Methods

All cases of newly diagnosed keratoconus patients seen in the Ophthalmology Department between September 1997 and December 2001 were analysed retrospectively using the videokeratography database. This included the vast majority of cases of keratoconus seen in the department during the analysed period—the clinical diagnosis on presentation was made by an ophthalmologist.

BRI is the only ophthalmology service in the city of Bradford (catchment population of 470 000) and receives all GP or optician (via GP) referrals of this type.

According to Census 2001, the ethnic background of population in Bradford is white 93.48%, Asian 4.49%, black 0.5%, Chinese 0.25%, and others 1.18%. The ethnic groups in the Asian population in Bradford are Pakistani (87%), Indian (7.3%), and Bangladeshi (5.7%).

The proportion of population other than white and Asian (ie 1.93%) was considered negligible for the purpose of this study.

Normally distributed data were analysed using Student's *t*-test for unpaired groups of patients. A significance level of $<5\%$ was chosen in all tests. Data are presented as mean $\pm$ standard deviation.

Results

In all, 197 new patients were diagnosed with keratoconus over this 4-year period. There were 130 Asians (69 men and 61 women) and 67 whites (49 men and 18 women).

The incidence rate was approximately 32.3 per 100 000 per year for Asians and 3.5 per 100 000 per year for whites. The relative incidence is 9.22–1.

Asian patients were significantly younger than white patients at presentation (mean age 23.0 ± 7.0 years *vs* 27.8 ± 8.1 years, $P<0.001$).

The first corneal graft was performed on 15 (7.06%) Asian patients and on four (5.9%) white patients.

Of those having grafts, Asian patients were significantly younger than white patients at the time of diagnosis (mean 20.8 ± 5.3 years *vs* 32 ± 4.9 years, $P<0.002$) and operation (mean 22.13 ± 5.6 years *vs* mean 33.5 ± 5.0 years , $P<0.002$).

Comment

Our study showed a relative incidence of keratoconus of 9.22–1 in Asian patients compared to white patients. This is comparable to the ratio of 7.5–1 in the study from Dewsbury,[1] where the ethnic distribution of the Asian population is similar to Bradford, predominantly of Pakistani extraction, and is much higher than in the Leicester study of Pearson *et al*[2] of 4.4–1 with a majority of Asian population of Indian extraction. None of the patients in our study admitted to a family history of keratoconus.

The Asian population in this study had a similar higher than average prevalence of consanguineous marriages as in the Dewsbury data,[1] therefore our results support the attractive postulate of the role of genetics in keratoconus.

There is evidence in the literature that prolonged contact lens wear may induce keratoconus in predisposed individuals, if worn over 4 years.[3] Further studies are needed to assess the causative role of difference in the duration of contact lens wear between different populations diagnosed with keratoconus. This is relevant because, for cultural reasons, the Asian population tends to prefer contact lenses to glasses.

References

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2 Pearson AR, Soneji B, Sarvananthan N, Sandford-Smith JH. Does ethnic origin influence the incidence or severity of keratoconus? *Eye* 2000; **14**: 625–628.
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Sir,

Reply to I Cozma *et al*

We are pleased that Cozma *et al* have been able to support our finding (Georgiou *et al*[1]) of a higher incidence of keratoconus in Asian populations, particularly those from Pakistan.

We understand cultural differences in contact lens wear may influence the incidence of keratoconus between the ethnic groups, however, the evidence for this to cause such a significant difference is limited. Weed *et al*[2] reported only 28% of patients presenting with a new diagnosis of keratoconus had a history of contact lens use. In our study, the most common treatments for refractive error, prior to referral, were glasses or soft contact lens rather than rigid lens, which are postulated to be the most likely cause for keratoconus. Interestingly, in our study, there was no significant difference in initial hospital treatment between the white and Asian patients. A total of 41% of white patients and 44% of Asian patients were treated with contact lens.

Cozma *et al*'s findings further support the theory of a genetic basis to keratoconus and emphasizes the need for genetic research.

References

1 Georgiou T, Funnell CL, Cassels-Brown A, O'Conor R. Influence of ethnic origin on the incidence of keratoconus and associated disease in Asians and white patients. *Eye* 2004; **18**: 379–383.
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Sir,

Mucous plaque keratitis associated with aniridia keratopathy

Aniridia is a rare congenital disorder with autosomal dominant inheritance in the majority of cases, but it occurs sporadically in 15% of cases.[1,2] Affected individuals show a spectrum of clinical features. Aniridia is the defining feature but developmental defects are not restricted to the iris. Corneal opacification, cataract, glaucoma, and macular hypoplasia all contribute to visual loss. Aniridia patients with sporadic mutation have a higher incidence of Wilms' tumour.[3]

We present a case where marked mucous plaque keratitis developed in a patient with aniridia keratopathy following a corneal graft. Mucous plaques are not a known feature of aniridia keratopathy. It is unusual for mucous plaques to develop unless very severe atopic disease is present. Our patient had marked corneal disease with large mucous plaques in the presence of only mild atopic conjunctivitis. We propose that the nonhealing corneal epithelial defect of aniridia keratopathy provided the environment for rapid formation of mucous plaques.

Case report

A 32-year-old woman with familial aniridia had a long history of progressive corneal opacities in both eyes. Her father, grandfather, and sister were also affected. At the age of 12 years, her visual acuity was 6/36 in both eyes; by the age of 19 years, it dropped to 6/60 and she had developed chronic nonhealing corneal ulcers that were worse in her left eye. Exacerbations of the corneal ulceration were treated with topical lubricants and antibiotics. In 1993, the patient underwent left lamellar keratoplasty with an initially satisfactory result with visual acuity improving from 1/60 preoperatively to 6/36 three months postoperatively. After 3 years, she developed glaucoma in the left eye and her intraocular pressures became refractory to medical treatment. Trabeculectomy augmented with mitomycin C was performed on the left eye in March 1998 and the intraocular pressure has since been maintained below 20 mmHg with timolol drops.

The right eye later developed nonhealing corneal epithelial defects similar to those in her left eye and a lamellar keratopathy was performed in the right eye in October 2000. The corneal graft was initially clear but later developed epithelial disturbance with deposition of mucus which soon turned into persistent plaques on the surface. This was associated with deterioration of vision and pain.

At that time we were not aware of any coexisting problem that might have contributed to the formation of mucous plaques. She used topical steroids and mucolytic agents but the mucous plaques did not resolve. She therefore had a repeat lamellar keratoplasty in October 2002. Further questioning revealed a history of hay fever associated with watery eyes. In addition, she was prone to bronchospasm and was allergic to house dust and cats.