proteases may have a regulatory role in the destructive processes which can occur in the cornea following infection, inflammation, and trauma. The levels of antiproteases in tears are found to rise dramatically in patients with corneal ulceration and they are thought to leak from inflamed conjunctival vessels.^{4,5} This is supported by the fact that the level of serum albumin in tear fluid rises together with α_1 antitrypsin in conditions involving conjunctival inflammation. The ratio of serum albumin to α_1 antitrypsin is 20:1 in serum but can be up to 3:1 in tears of inflamed eyes. This relative increase in tear fluid α_1 antitrypsin may simply reflect the fact that the molecule is smaller than albumin and can thus leak from blood vessels more easily.⁶ However, the tear fluid concentration of α_1 antitrypsin decreases at a slower rate than other serum antiproteases following resolution of conjunctival or corneal pathology, and the possibility of its local release or production has been suggested.³

Berman *et al*⁶ found one patient with genotype PiMZ and measured α_1 antitrypsin levels in both serum and tear fluid of an eye with extensive corneal ulceration. Both levels were low when compared with other patients with similar ocular pathology. They found no patient with corneal ulceration who had genotype PiZZ. Our patient was known to have low serum α_1 antitrypsin and was found to have a very low level of α_1 antitrypsin in tear fluid when compared with our normal controls. Sen *et al*⁵ found levels of α_1 antitrypsin to be independent of age and sex and thus we ignored the age difference between our subject and controls. The α_1 antitrypsin level in tears in our patient was also low when compared with other published values in normal healthy individuals without ocular pathology (Table 2).

Table 2 Previously published values of α_1 antitrypsin concentration in tears of normal healthy subjects without ocular pathology

Author	Tear fluid (mg/l)		
	Normal (SD)	No of subjects	99% Confidence interval
Prause ³	3.5 (2.2)	14	1.73–4.77
Gupta <i>et al</i> ⁴	4.2 (2.2)	76	2.92–5.48
Sen <i>et al</i> ⁵	4.0 (3.34)	81	3.01–4.99

We suggest that insufficient levels of α_1 antitrypsin in serum in our patient who was homozygous PiZZ, resulted in low tear fluid α_1 antitrypsin levels during an acute infective episode of the adjacent conjunctiva and lid margin. This enabled protease activity on the cornea to proceed without one of the normal regulatory processes, resulting in extensive corneal ulceration and subsequent scarring. This complication of α_1 antitrypsin has not previously been reported.

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Progressive corneal vascularisation as a previously unreported complication of neonatal herpes simplex infection

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Bilateral, widespread progressive corneal vascularisation as a result of neonatal herpes simplex infection has not been reported previously. The child presented, one of the first treated with systemic acyclovir, demonstrates the morbidity associated with herpes simplex infection.

Case report

JP was born in January 1982 at 33 weeks'

gestation by caesarean section 5 days after spontaneous rupture of membranes with an antepartum haemorrhage. She was initially well, but on day 3 suffered three apnoeic attacks requiring ventilation, and developed bilateral purulent conjunctivitis. On day 5 she developed an erythematous desquamating rash over her scalp which became generalised (Fig 1), mouth vesicles, and pneumonia. Cultures grew herpes simplex type II virus and a diagnosis of dissemi-

nated herpes simplex was made. Serial antibodies and maternal status are unavailable. The child was commenced on topical and intravenous acyclovir 6.25 mg three times a day. A cerebrospinal fluid leucocytosis ($150 \times 10^6/l$) suggested central nervous system involvement. Ocular examination showed a marked bilateral keratitis affecting all corneal levels, initially obscuring iris and other details, shown a few days later in Figure 2.

Figure 1 Baby JP at 5 days of age with widespread erythematous vesicular desquamating rash.

Figure 2 Baby JP at 9 days. Note resolution of rash and conjunctivitis, but marked keratitis.

Figure 3 JP at 10 years of age. Superficial corneal vascularisation with epithelial opacification. Epiphora but normal lid architecture.

Her general condition improved and she was discharged at 42 days, with no obvious neurological deficit.

At 1 year examination revealed a small left corneal opacity and visual acuity estimated with a Catford drum was 6/9 in her right eye and 6/12 in the left. She had epiphora, due to an HSV canaliculitis, and repeated vesicular eruptions over all parts of her body, requiring intermittent systemic and topical acyclovir. An intermittent left divergent squint resulted in left amblyopia despite patching, and a Snellen acuity of 6/36, compared with 6/12 on the right.

From 5 years she developed a progressive circumferential bilateral superficial vascularisation with central epithelial cysts and opacification ahead of the vessels (Fig 3). This has reduced her vision to 6/60 in the left and 6/36 in the right eye. Typical recurrent HSV keratitis or uveitis have never been observed.

Comment

Neonatal HSV may result in devastating morbidity and mortality; before antiviral therapy 75% of these patients died by 6 months.¹ Agents such as acyclovir reduce mortality, but even recent multicentre trials reported 54% mortality in disseminated neonatal HSV infection by 1 year.²

Widespread superficial corneal vascularisation has not been reported after neonatal HSV infection. Acutely, conjunctivitis occurs most frequently (10%), followed by keratitis (7%). Acute series of neonatal HSV suggest 17% ocular involvement,³ but follow up series report up to 60%,⁴ recognising more retinochoroidal scars, optic atrophy, and squint. Other reported complications include cataracts, phthisis, corneal scarring, recurrent keratitis, and epiphora.

We consider four mechanisms for this clinical picture. First is chronic or recurrent HSV infection. Despite close ophthalmic monitoring, typical HSV keratitis was not seen: no dendrites, faceting, disciforms, or deep vessels appeared and the resulting diffuse superficial vascularisation is atypical.

Second, HSV is a trigger for Stevens-Johnson syndrome as are drugs (unreported for acyclovir). The skin and mucosal lesions, photophobia, vessel pattern, and ocular surface disorder are similar to Stevens-Johnson syndrome, as is the progression over years.⁵ However, keratinisation, metaplastic lashes, and conjunctival scarring were absent.

Third is pemphigoid. Superficial vascularisation and loss of normal corneal epithelium occur, but always accompanied by conjunctival scarring.

Fourth, and perhaps most likely, is loss of limbal stem cells. Surgical or chemical injuries can lead to a progressive superficial vascularisation and loss of normal epithelium if they involve a major portion of the limbus.⁶ This probably also occurs in Stevens-Johnson syndrome, pemphigoid, and aniridia, but with additional features. Our patient had only corneal features, which suggest a pure syndrome of stem cell loss.

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Bilateral angle closure glaucoma and accelerated cataract formation in a patient with AIDS

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Recent reports have described the occurrence of bilateral angle closure glaucoma secondary to choroidal effusions in patients with AIDS. We report the rapid onset of cataracts in a patient with this syndrome.

Case report

A 44-year-old white man with *Pneumocystis carinii* pneumonitis, presented with visual deterioration over the course of 1 day. His past ocular history was unremarkable. Visual acuity on presentation was counting fingers at 1 metre in both eyes, which improved to 6/12 with pinhole correction. Anterior segment examination revealed bilateral chemosis and microcystic corneal oedema. The anterior chambers were shallow, especially peripherally; the angles were closed 360 degrees in both eyes on gonioscopy. The pupils were in a mid-dilated position, and reacted to bright illumination segmentally. The intraocular pressure on applanation tonometry was 38 mm Hg (right eye) and 36 mm Hg (left eye). The lenses were clear and the vitreous was quiet. The retinal vasculature was tortuous and dilated, there were scattered cotton wool spots and peripheral retinal haemorrhages, consistent with HIV microvasculopathy. The ora was posteriorly displaced. Bilateral acute angle closure glaucoma was diagnosed.

Treatment was started with intravenous and oral acetazolamide therapy, and bilateral topical pilocarpine 4% and dexamethasone 0.1%. The following day the intraocular pressures remained elevated at 34 mm Hg in both eyes. Bilateral YAG iridotomies were performed, with no appreciable change in anterior chamber depth or change in intraocular pressure. Pilocarpine was discontinued and cyclopentolate 1% therapy begun following the laser procedure. A B ultrasound scan revealed choroidal and ciliary body thickening in both eyes (Fig 1). Within the course of the next 36 hours of instituting cycloplegic therapy the anterior chambers deepened

fully and the intraocular pressure decreased to 10 and 12 mm Hg. The following day his intraocular pressures were below 10 mm Hg in both eyes and remained so subsequently. Cortical haze was noted in both lenses; the opacification continued to increase and, within a week, both lenses were completely opaque. The following week the patient underwent cataract extraction and intraocular lens insertion, with a final best corrected visual acuity of 6/9 (right) and 6/6 (left). All the ocular samples removed at the time of surgery including aqueous, cortical aspirate, and the lenticular nuclei from both eyes were examined for cytomegalovirus (CMV) and HIV p24 antigens. These tests were all negative. Serological studies confirmed infection with HIV, with a CD4 count of 32. A significant fourfold rise in CMV antibody titre was detected but no CMV virus was isolated from any source.

Figure 1 B scan ultrasound of left eye demonstrating annular choroidal effusion.

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