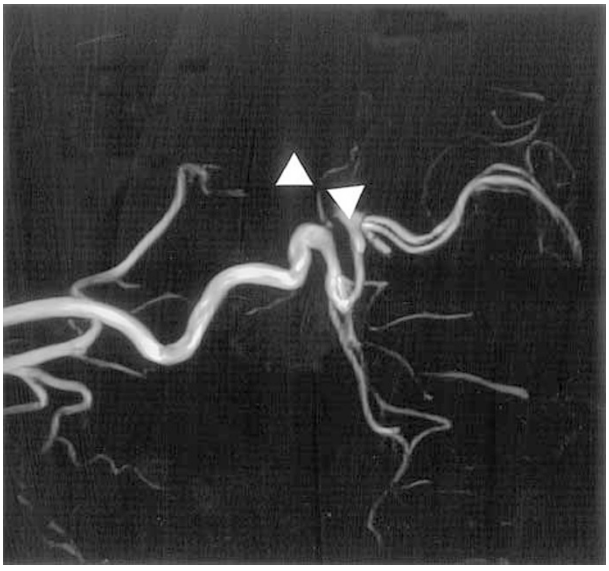

Figure 1 Magnetic resonance angiography showing stenosis of the ophthalmic artery 2 cm from the globe—marked with arrow.

Discussion

External beam radiotherapy[1] is an effective and established treatment for retinoblastoma. Radiation-induced cataract,[4] radiation retinopathy, mild keratopathy, xerophthalmia,[2] and cosmetic facial deformity[3] have all been reported following the treatment. Secondary orbital and facial nonretinoblastoma malignancies (Meningioma of the right temporal lobe as seen in the present case) in these patients are mostly radiation induced.[5] Stenosis of the intracranial arteries[6] following irradiation for craniopharyngioma, and that of vertebral arteries in the neck after X-ray treatment in childhood[7] are well known; however, stenosis of the intraorbital ophthalmic artery secondary to external beam irradiation for retinoblastoma has not been reported before. The mechanism of stenosis appears to be similar as seen in other vessels.[6] A combination of a stenosed artery, which we presume resulted from radiation-induced endothelial damage combined with an episodic drop in blood pressure during the general anaesthetic for lipoma excision, precipitated the retinal infarct in our patient.

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Sir,

An unusual case of corneal perforation with crystalline lens extrusion secondary to pseudomonas keratitis in the presence of rheumatoid arthritis

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A common complication of rheumatoid arthritis (RA) is dry eye, which can compromise the ocular surface and predispose it to infective keratitis. Both RA and infective keratitis can lead to corneal melt but this rarely results in the extrusion of intraocular contents. We report a case of spontaneous lens extrusion in a patient with dry eyes and infective keratitis.

Case report

A frail 96-year-old lady with previously well-controlled RA and dry eye presented to her GP with a 3-day history of a red and gritty dry eye. There had been no perception of light in this eye for several years due to rubeotic glaucoma secondary to central retinal vein occlusion.

Conjunctivitis was diagnosed and topical fucithalmic produced an encouraging initial response. After 6 days, she presented to eye casualty holding her spontaneously extruded crystalline lens (Figure 1). Examination revealed a corneal abscess with a considerable area of corneal melt resulting in a large open wound exuding pus (Figure 2).

Corneal cultures identified a heavy growth of *Pseudomonas aeruginosa* that had probably been contracted from a hospitalisation for gastrointestinal bleeding 2 weeks prior. Management with topical and systemic antibiotics to prevent spread of infection was instigated. The eye settled and became phthisical.

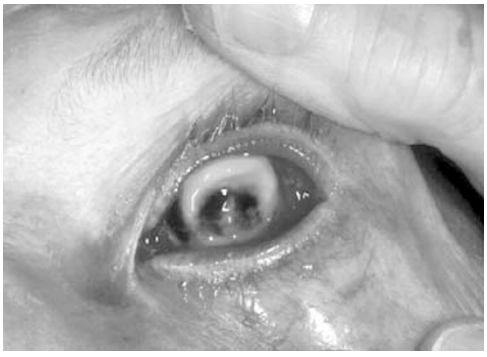

Figure 2 Total central corneal melt through which the crystalline lens was extruded. Exuding pus with prolapsed uveal tissue also present.

Comment

In the context of RA, corneal melting and perforation are more likely in the presence of microbial keratitis and dry eyes.[1] Although *P. aeruginosa* cannot penetrate intact epithelium, once it does infect the cornea it can release a host of proteolytic enzymes and toxins causing additional direct lytic damage to the collagenous structure of the cornea. Untreated this can lead to rapid ulceration, stromal necrosis, and perforation.[2]

Lens expulsion is an uncommon complication of corneal melting. It has been previously reported in association with Kwashiorkor,[3] in premature infants,[4] in an obtunded patient with nosocomial keratitis,[5] and after anterior chamber intraocular lens implantation in the presence of concurrent illness such as RA, herpes zoster ophthalmicus, and glaucoma.[6,7]

Figure 1 Spontaneously extruded crystalline lens.

In an increasingly ageing population, it is of utmost importance that early signs of corneal involvement in RA patients are recognised and treated promptly. This is especially true of the debilitated or recently hospitalised elderly. This is the only reported case we found that shows corneal melting allowing extrusion of the crystalline lens in a patient with RA during infective keratitis.

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Sir,

Central retinal vein and ophthalmic artery occlusion in primary antiphospholipid syndrome

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Primary antiphospholipid syndrome is characterized by the production of moderate to high levels of antiphospholipid antibodies, associated with thrombotic phenomena (arterial or venous) and recurrent spontaneous abortion due to placental vascular insufficiency, in the absence of any other recognizable autoimmune disease.[1]

Primary antiphospholipid syndrome has been found to be associated with ocular ischaemia and retinal vascular occlusion, due to arteriolar or venular thrombosis.[2–6] We report a case of a 60-year-old woman with primary antiphospholipid syndrome, who developed consecutive occlusion of the central retinal vein and ophthalmic artery. This is the first reported case in patients with this syndrome.

Case report

A 60-year-old woman presented with a 1-day history of slurred speech and left-sided weakness. She also described an episode of transient dimming of vision in the right eye that lasted 4 h. She had a history of mild hypertension and noninsulin-dependent diabetes mellitus that was diagnosed 6 months before, both of which were adequately controlled by lifestyle modification and dietary measures alone. Neurological examination revealed a left-sided haemiparesis and a left upper motor neuron facial nerve palsy. Visual acuity was 6/7.5 OU. Funduscopic examination was normal. Magnetic resonance imaging of the brain demonstrated the presence of a right corona radiata infarct. Duplex carotid ultrasonography showed moderate dampening of the velocity and waveform of the right internal carotid artery blood flow compared to the left. The patient was started on ticlopidine.

After 5 months, she presented with a sudden reduction in vision acuity in the right eye to 6/18. Intraocular pressures were normal. Fundus examination revealed a right central retinal vein occlusion. She was screened for autoimmune and procoagulant disorders. Pertinent laboratory investigations were as follows: the erythrocyte sedimentation rate was 57 mm/h; IgG anticardiolipin was raised to 33 GPL units/ml; antinuclear antibody and lupus anticoagulants were negative. Serum total complement, C3, C4, and protein S, protein C and antithrombin III were normal. The diagnosis of primary antiphospholipid syndrome was established and the patient was started on anticoagulation with warfarin.

After 1 month, she presented with sudden deterioration of her right vision to perception of light only. A right relative afferent pupillary defect was present. There was neovascularization of the iris, with a raised intraocular pressure of 26 mmHg. Fundus examination revealed the presence of a right ophthalmic artery occlusion, with severe whitening of the retina and markedly attenuated retinal arterioles and venules (Figure 1). Fundus fluorescein angiography demonstrated blocked choroidal and retinal artery filling. Transcranial doppler ultrasound of the ophthalmic arteries revealed an absent signal over the right ophthalmic artery and a normal signal from the left. The international normalized ratio (INR) was 1.6. Immediate panretinal photocoagulation was instituted. She was started on oral acetazolamide 250 mg bid and topical timolol 0.5% bid and atropine 1% tid to the affected eye. Warfarin therapy was optimized to INR 2.5–3.0. There was gradual resolution of the iris neovascularization and eventual normalization of the intraocular pressure. However, the final visual acuity in the right eye was hand movements only. She remained asymptomatic in the left eye.

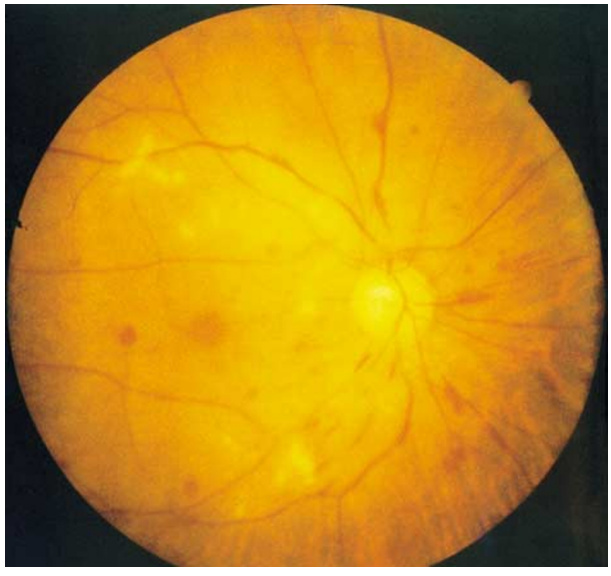

Figure 1 Fundus photograph of the right eye following central retinal vein and ophthalmic artery occlusion.