related to the passage of the metallic fragment as it passed into the eye. A number of possible mechanisms for this can be postulated. Firstly the low pressure zone immediately following a high velocity projectile would draw air in its wake down an induced pressure gradient. This could have resulted in air being drawn into the eye, although the absence of air at the entry site or related to the path between this and the presumed site of impact is against this. The second possibility is that the bubbles represent the result of cavitation induced by the passage of the solid fragment through the semi-liquid vitreous medium.[4] A low pressure region in the wake of a fast moving object can result in dissolved gases coming out of solution. Again though, it is odd that they are solely related to the final resting site of the fragment. Thirdly, a thermal or chemical reaction between the fragment and the vitreous gel could result in the liberation of free gas, and this cannot be excluded.

It therefore remains uncertain as to the exact mechanism that induced this interesting phenomenon. The authors would be pleased to hear of any further cases where similar findings were observed.

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Sir,

Palliative vitrectomy for intraocular metastasis from cutaneous melanoma

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Primary cutaneous melanoma metastatic to the eye is rare.[1–4] Still rarer is melanoma metastasis to the vitreous cavity. A review of the literature showed only seven such cases—Robertson *et al*[5] (two cases), Cole *et al*[6] (one case) and Gunduz *et al*[7] (four eyes of three patients). Diagnosis in one was by primary enucleation for a painful blind eye.[6] Two underwent fine needle aspiration biopsy and three had diagnostic vitrectomy. Neither external beam radiotherapy nor chemotherapy was successful in achieving tumour control, with these eyes ultimately being lost as a result of neovascular glaucoma. Non-ocular systemic metastatic disease was usually present at the time of ocular diagnosis. The patients tended to develop cerebral metastases and had a mean survival of 5 months from the time of development of ocular metastases.[5,7,8] The present case shows how vitrectomy may have a therapeutic role by providing functional vision and preventing neovascular glaucoma from developing.

Case report

An 87-year-old Caucasian lady was referred with a history of floaters and progressive decrease in vision in the left eye over a 4-week period. She had previously been operated in both eyes for cataract and had good postoperative vision.

She had undergone excision of a cutaneous melanoma from the right cheek 15 months earlier. This was followed by radical neck dissection and radiotherapy. Six months before her ophthalmic presentation, several metastatic lesions were excised from the back of the neck, left arm and anterior abdominal wall.

On examination, the visual acuity was 6/6 in the right eye and hand movements in the affected left eye. Anterior segment examination showed bilateral, quiet pseudophakia. Numerous pigmented clumps were seen on the anterior hyaloid face. The vitreous cavity showed multiple brown cannon-ball opacities. No retinal details were discernible. B scan ultrasonography showed several echogenic points in the vitreous cavity with a flat retina. No choroidal thickening was seen.

The patient underwent a diagnostic and therapeutic pars plana vitrectomy under local anaesthesia. The vitreous cavity showed a dense collection of brown cannon-ball opacities. No choroidal or retinal masses

were seen. Histology of the vitreous sample showed small clusters of non-pigmented epithelioid cells with variable amounts of eosinophilic cytoplasm and anisonucleosis (Figure 1). The cells were HMB45 positive, cytokeratin and CD45 negative, consistent with melanoma cells. Postoperatively, the visual acuity improved to 6/24 with correction. Her affected eye retained this level of vision and remained comfortable thereafter with no evidence of recurrence.

A few days after vitrectomy, the patient developed weakness of her right arm caused by multiple cerebral metastases, which were confirmed by computerised tomography. She was given palliative whole brain radiotherapy consisting of 20 Gy in five fractionated doses and a course of prednisolone 5 mg twice a day. She died of metastatic disease 3 months after vitrectomy.

Discussion

The present case is interesting because the vitrectomy not only achieved a diagnosis but also conserved a comfortable and seeing eye until the patient died a few months later.

Previously reported cases have generally not done so well. Gunduz *et al*[7] treated four eyes of three patients with external beam radiotherapy, three of which continued to develop progressive disease leading to neovascular glaucoma. The fourth eye retained 20/20 vision. Robertson *et al*[5] reported two cases, which were treated with chemotherapy. Both developed neovascular glaucoma with one requiring enucleation for a painful blind eye. Such case reports suggest that neither radiotherapy nor chemotherapy is successful in preventing loss of vision and neovascular glaucoma in these patients. Radiotherapy is unsatisfactory also because the prolonged course of hospital treatment severely disrupts the patient's last few months of life. Chemotherapy can cause systemic complications, which may be serious.

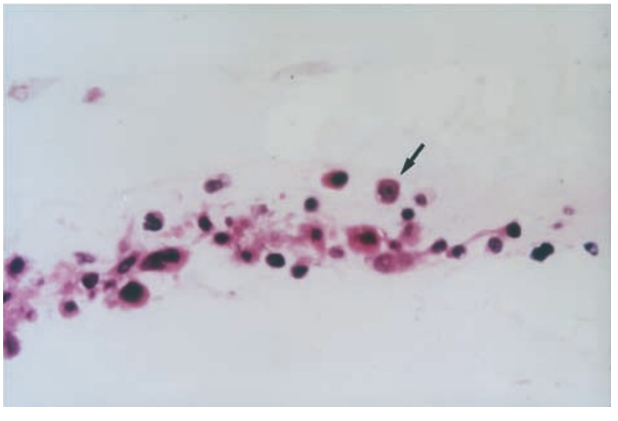

Figure 1 Haematoxylin & Eosin stain of vitreous sample showing clusters of non-pigmented epithelioid cells with variable eosinophilic cytoplasm and anisonucleosis. The arrow shows one cell with a large, prominent central nucleolus in the nucleus. (× 400).

The vitrectomy we performed was done under local anaesthesia taking only about 30 minutes, and could have been repeated, if necessary, as an out-patient procedure. It was successful in avoiding the development of a painful eye and the need for enucleation. The vitrectomy also improved the vision from hand movements to 6/24. Although our patient had good vision in the fellow eye, the improvement we achieved would have been extremely valuable if the fellow eye had been abnormal.

It would have been interesting to confirm the beneficial effects of vitrectomy in a series of patients. However, vitreous metastases are rare, with only a handful of cases having been reported in the world literature.

In conclusion, if a diagnostic vitrectomy is required in a patient suspected of having vitreous metastases from cutaneous melanoma, it would seem reasonable to perform a total vitrectomy, which may conserve a comfortable seeing eye for the patient's limited life span without the need for enucleation or radiotherapy.

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Sir,

Bilateral acute angle-closure glaucoma after use of Fenox™ nasal drops

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Fenox™ nasal drops are an over-the-counter nasal decongestant preparation which contain phenylephrine and naphazoline, both adrenergic sympathomimetics. We describe a case of bilateral acute angle-closure glaucoma (AACG) presenting following the use of Fenox™ nasal drops.

Case report

A 53-year-old taxi driver presented to the Eye Casualty Department with a one-week history of painful eyes, worse in the right, associated with photophobia and haloes. He had been suffering from symptoms of a cold and sinusitis and had been using Fenox™ nasal drops regularly for the previous week. His past medical history included an operation for a deviated nasal septum. He was otherwise well and was on no other medication. He was a low hypermetrope, wearing right: + 1.00 DS and left: +0.50 D/+0.50 D at 100°.

Examination revealed an unaided visual acuity (VA) of counting fingers with his right eye and 6/36 with his left. Both eyes were injected with corneal oedema, shallow anterior chambers and fixed, mid-dilated pupils. Both crystalline lenses exhibited glaucomflecken. His intraocular pressure (IOP) was 38 mmHg in the right eye and 28 mmHg in the left. Gonioscopy was impossible in the right eye and only the inferior 180° of the left was visible, which was closed.

A diagnosis of bilateral AACG was made and he was treated with intravenous acetazolamide and topical pilocarpine 4% and dexamethasone to both eyes. Four hours later, both eyes remained painful. Only his left pupil had miosed and his IOP was 42 mmHg (right eye) and 06 mmHg (left). He was commenced on oral acetazolamide plus topical timolol 0.5% and latanoprost to his right eye, in addition to his other topical agents. A further dose of intravenous acetazolamide was also required.

The following day, his symptoms had resolved. His IOP had fallen to 15 mmHg in his right eye and 04 mmHg in his left and systemic treatment was stopped. Indentation gonioscopy confirmed that both drainage angles were closed but no peripheral anterior synechiae were observed. The IOPs remained satisfactory on topical medication alone and a left Nd:YAG laser iridotomy was performed 2 days later. Treatment to the left eye was then reduced to topical steroids only. Eight days later, his IOP was 11 mmHg in his right eye and 13 mmHg in his left, although little miosis had occurred in the right eye. Topical treatment was further reduced to pilocarpine 4% in the right eye and dexamethasone to both. IOP control remained satisfactory for a further 4 days, when a right Nd:YAG laser iridotomy was performed. All ocular hypotensive medications were stopped and his topical steroids were tapered off over a 3-week period. At review 6 weeks after his initial presentation, his unaided VA had improved to 6/6 in each eye. The IOP was 15 mmHg in his right eye and 14 mmHg in his left off all treatment. Both iridotomies were patent with deepened anterior chambers, although both irides showed evidence of ischaemic damage and the glaucomflecken persisted. At final review 8 months after presentation, his IOP remained normal. His optic discs did not show any pathological damage and his visual fields were full. His nasal symptoms had resolved following further surgery.

Comment

Many drugs have been reported to precipitate AACG, either by direct or indirect sympathomimetic activity. Diagnostic mydriatics[1,2] as well as systemic agents such as tricyclic antidepressants,[3] serotonin re-uptake inhibitors,[4] diuretics,[5] inhaled bronchodilators,[6] intranasal cocaine[7,8] and other proprietary medicines[9] have all been implicated. Fenox™ nasal drops contain two direct adrenergic sympathomimetic agents, phenylephrine and naphazoline, producing vasoconstriction of the skin and reducing congestion when applied to mucous membranes. The recommended frequency of use for Fenox™ nasal drops is morning and night and every 4 hours if necessary, and for no longer than 7 days (manufacturer's instructions).

To the best of our knowledge, this is the first