signs and are less likely to harbour occult ocular or systemic pathology than children with high myopia.

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JE Marr, R Harvey and JR Ainsworth

Department of Paediatric Ophthalmology
Diana Princess of Wales Children's Hospital
Steelhouse Lane
Birmingham B4 6NH
UK

Correspondence: JR Ainsworth
Tel: 0121-333-9464
E-mail: j.r.ainsworth@bham.ac.uk

Sir,

Choriovitreal neovascularization following transpupillary thermotherapy for choroidal melanoma

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Transpupillary thermotherapy (TTT) has recently emerged as a first-line treatment modality for some selected small posterior uveal melanomas.[1] This is largely because of the relative ease of performing the procedure on an outpatient basis, reduced costs compared with other modalities and perhaps, most important of all, few side effects and collateral damage depending on the location of the tumour. Currently, tumours posterior to the equator and measuring less than 10 mm in basal diameter and less than 3.5 mm in thickness can be predictably and safely treated with TTT.[1] We herein report a patient with a small choroidal melanoma treated with TTT, who shortly after the treatment developed choriovitreal neovascularization that led to a chain of disproportionately severe complications.

Case report

In June 1999, a 63-year-old woman presented with gradual loss of vision in the left eye within 6 months. Except for chronic systemic hypertension, she was in good health. On examination, her best corrected visual acuity was 20/25 in the right eye and 20/60 in the left. Intraocular pressures and anterior segments were within normal limits except for the presence of 2+ nuclear sclerosis in the left eye. Left fundus examination revealed a circumscribed, partially amelanotic choroidal melanoma that measured $6.5 \times 6.5\,mm^2$ in basal dimensions and 3.5 mm in thickness (Figure 1). There was no overlying subretinal fluid. We performed TTT with confluent 3 mm spots at 550 mW power, each lasting 60 s. The procedure was repeated 6 months later with the same settings at 600 mW power. The tumour steadily regressed during the following 10 months until the patient presented with a mild vitreous haemorrhage. Her left visual acuity dropped to counting fingers. Choriovitreal neovascularization originating from the tumour was observed (Figure 2). The patient then was temporarily lost to follow-up. When she presented again in June 2001, a dense vitreous haemorrhage precluded any fundus view. A standard three-port vitrectomy was performed with sector endolaser photocoagulation also surrounding the tumour. The neovascular fronds rapidly regressed into fibrotic sheaths while the tumour thickness decreased to 1.5 mm. The visual acuity stabilized at 20/100. No tumour recurrence or metastasis was noted during later follow-up.

Comments

Infrared thermotherapy induces tumour cell necrosis, probably by disruption of intracellular mitochondria, through raising the local temperature between 45 and 60°C.[2] Histopathological studies on eyes following TTT showed necrosis into a depth of 3.9 mm within the tumour and scattered haemorrhages between the necrotic and viable parts.[3] However, no evidence of choroidal neovascularization was mentioned in this

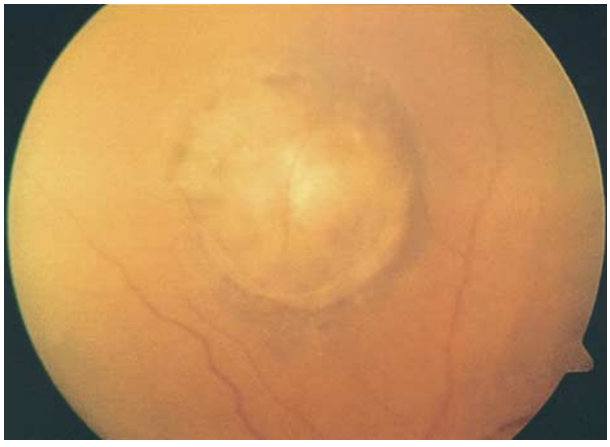

Figure 1 Choriovitreal neovascularization: overall pretreatment view of the choroidal melanoma that is located 12 mm superior to the optic disc.

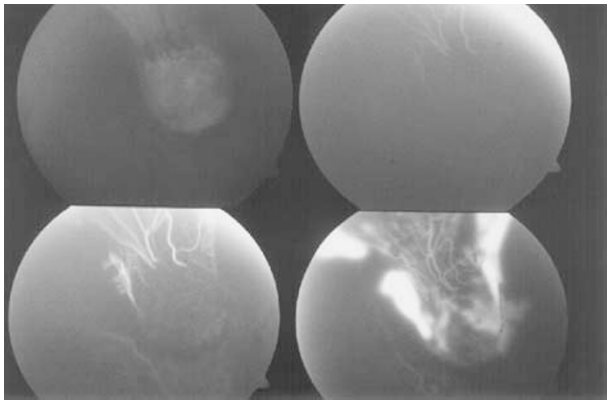

Figure 2 Choriovitreal neovascularization. The newly formed vessels extend from the anterior part of the tumour into the vitreous (upper left). The abnormal vessels become apparent as early as the choroidal circulation phase of fluorescein angiography (upper right). The early arterial filling phase demonstrates the full extent of neovascularization (lower left) that leaks profusely in the mid-venous phase (lower right).

report. A recent study suggested that occlusion of choriocapillaris occurred in treated areas with choroidal vascular remodelling in 31% of the cases.[3] In 10% of the cases, retinochoroidal anastomosis was the most prominent feature.[4]

Retinal vascular occlusions and retinal traction were identified as the most significant vision-threatening side effects of TTT.[5] In fact, an analysis of 256 patients treated with TTT showed that retinal traction (44%) followed by branch retinal vein obstruction (41%) and branch artery obstruction (12%) were the most common complications.[1] Neovascularization of the retina was seen in 6% of the cases.[1] Other series found a similar incidence of side effects and also reported focal iris atrophy, preretinal fibrosis, cystoid macular oedema, disc oedema, nerve fibre bundle field defects, arteriolar sheathing, vitreous haemorrhage, subretinal and choroidal haemorrhages.[5–8]

Retinal neovascularization, as reported in the literature, results from occlusion of retinal vessels leading to ischaemia of the involved area. In our case, there was no retinal vascular occlusion and therefore no retinal neovascularization but choroidal neovascularization that occurred over the tumour suggesting direct damage to Bruch's membrane. Since amelanotic melanomas have poor heat absorption, we used relatively high powers to produce faint grey spots that were necessary to destroy the tumour in our judgement. This might have induced choriovitreal neovascularization. In a patient similar to ours, a chorioretinal anastomosis with a localized intravitreal neovascularization over the treated area developing 1 year after TTT was reported.[8] This case was managed by additional TTT and sector panretinal photocoagulation.[8] Given the dimensions of the tumour and other favourable associated findings, we had every reason to expect an uneventful tumour regression following TTT. At this juncture, our decision to employ TTT should be questioned. Perhaps a low-energy ruthenium-106 plaque would have been preferable, and we now believe that this would cause considerably less morbidity. This patient once again demonstrates that TTT is not an innocent procedure, and unexpectedly severe complications may develop in cases that initially appear as straightforward or 'routine'.

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H Kiratli and S Bilgiç

Ocular Oncology Service
Department of Ophthalmology
Hacettepe University School of Medicine
Ankara, Turkey

Correspondence: Hayyam Kiratli
Hacettepe Hastanesi Goz ABD
Sihhiye, 06100 Ankara, Turkey
Tel: +90 312 309 4101
Fax: +90 312 305 1790
E-mail: hkiratli@hacettepe.edu.tr

Sir,

Massive spontaneous expulsive suprachoroidal haemorrhage in a blind glaucomatous eye treated with chronic topical steroid

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Expulsive suprachoroidal haemorrhage is a rare event, usually complicating intraocular surgery, with often devastating results. There are few reports of spontaneous expulsive haemorrhage in the literature. We present a case of spontaneous expulsive choroidal haemorrhage and highlight potential aetiololgies.

Case report

A 74-year-old lady presented as an emergency with a history of severe pain of sudden onset and bleeding from her left eye while sitting at home. She described enough blood to fill two large tissues trickling onto her cheek. Her medical history included systemic hypertension, chronic renal failure, gout, and hypothyroidism.

On examination, she had no light perception in the affected eye and 6/6 in the fellow eye. There was extensive prolapse of tissue through a large corneal defect onto the left cheek with blood-clot admixed (Figure 1). The right eye was normal apart from a shallow anterior chamber and a peripheral surgical iridectomy.

Twenty years earlier, she had a broad surgical iridectomy after admission for left acute-angle closure glaucoma, plus contralateral prophylactic peripheral iridectomy. The patient was lost to follow-up for 17 years, when she re-presented with failing painless vision in her left eye over several weeks. On this occasion she had perception of light only, corneal oedema, a shallow anterior chamber, a large cataractous lens, a raised intraocular pressure (IOP) of 44 mmHg, a closed drainage angle, a left afferent papillary defect, and a grossly cupped optic disc. Her other eye saw 6/6 with a pressure of 13 mmHg and a healthy optic disc. After intense medical treatment failed to control her IOP, ocular comfort was maintained with a drop regime of dexamethasone 0.1% b.d. and timolol 0.25% b.d. Unfortunately she was again lost to follow-up shortly with persistently raised IOP but a comfortable eye and a clear cornea, until her spontaneous expulsive choroidal haemorrhage.

An evisceration with a 16 mm acrylic ball implant was performed. Histology confirmed the extent of corneal perforation with stromal infiltration by neutrophils but no identifiable organisms. The iris was degenerate with necrotic uveal tissue and blood clot. There was no evidence of malignancy or vasculopathy.

Comment

There are few reports of spontaneous corneal perforation with expulsive haemorrhage in the literature.[1] Our patient had multiple predisposing factors for this dramatic event, including advancing age, arteriosclerosis, systemic hypertension, persistently raised IOP, and chronic use of topical steroid. De Laage[2] reported a case of giant corneal perforation secondary to chronic misuse of local steroid to the eyelids. We feel that this was the most likely trigger factor in our patient, resulting in a sudden reduction in the IOP and massive suprachoroidal haemorrhage in the presence of multiple other predisposing factors. Although the cornea was noted to be clear at her last assessment, 2 years elapsed before she presented with spontaneous expulsive suprachoroidal haemorrhage, and bullous keratopathy may have ensued in that time in an eye known to have a chronically raised IOP. It is unlikely, however, that bacterial keratitis would have complicated pre-existing bullous keratopathy as non identifiable organisms were noted on histological examination. Of note, there was no evidence of keratopathy in the fellow eye that could predispose to perforation,[3] and a vasculitis screen was

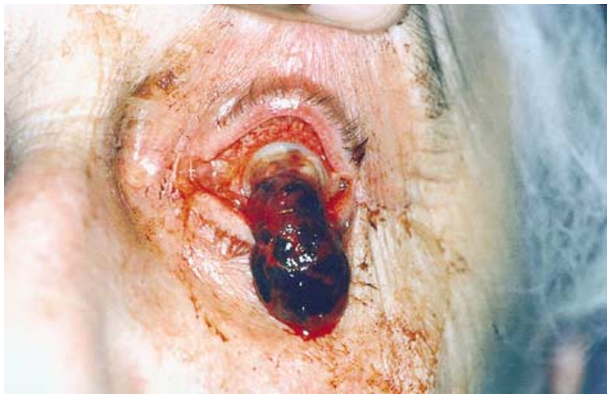

Figure 1. Acutely prolapsed oveal contents from left eye.