Correspondence

Persistent choroidal detachment

SIR, Idiopathic uveal effusion syndrome (IUES) is characterised by serous detachment of the peripheral uvea in healthy, middle aged individuals frequently with associated bullous, non-rhegmatogenous retinal detachment. Intraocular surgery is a more common cause of choroidal detachment. We report the persistence of choroidal detachment after cataract extraction in a patient with documented IUES.

Case report

A 63-year-old black man with minimal background diabetic retinopathy (BDR) OS presented to the North Central Bronx Hospital eye clinic in November 1979 with a complaint of decreased visual acuity for two months. Examination was significant for corrected visual acuity of 20/20 OD and 20/100 OS with a spectacle correction of +2-25 sphere OD and +2-50 sphere OS. The left vitreous had a mild cellular reaction. The left fundus was characterised by a bullous, non-rhegmatogenous retinal detachment with an associated choroidal effusion (Fig. 1). Fluorescein angiography OS revealed retinal pigment epithelial changes, mild optic disc oedema, and mild BDR with macular oedema. There was late staining of the bullous retinal elevation. Ultrasonography OS disclosed vitreous band formation, a choroidal detachment, a possible retinal detachment, and thickening of the choroidal layer. No intraocular or retrobulbar mass was identified, and a diagnosis of IUES was made. The exudative detachment persisted OS and did not improve with subtenon methyl prednisolone (Depo-Medrol) 40 mg administered one month after presentation. A gradual spontaneous reduction in the bullous detachment was observed, with complete resolution of IUES over one year without additional therapy.

No further effusion was noted, but progressive posterior subcapsular cataractous changes occurred OU over the following five years. Extracapsular cataract extraction with a posterior chamber pseudophakos was performed OD five years after the spontaneous resolution of IUES in the contralateral (OS) eye. On the ninth postoperative day the patient developed a 360° postoperative choroidal detachment OD without evidence of hypotony or wound leak. The choroidal detachment persisted (Fig. 2), and five months postoperatively B ultrasonography confirmed the persistent 360° choroidal elevation (Fig. 3).
Cataractous changes OS progressed, and extracapsular cataract extraction was performed nine months after cataract extraction OD. The postoperative course was uncomplicated OS despite the previously documented history of IUES. The postoperative choroidal detachment OD has persisted for a total of nine months.

Discussion

The persistence of choroidal detachment after cataract extraction five years after spontaneous resolution of contralateral IUES raises several issues relevant to the pathogenesis of these entities. The unusual persistence of a postoperative choroidal detachment OD supports the possibility of predisposing anatomical factors in the development of uveal effusion. Brockhurst\(^1\) classifies uveal effusions as inflammatory, hydrodynamic, and idiopathic. Post-surgical hypotony and/or inflammation may precipitate uveal effusion in anatomically predisposed eyes. Such anatomical features have an apparent role in development of uveal effusion in patients with nanophthalmos.\(^1\) Surgical inflammation and hypotony may precipitate persistent uveal effusions in nanophthalmic eyes with a known anatomical abnormality. Gass\(^2\) suggests that IUES eyes are also predisposed because of congenitally thickened sclera. A thickened sclera apparently predisposes the eye to vortex vein obstruction and more importantly acts as an abnormal barrier to the transport of protein out of the eye by way of the uveoscleral pathway. These important underlying anatomical abnormalities have formed the basis for surgical attempts to cure IUES\(^2\) and nanophthalmic uveal effusions.\(^3\)

While nanophthalmic eyes have been proved to have thickened sclera,\(^4\) not all IUES sclera have been demonstrated to be thickened.\(^5\) Our patient was not nanophthalmic, and no other predisposing factors were apparent. It seems obvious that there was a predisposition to effusion in this case, but its nature remains obscure.

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References

Obituary

T Keith Lyle, CBE, MA, MD, M Chir, FRCP, FRCS

Thomas Keith Lyle died on 9 May at the age of 83. He was the son of Herbert Willoughby Lyle, MD, FRCS, who was an ophthalmic surgeon at King’s College Hospital and at one time dean of the Medical School. Educated at Dulwich College and Sidney Sussex College, Cambridge, where he was an exhibitioner, Keith Lyle undertook his medical training at King’s College Hospital, where he was an outstanding undergraduate, winning the Todd medal for clinical medicine.

He was in some doubt as to which branch of medicine he wished to follow and considered neurology as a possible choice but settled eventually for ophthalmology, receiving his early training at the Royal Westminster Ophthalmic Hospital. He was appointed to the consultant staff of the Royal Westminster Ophthalmic Hospital and of the National Hospital, Queen Square, in 1936 and to the consultant staff of King’s College Hospital in 1938.

His career was interrupted by the outbreak of war, and as he had been made a civil consultant in ophthalmology to the RAF in 1934 he found himself in uniform immediately at the outbreak and served until the close of hostilities, during which time he was mentioned in dispatches. He reached the rank of temporary air commodore.

Keith Lyle took an early interest in the Faculty of Ophthalmology and became its president in 1968. He was