocular tensions (aplanation) were 16 and 14 mmHg. Fluorescein stain showed 3+ interpalpebral superficial punctate staining in both eyes, with a 1 mm round erosion centrally in the right eye. The punctate staining was coarse, pleomorphic, and too numerous to count. Thick, active pannus was noted inferiorly in both eyes, extending from 5 o'clock to 7 o'clock with vessel ingrowth reaching approximately 3 mm anterior to the limbus in the right eye, and 2.5 mm in the left (Figs. 1, 2). Anterior chambers were without reaction in either eye, and conjunctivae were not injected and had no significant follicular or papillary response. Schirmer's test with anaesthesia showed more than 15 mm of tear production bilaterally.

The lesions were cultured for bacteria, *Chlamydia* and viruses, and the patient was started on topical trifluorothymidine (Viroptic, Burroughs Wellcome). A complete blood count revealed a normal haemoglobin level with a white blood cell count of 4.1. A differential count revealed

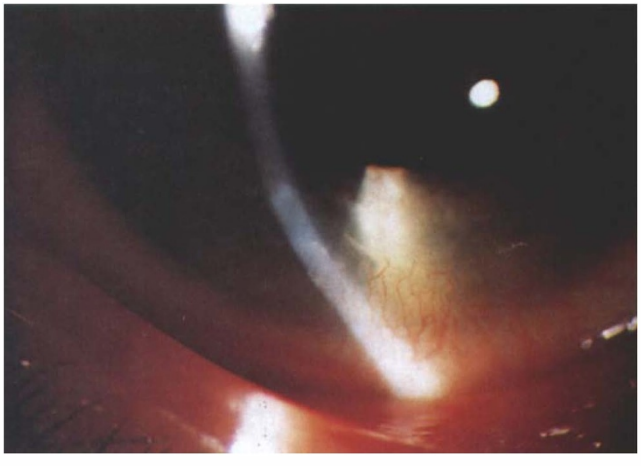

Fig. 2. *Left eye showing active 2.5 mm inferior pannus and superficial punctate keratopathy.*

55% polymorphonucleocytes, 3% bands, 27% lymphocytes, 14% monocytes and 1% eosinophils. Sedimentation rate, serum creatinine and fasting glucose were normal. Tests for syphilis (VDRL) and *Treponema* (FTA-ABS) were non-reactive, while anti-nuclear antibody, rheumatoid factor and *Chlamydia* antigen tests were negative. Noting the increased monocytes, a repeat complete blood count was ordered, as well as a Heterophile antibody test and EBV serology.

One week later, on 28 May 1992, the patient reported no significant improvement in her symptoms. Visual acuity was unchanged, but the right cornea showed resolution of the central erosion. The remainder of the examination was unchanged. *Chlamydia* and viral cultures were negative, but the bacterial culture showed light growth of coagulase-negative *Staphylococcus* which was susceptible to all antibiotics tested. The Viroptic was discontinued and bandage contact lenses were inserted bilaterally. No antibiotics were started as the coagulase-negative *Staphylococcus* was felt to be normal flora.

The following day the patient reported marked decreases in both corneal irritation and photophobia. Visual acuity remained unchanged, but corneal epithelial staining was mildly decreased in both eyes. Artificial tears were started in addition to the bandage contact lenses.

One week later, on 3 June, the patient remained symptomatically improved. Visual acuity improved to 20/30 in both eyes, and epithelial staining was reduced to 'trace' in both eyes. In addition, the inferior pannus showed significant resolution bilaterally, with moderate regression of vessels. A repeat white blood cell count was 3.7 with 44% polymorphonucleocytes, 10% bands, 35% lymphocytes, 10% monocytes and 1% basophils. Heterophile antibody screen was negative, but EBV serology showed a VCA/IgG titre of 1:1280, and an anti-EBNA titre of 1:128. VCA/IgM and EA titres were normal. The treatment plan remained unchanged.

Six weeks later, on 4 August, the patient complained of dryness and irritation in both eyes. Visual acuities had improved to 20/20 bilaterally, but her superficial corneal staining increased slightly to 1+. Anterior chambers were without cell or flare, and both inferior corneal vascular pannuses remained relatively quiet. Repeat EBV titres showed VCA/IgG of 1:2560 and EA of 1:640. VCA/IgM and anti-EBNA were unchanged. Because of the rising EBV titres the patient was referred to an Infectious Disease clinic, where EBV serology was repeated. VCA/IgG with VCA/IgM were unchanged, but EA had normalised and anti-EBNA had increased to 1:256. No additional treatment modalities were recommended, and bandage contact lenses with artificial tears were continued.

On 1 September, approximately 1 month after the last ophthalmological examination, the patient was again symptomatically much improved. Visual acuities were 20/20 in both eyes, and both corneas were completely without staining. Residual corneal haze was present only in the area of previous vascular pannus, and vessels had become completely quiet. There was no change in the treatment plan. On 6 October the patient remained asymptomatic and the physical examination was unchanged. EBV titres, however, again showed an increase, with VCA/IgG and VCA/IgM remaining the same but EA rising to 1:2560 and anti-EBNA rising to 1:2048. As the patient was asymptomatic and the corneas remained quiet under the bandage contact lenses no other therapies were undertaken. In April 1993 contact lenses were discontinued and the patient did well without them for several months. On 16 December 1993 she returned with severe discomfort and presented with 2+ superficial punctate epitheliopathy. Bandage contact lenses were restarted and the patient quickly became asymptomatic. As of 14 January 1994 she was still wearing bandage contact lenses.

Comment

Our patient represents a possible case of bilateral EBV keratitis, persistent or recurrent in its course, and not associated with any signs of mononucleosis or other systemic EBV-related disease. While the patient did exhibit mild disturbances in her blood count differential, there was no evidence of a primary immunological disorder that could account for her susceptibility to the continuing illness.

Her first EBV serologies suggested recent infection by the virus, with a moderately elevated VCA/IgG (1:1280) and a mildly elevated anti-EBNA (1:128). Subsequent serologies showed periodic peaks and valleys in the early phase markers (VCA/IgM and EA), which is taken as evidence for continuing infection/reinfection. Furthermore, rises in both VCA/IgG and anti-EBNA accompanied the fluctuations in early phase markers, thus signifying an overall continuing immune response aimed at the offending EBV.

As the EBV serologies exhibited a fluctuating course, the bilateral keratitis also showed regressions and relapses. Viroptic was discontinued after 1 week when the patient failed to show a clinical response. Bandage contact lenses and artificial tears seemed to have resolved the problem after approximately 1 month of treatment. Six weeks after the disappearance of superficial staining, however, the patient's symptoms of photophobia and irritation recurred. Superficial corneal epithelial staining had also recurred bilaterally, which again resolved with continued use of bandage contact lenses and artificial tears. A pattern linking the severity of her keratitis to the activity of the serologies is elusive. More study is needed in this area to establish the relationship between the two factors.

Like other herpes viruses, one would think, treatment of EBV keratitis would most probably rely on steroids and anti-viral therapy. Acyclovir has been shown to limit replication of EBV *in vitro* by interfering with its DNA polymerase.[7] The use of acyclovir has been reported to improve EBV ocular infection.[8] Several authors also reported the use of topical steroids for EBV keratitis and improvement was seen in all cases.[4,9] One patient suffered a recurrence after a rapid taper, but no patients experienced a worsening of the keratitis when the steroids were begun. The true clinical benefit of these treatment modal-

ities is difficult to assess, however, as the patient population is very small and the viral keratitis is possibly self-limiting anyway. Our patient did not have any improvement from either topical steroids or anti-virals.

Most of the previously reported cases of EBV keratitis have occurred in conjunction with mononucleosis;[4,5,9,10] however, 3 cases reported by Matoba and Wilhelmus in 1986 and 2 cases reported by Matoba and Jones in 1987 were without systemic symptoms. Corneal findings in the previously reported cases are broad, ranging from superficial/dendritic to stromal lesions of all depths.[1,4,5,8,10] Our patient represents another possible case of chronic EBV keratitis without systemic symptomatology, which displayed only superficial punctate staining throughout the course of the illness.

Matoba and Jones[6] in 1987 suggested that EBV keratitis might be a much more common entity than previously thought. Due to the variety of appearances of its keratitis and its propensity for causing a follicular conjunctivitis, it is easily confused with either adenoviral or herpetic lesions. These cases are usually self-limiting, however, so we do not routinely recommend Heterophile antibody screen or EBV serology. If the case is refractory or otherwise complicated, the above tests should be included in the diagnostic investigation of the keratitis.

Thygeson's superficial punctate keratitis (TSPK) is also without significant conjunctival injection, but causes no pannus and usually responds well to steroids.[11,12] There is also evidence suggesting that TSPK is responsive to trifluridine.[13] Our patient had proved unresponsive to weeks of topical steroid therapy by the time we evaluated her, and she already exhibited inferior pannus bilaterally. Additionally, her keratitis was largely unchanged by a 1 week course of Viroptic which was begun immediately after we saw her.

TSPK was first described in 1950. It is a chronic or recurring, usually bilateral condition which is accompanied by tearing, irritation and photophobia. Corneal lesions tend to exhibit microscopic coarse, granular opacities which often grouped into a larger round or oval conglomerate.[12,13] Whereas the lesions strongly resemble those of adenoviral keratoconjunctivitis, the conjunctivae in TSPK remain uninvolved. The lesions of our patient's EBV keratitis (which may or may not be accompanied by a conjunctival response) closely resembled those of TSPK. It may be that an EBV panel and/or Heterophile antibody test should be included in the investigation of TSPK in order to rule out EBV as a possible cause.

The authors of this article, as well as their children and spouses, possess no proprietary interest in any of the companies or products mentioned in the text, nor are they receiving reimbursements of any sort for its publication.

Supported in part by Research to Prevent Blindness.

Hamid Sajjadi, MD
Mahyar Parvin, BA, BS

Kansas Eye Center
Kansas University Medical Center
Kansas City, KS 66160-7379
USA

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Sir,

Nasopharyngeal Carcinoma: A Cause of Foster Kennedy Syndrome

In 1911, Foster Kennedy presented 6 cases of unilateral papilloedema with contralateral optic atrophy, often accompanied by loss of smell on the atrophic side, as pathognomonic of a space-occupying basofrontal lesion on the side of the optic atropy.[1] We describe a 33-year-old patient with nasopharyngeal carcinoma with extensive intracranial extension causing ipsilateral optic atrophy and contralateral papilloedema as a result of increased intracranial pressure.

Case Report

A 33-year-old white male patient had noted sudden visual disturbances with his right eye in the form of blurred vision and dull colours. He also complained of headaches, worst in the morning, but he denied any sickness. His wife added that he had appeared confused on a few occasions recently.

There had been a past history of left squint and amblyopia when the patient was a child. Eighteen months previous to his referral to the Eye Department, he was diagnosed as having nasopharyngeal carcinoma on the left side. It was noted then that the tumour mass had extended up towards the base of the skull through the foramen lacerum and was encroaching onto the left cavernous