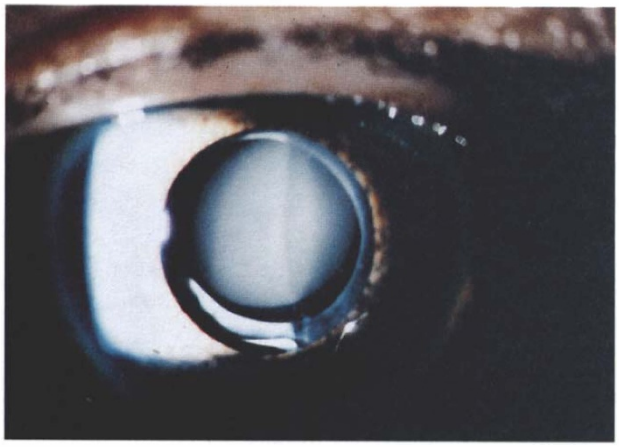

Fig. 1. *Photograph showing the central cloudiness of the intraocular lens.*

difficult. Her vision dropped to 6/18 corrected. We are considering exchanging the IOL as she is significantly affected by the drop in vision. However, she has significant corneal guttata in that right eye.

Comment

Clinically insignificant glistenings on acrylic IOLs have been described a week following surgery or after 48 h in laboratory conditions.[8] Temperature fluctuations were linked to these findings and the glistenings were thought to be due to microvacuole formation within the lens polymer as the temperature exceeded the glass transition temperature. Water from the anterior chamber was then able to enter these vacuoles and cause the glistenings, due to the different refractive indices of water and the lens polymer. These glistenings disappeared when the IOLs were dehydrated/dried.[8]

Fogging of excessively warmed AcrySof IOLs has been reported when the lenses were unfolded in the eye. When explanted they became clear again, presumably due to drying.[9] The IOL in this case was kept at room temperature and not pre-warmed. Condensations on acrylic[10] and silicone[11] IOLs have been noted after vitrectomy and air-fluid exchange, but these only occur on posterior surfaces of IOLs. As far as we are aware there has been no such reported clouding of an IOL this long after routine phacoemulsification surgery.

The ideal management of any foggy IOL is removal at the time of primary surgery, but in this case the fogging only became apparent about 7 months post-operatively. This incident has been reported to the distributor and the Medical Devices Agency. We speculate whether this was a problem in design/manufacture and whether temperature fluctuations that occur daily in the human body can cause this. We are not aware of another published report of this problem although a significant number of these lenses have been implanted. We recommend caution in the use of IOLs made from poly-2-hydroxyethyl methacrylate and that patients with such implants should be followed up for longer than usual.

We would like to thank Mike Geall, Leeds General Infirmary, for the photograph.

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Sir,

Familial intermediate uveitis: a case report of two brothers

Intermediate uveitis (IU) is a well-recognised chronic ocular inflammation characterised by an inflammatory aggregate at the inferior vitreous base, pars plana and peripheral retina. The aetiology of the condition remains obscure, but there is some indirect evidence that autoimmunity plays a role. We present two brothers with IU, together with the results of their HLA typing and that of their immediate family.

Case reports

Case 1. An otherwise healthy male initially presented at the age of 3 years with a right convergent squint. Ocular examination was otherwise normal, visual acuity being 6/5 bilaterally. He underwent right recession/resection at the age of 4 years. Three years later at a routine review he had a left convergent squint. Visual acuities were 6/4 right and 6/6 left. Examination revealed vitreous cells and inferior 'snowball' opacities on the left, with no other fundal abnormality. The right fundus was normal. Investigations including chest radiograph, ESR, *Toxocara* and *Toxoplasma* titres were all normal. Since this time the right eye has remained unaffected, and the left has been treated with topical and periocular steroids for exacerbations of increased vitreous opacity and macular oedema. His visual acuities are currently 6/4 right and 6/12 left.

Case 2. The brother of case 1, 6 years his junior, was first examined at the age of 2 years when he was found to have a left convergent squint. Ocular examination was normal. Corrected visual acuity was 6/6 right and left. At a routine review at the age of 6 years he was found to have mild cellular infiltration and inferior 'snowball' opacities in the vitreous of both eyes, with no other fundal pathology. Visual acuities were 6/5 right and left, and he had no visual symptoms. Since then there has been no significant increase in inflammatory signs and vision remains unaffected. He has received no treatment.

After the diagnosis of case 2, the boys' parents and two sisters were examined. None had any signs of IU, nor a history of relevant symptoms. HLA typing was performed, with the following results: Case 1: HLA A 24,31; B 7,62; DRB1 0301,1201/2; DQB1 0201, 0301. Case 2: HLA A 3,31; B 7,62; DRB1 0301,1201/2; DQB1 0201,0301. Sister 1: HLA A 24,11; B 7,35; DRB1 0301, 0401-22; DQB1 0201, 0302. Sister 2: HLA A 3,11; B 7,35; DRB1 0301, 0401-22; DQB1 0201, 0302. Father: HLA A 11,31; B 35,62; DRB1 1201/2, 0401-22; DQB1 0301,0302. Mother: HLA A 3,24; B 7,−; DRB1 0301, −; DQB1 0201, −.

Comment

There is conflicting evidence in the literature concerning HLA associations in IU. Davis *et al.*[1] found an increased incidence of the DR-2 allele (DRB1 15 and 16 under current nomenclature) and/or DQw-1 in a study of 43 cases. Malinowski *et al.*[2] also found an association with DR 2, together with B 8 and B 51 in a similar-sized group. Malinowski also suggested that the association with DR 2 provided further evidence for a link between IU and multiple sclerosis. Martin *et al.*[3] found a significant incidence of A 28. There are some reports in the literature of IU occurring in familial clusters,[4–8] but only three of these cite HLA typing.[6–8] The latter are also conflicting: Duinkerke-Eerola *et al.*[6] found no common typing in a father and son, but Wetzig *et al.*[7] and Augsburger *et al.*[8] each found similarities between affected family members.

The two affected children but not their sisters share a paternal haplotype: A 31, B 62, DRB1 1201/2, DQB1 0301. As the mother is homozygous for all but the A locus all four children have the same maternal B, DR and DQ alleles, but the two brothers have different maternal A alleles, as do the two sisters. It must be stated, however, that although neither parent nor the sisters of our two cases had any evidence of IU, a previous subclinical episode cannot be excluded. There is no commonality between these types and the previously reported HLA associations of IU. There is also no common link between any of the HLA types found in familial IU, including the current report.[7,8]

The pathogenesis of IU remains poorly understood but it is widely assumed that there is an underlying abnormality of T cell function.[7,9] The apparent HLA associations, and the occasional association with other presumed autoimmune diseases such as multiple sclerosis, arguably provide indirect evidence that IU is an autoimmune disease. The occurrence in familial clusters may mean that there is a genetic susceptibility to the disease, or that there is a common environmental trigger. HLA similarities between affected family members may be nothing more than a chance association, but there may be other, unknown genetic determinants. If HLA typing is of relevance in IU, the lack of a consistent pattern perhaps supports the theory that it is a group of diseases that share a common anatomical distribution.

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