Resolution of calcific band keratopathy after lowering elevated serum calcium in a patient with sarcoidosis

EDITOR,—Calcific band keratopathy and its relation to hypercalcaemia is well known, but resolution following correction of a high serum calcium level is rare. We report a case where such resolution was documented using corneal light scattering measurements in a patient with sarcoidosis.

CASE REPORT
A 32-year-old black man presented with a 6 month history of blurred vision in both eyes but no photophobia or ocular discomfort. Three months earlier he had developed a right sided facial palsy which resolved spontaneously over 2 weeks. His medical history was notable for weight loss of 12 kg over the previous 6 months, excessive thirst, a dry cough, and shortness of breath on exertion.

General examination was normal. The best corrected visual acuities were 6/12, N8 right, 6/9, N6 left. Yellow nodules were present in the inferior fornices of both eyes. Both corneas had interpalpebral calcific band keratopathy and occasional mutton fat keratic precipitates (Fig 1A). A mild anterior uveitis was present but examination of the vitreous, fundi, and ocular motility was normal.

The clinical diagnosis of sarcoidosis with hypercalcaemia was supported by findings of bilateral hilar lymphadenopathy on chest x ray; raised angiotensin converting enzyme level (188 IU/l; normal range under 53 IU/l); and abnormal liver and renal function tests. The corrected serum calcium was elevated at 3.56 mmol/l (normal range 2.1–2.5 mmol/l) with a normal serum phosphate and albumin. The diagnosis was confirmed histologically by biopsy of a conjunctival granuloma.

The patient was treated with intravenous rehydration and high dose systemic steroids (80 mg orally tapered over 6 months). Within 1 week his general condition had improved and his serum calcium was normal. Almost complete resolution of the band keratopathy (Fig 1B) and improvement of the visual acuity to 6/6, N4-5 in both eyes occurred over the following 6 months. Corneal light scatter (glare) measurements confirmed the resolution of his band keratopathy (Fig 2).

In those systemic diseases where band keratopathy arises as a result of hypercalcaemia a period of observation is indicated, following normalisation of serum calcium, before considering surgical or excimer laser therapy, as the keratopathy may improve as happened in this case.

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Figure 2 Glare (upper two lines) and Snellen visual acuity (lower two lines) measurements over the treatment period. Glare of under 50 standardised grey units is normal.

COMMENT
Calcific band keratopathy is associated with systemic conditions causing hypercalcaemia or raised serum phosphate and a number of chronic ocular conditions, including glaucoma, uveitis, corneal infections, and long term intraocular silicone oil.2 Sarcoidosis is the most common cause of hypercalcaemia associated with band keratopathy and is thought to be due to activated pulmonary macrophages producing 1,25 dihydroxyvitamin D.3 In patients who have sarcoidosis with ocular involvement, hypercalcaemia occurs in 17% but calcific band keratopathy is rare occurring in only 4-5% of cases.4 The deposition of calcium salts is dependent not only on the solubility product of calcium and phosphate being exceeded, but also on altered tissue physiology5 and raised pH which occurs interpalpebrally and accounts for the distribution of band keratopathy.6 Patients may be asymptomatic or complain of reduced vision or glare, and if the calcium breaks through the corneal epithelium it causes ocular discomfort. It is widely assumed that when calcific band keratopathy is present the only effective treatment is surgical or excimer laser therapy. There have been two case reports of early band keratopathy improving with treatment to lower the serum calcium (one associated with renal failure7 and one with hypervitaminosis D8). This is the first reported case of advanced band keratopathy resolving in a patient with sarcoidosis. Photography and glare measurements using a PC generated flickering glare source, were used to document improvement.1 The latter is a measure of visual impairment due to ‘forward scattering’ of light by corneal opacities. It is a more sensitive measure of the effect of corneal opacities on visual function than Snellen visual acuity and may be useful in deciding an endpoint to treatment (Fig 2).

Pseudoxefoliation material on an acrylic lens

EDITOR,—We report deposition of pseudoxefoliative material on the anterior surface of an acrylic posterior chamber intraocular lens which has been observed for 5 years. The morphological pattern of radial striations closely resembles that normally seen on the crystalline lens and detailed examination of the distribution pattern suggests that deposition has occurred in areas exposed to highest flow of aqueous humour.

CASE REPORT
A 59-year-old Somali seaman presented in January 1980 with a 2 year history of decreased vision in his left eye. Exophthalmos was noted by others. Ocular examination was otherwise unremarkable and he had extracapsular cataract extraction from the left eye in August 1980. The procedure was uneventful. A Pearce tripod posterior chamber intraocular lens was inserted into the capsular bag inferiorly and the sulcus superiority.

In June 1990 following dilatation of the left pupil pseudoxefoliative material was found on the temporal half of the anterior surface of the intraocular lens (Fig 1). Radial striations were observed, similar to those commonly seen in

Figure 1 (A) Interpalpebral calcific band keratopathy at presentation with a serum calcium level of 3.56 mmol/l. (B) Resolution of calcific band keratopathy after 3 months of treatment with a tapering course of oral prednisolone and a normal serum calcium.
pseudoexfoliative material on a crystalline lens. The pattern of deposition has remained remarkably constant over the subsequent 4 years (Fig 2). Local extension was noted at position A in 1992 but no further change had occurred by 1994. This is the only definite progression in deposition we have identified. The consistency of the pattern between 1990 and 1994 is particularly obvious at positions B, C, and D. Pseudoexfoliative material was first noted on the pupil margin and on the anterior iris surface in this eye only in 1994 (Fig 3A).

Extracapsular lens extraction with peripheral iridectomy was performed in the right eye in 1991. No pseudoexfoliative material was observed in that eye until 1994 when heavy deposits were noted at the edges of the peripheral iridectomy (Fig 3B). Deposits have not been noted on the intraocular lens in this eye. There is no pseudoexfoliative material on the posterior corneal surface or in the chamber angle in either eye but there is heavy angle pigmentation. Intraocular pressures remain normal in both eyes.

**COMMENT**

Pseudoexfoliative material has been reported on the anterior hyaloid face following intraocular lens extraction. There is one report of diffuse deposition on the anterior surface of a posterior chamber lens where the posterior capsule was intact. It has also been noted on the posterior surface of three posterior chamber lenses following posterior capsular disruption.

While our case resembles that of Ringvold and Bore with anterior surface deposition and intact posterior capsule, it clearly shows the additional feature of radial striations in the pattern normally seen on the crystalline lens. (As in Ringvold and Bore’s case we detected scanty deposits in the undilated pupil zone.) The sectoral distribution of the deposit is interesting. This temporal sector was the only area free of peripheral adhesion between iris and capsule and was therefore presumably subjected to greatest flow of aqueous humour. If deposition is related to aqueous flow it might also explain the radial striations which lie along the direction of flow. The absence of material on the lens implant in the right eye, if not simply a feature of the shorter interval following surgery in this eye, might be explained by lower rate of aqueous flow over the implant surface. This eye had no peripheral adhesions of the posterior iris surface which might channel and enhance the flow in a specific sector. With the pupil undilated the iris in each eye seemed equidistant from each quadrant of the lens implant. There has been little change in the pattern of deposition over a 5 year period. Ten years after implantation deposition on the lens appears to have reached equilibrium, but deposition on the iris was not seen until a further 4 years had passed. The acrylic lens in the right eye shows no material at 3 years after lens extraction, although heavy iris deposition has developed. Ringvold and Bore noted deposition on a lens implant only 18 months after surgery.

It is generally agreed that pseudoexfoliative material is produced by both the lens epithelium and other intraocular tissues. Pseudoexfoliation appears to be a systemic connective tissue metabolic disorder, perhaps resulting in the disordered synthesis or assembly of microfibrils. Pseudoexfoliative material is produced by a variety of different cell types such as epithelial cells, fibroblasts, and all types of muscle cells, and is found not only throughout the orbit, but widespread in the body of patients with pseudoexfoliation syndrome.

Our observations confirm that pseudoexfoliative material produced by one intraocular tissue can be deposited on remote structures including a polymethyl methacrylate lens.

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Giant dacryops in a patient with ocular cicatricial pemphigoid

EDITOR,—The term 'dacryops' was introduced by Schmidt in 1803 when he described cysts arising from the palpebral lobe of the lacrimal gland.1 Its formation is believed to be caused by occlusion of the lacrimal duct openings due to conjunctival inflammatory or traumatic changes followed by dilatation of the inflamed and weak duct walls. Conditions such as trachoma which cause conjunctival scarring have been reported as antecedents of ductal cyst formation.2 Ocular cicatricial pemphigoid (OCP), a relatively rare, chronic, progressive disease causing significant conjunctival scarring to our knowledge has not been documented as a cause of dacryops. We describe a patient with OCP who had a giant cyst on his left upper lid which was proved on histopathology to be a lacrimal duct cyst.

CASE REPORT

A 63-year-old male patient, diagnosed 16 months earlier as having OCP, developed massive swelling of the left upper lid over 1 month (Fig 1A). No improvement was noted with either antibiotic or steroid treatment. B-scan ultrasound demonstrated a cystic lesion which on aspiration revealed 6 ml of clear yellow fluid. The decompressed cyst reformed within 24 hours. A computed tomogram showed that the cystic mass, which measured 3.4×1.8×1.8 cm, was confined to the left preseptal space slightly displacing the left eyeball posteriorly (Fig 1B). The patient underwent total excision of the cyst through a lid crease incision. The cyst was noted to be posterior to the markedly attenuated levator aponeurosis and Müller's muscle, adherent to the superior border of tarsus, and in a subconjunctival location. A portion of the palpebral lobe of the lacrimal gland to which the cyst was attached was also excised. Histopathology showed the cyst wall (Fig 2A) and dilated ducts of the lacrimal gland with surrounding acute and chronic non-granulomatous inflammatory reaction (Fig 2B) consistent with a diagnosis of lacrimal duct cyst with dacryoadenitis. No recurrence of the cyst was noted for the next 3 months.

COMMENT

Ocular cicatricial pemphigoid is a chronic disease characterised by conjunctival shrinkage, entropion, trichiasis, xerosis, and corneal opacification causing blindness.3 Fibrous occlusion of the lacrimal ducts in OCP can cause decreased aqueous tear production. The development of lacrimal duct cyst, however, has not been described as a common finding. Cyst formation as a result of collection of tears proximal to the obstruction does not readily occur because obliterating these ducts leads to gland atrophy and cessation of secretion.1

In this patient we believe that the dacryops originated from an inflamed main lacrimal gland duct containing tears produced by the

Figure 1 (A) A 63-year-old male patient with ocular cicatricial pemphigoid and a giant cyst on the left upper lid. His left eye was covered by the left upper lid cyst that caused stretching of both the lid skin and the upper palpebral conjunctiva. (B) Computed tomography scan demonstrates the preseptal location of the cystic mass (arrow) displacing the left globe posteriorly.

Figure 2 (A) Portion of the giant cyst wall with surrounding haemorrhagic connective tissue containing inflammatory cells. (Haematoxylin and eosin, ×15.) (B) Part of the main lacrimal gland showing moderate infiltration of lacrimal gland acini with acute and chronic inflammatory cells. (Haematoxylin and eosin, ×38.)
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Office of Continuing Medical Education

An update on the management of age-related macular degeneration will take place on 7–8 June 1996 at the Johns Hopkins University School of Medicine, Johns Hopkins Medical Institutions, Baltimore, Maryland, USA. Further details: Office of Continuing Medical Education, Johns Hopkins Medical Institutions, Turner 20, 720 Rutland Avenue, Baltimore, MD 21205–2195, USA. (Tel: (410) 955–2959; Fax: (410) 955–0807.)

International Society of Dacryology

The IVth International Congress of the International Society of Dacryology will be held in Stockholm on 9–11 June 1996. Further details: Dr G B van Setten, St Eriks Eye Hospital, Fleminggatan 22, S-112 82 Stockholm, Sweden.

The Brian Harcourt Memorial Symposium

The 7th Brian Harcourt Memorial Symposium will take place on 2 July 1996. The symposium topic will be glaucoma. Further details: Mr Mitchell Ménage, Eye Department, Leeds General Infirmary, Clarendon Wing, Belmont Grove, Leeds LS2 9NS. (Tel: 0113 243 2799; Fax: 0113 292 6479.)

International Congress New Developments in Ophthalmology 1996

An international congress on 'New developments in ophthalmology' will be held on 29–31 August 1996 in Nijmegen, the Netherlands. Further details: Professor dr AF Deutman/Mrs Y Hennink, University Hospital, Department of Ophthalmology, PO Box 9101, 6500 HB Nijmegen, the Netherlands. (Tel: (31)24 361 5105; Fax: (31)24 354 0522.)