

markers and no systemic immunodeficiency disorder. She had reduced numbers of B cells and tests of T cell function showed a global reduced response to stimulation by antigens. The CMV retinitis probably resulted from long term oral corticosteroid use.

CASE REPORT

A 39-year-old woman presented in November 1994 to the Accident and Emergency Department of the Birmingham and Midland Eye Hospital with a recent onset of a shadow and floaters in the temporal field of her vision of her left eye.

She had previously attended in October 1993 with an unusual appearance of the temporal periphery of the right retina. This lesion was thought to be a retinoschisis and was adjacent to a region of chorioretinal atrophy. There was no evidence of inflammation in the vitreous or retina. The schisis was observed but a retinal detachment subsequently occurred and she underwent a vitrectomy in September 1994 following failure of conventional detachment surgery. A devastating *Haemophilus influenzae* endophthalmitis developed 3 days after surgery. Despite treatment with systemic and intravitreal antibiotics, and prednisolone 60 mg per day the eye became blind and phthisical. The corticosteroid dose was gradually reduced to 15 mg over the next 2 weeks.

She was a severe asthmatic who had suffered from recurrent bouts of lower respiratory tract infection and had been on systemic prednisolone continuously for the last 17 years. She had undergone many acute admissions to hospital for her asthma, necessitating short term increases in her corticosteroid therapy. This was gradually reduced to a maintenance dose of 5 mg per day. She presented to us 2 months after the episode of endophthalmitis affecting her right eye and was still on oral prednisolone 15 mg per day. She had active cold sores on her lips and in her left nostril. She did not appear malnourished.

Visual acuity was 6/9 in the left eye. There was a mild anterior uveitis, with a 1+ cells in the anterior chamber and fine keratic precipitates on the inferior cornea. There was 1+ of cells in the vitreous. Ophthalmoscopic examination showed a small, circumferential white area in the inferonasal periphery of the retina associated with sheathing of retinal arterioles and scattered haemorrhages (Fig 1).

The lesion in the nasal periphery of her left retina slowly progressed and a provisional diagnosis of acute retinal necrosis was made. Treatment with intravenous acyclovir, 10 mg/kg three times a day, was given and indirect laser photocoagulation applied around the lesion.

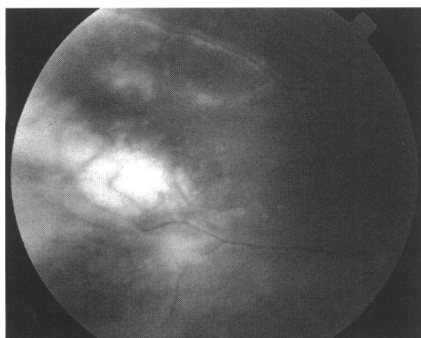


Figure 1 Necrotising retinitis in nasal periphery of left eye.

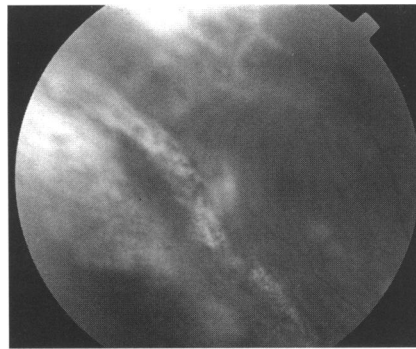


Figure 2 A recurrence detected superior to original lesion and laser photocoagulation scars.

After an initial improvement a recurrence resembling CMV retinitis was detected superior to the original lesion (Fig 2). An anterior chamber tap was performed. Polymerase chain reaction testing for herpesviral DNA detected CMV DNA in the aqueous humour but was negative for varicella zoster and herpes simplex type 1 and 2 viral DNA.

She was induced with intravenous ganciclovir at a dose of 5 mg/kg, twice daily for 2 weeks. Maintenance therapy was oral ganciclovir at a dose of 1 g three times per day, three times a week, resulting in resolution of the retinitis. Over the next 12 months the oral ganciclovir was reduced to a once a week dosage with no recurrence of the retinitis.

Investigations showed a normal full blood count, serum biochemistry, and serum immunoglobulins. Anticardiolipin, antinuclear and antinuclear cytoplasmic antibodies were all negative. Her HIV antibody test was also negative. Peripheral blood B cell numbers were reduced to $0.02 \times 10^9/l$ ($0.22 \times 10^9/l$ in a healthy control). She had normal T cell numbers and subtypes with her serum CD4 count being $1.18 \times 10^9/l$ ($0.48 \times 10^9/l$ in a healthy control). Her T cells responded normally to concanavalin A, phytohaemagglutinin, and pokeweed mitogen stimulation, but poorly to purified protein derivative, *Candida albicans* and herpes simplex viral antigens. The global nature of the antigen response defect suggested that it was secondary to the corticosteroid therapy. These results implied that the CMV retinitis resulted from long term oral corticosteroid use.

COMMENT

Although the commonest intraocular infection seen in AIDS patients, CMV retinitis is also a recognised complication of immunosuppressive therapy,¹ particularly after organ transplantation. Cyclosporin, azathioprine, and cyclophosphamide have an intermediate effect and corticosteroids a minimal effect in inducing CMV disease.² Patients with rheumatological disorders on long term immunosuppression may develop a rapidly progressive CMV retinitis.³

There have been two reports in the literature of CMV retinitis in 'immunocompetent' patients.^{4,5} It was found to be reversible without treatment in one case,⁴ and in the other it was attributed to exposure to chronically sick children.⁵ No test results of immune function were given and the diagnosis was made on serological investigations alone.

A case of culture proved cytomegalovirus panuveitis involving both eyes of a previously healthy young woman receiving immunosuppressive doses of corticosteroids has been reported.⁶ The virus was isolated from subretinal fluid obtained during retinal detach-

ment surgery. The corticosteroids were given following craniotomy for evacuation of a subacute right subdural haematoma.

Ophthalmologists should now be aware of another potential complication of corticosteroid usage.

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Unilateral choroidal melanoma with bilateral retinal vasculitis

EDITOR,—The association of choroidal melanoma with ipsilateral ocular inflammation is recognised and has most frequently been noted in those with tumour necrosis and mixed or epithelioid cell types.¹ Sympathetic ophthalmitis has also been observed with necrotic choroidal melanomas, often with extrascleral extension.² Distant carcinoma associated retinopathy and more recently cutaneous melanoma associated retinopathy have both been reported with an accompanying bilateral retinal vasculitis.^{3,4} We report a case of a 22-year-old man with a left anterior choroidal melanoma in association with a bilateral retinal vasculitis.

CASE REPORT

A 22-year-old man was referred with a 6 week history of a gradually enlarging left nasal field defect in association with bilateral visual floaters and occasional photopsia. He was otherwise fit and well.

Examination revealed 6/6 vision bilaterally. There was a left nasal field defect. The right field was full. Bilateral medium sized keratic precipitates were present with a plus/minus of anterior chamber cells.

There were 1+ of vitreous cells bilaterally. On the left he had a large solid anterior choroidal mass in the temporal periphery with a base and depth of approximately 13 mm. There was some subretinal fluid inferiorly. Bilateral focal retinal venous sheathing was evident with some associated retinal new vessels inferiorly in the right fundus. General medical examination was unremarkable.

A fluorescein angiogram demonstrated mottled hyperfluorescence over the mass. There were focal areas of perivenous leakage in peripheral retinal venules but no capillary closure.

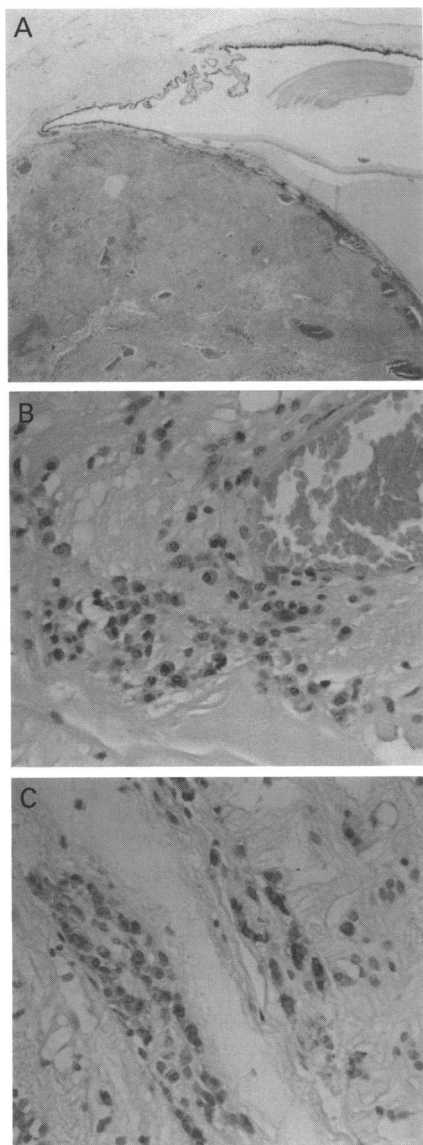


Figure 1 (A) Low power histological section of left eye stained with haematoxylin and eosin showing spindle cell melanoma arising from anterior choroid. (B) High power section of inner retina stained with haematoxylin and eosin showing a capillary in horizontal section with adjacent plasma cells. (C) High power section of inner retina stained with CD45 (common leucocyte antigen) showing a capillary in longitudinal section surrounded by plasma cells.

Initial normal investigations included a full blood count, electrolytes, liver function tests, calcium, toxocara, toxoplasma, and syphilis serology, Mantoux test (6 mm weal with 0.1 ml of 1:1000 tuberculin protein), immunoglobulins, and an autoantibody screen. A chest x ray and liver ultrasound were also normal. Plasma viscosity was raised at 1.84 (normal range 1.64–1.72) and angiotensin converting enzyme was also slightly raised at 113 (normal range 32–84).

A transbronchial lung biopsy was subsequently normal as were bronchial washings.

Differential diagnosis included sarcoid with a choroidal granuloma or a choroidal melanoma with an associated bilateral retinal vasculitis.

A trial of high dose oral steroids was given (80 mg prednisolone for 2 days, 60 mg for 4 days then 40 mg). Over the next 2 weeks vision in the left eye decreased to 2/60 with an increase in the amount of subretinal fluid and no significant improvement in the inflamma-

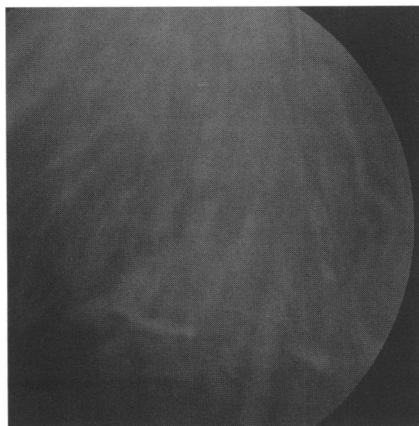


Figure 2 Peripheral fundus photograph of right eye showing areas of inactive perivenous sheathing and regressed neovascular tissue with epiretinal membranes.

tory signs. A biopsy of the mass was therefore performed. This showed a spindle type melanoma.

The eye was enucleated and the steroid dosage tapered rapidly with 30 mg for 2 days, 20 mg for 2 days, 10 mg for 2 days, and 5 mg for 2 days before stopping.

Histological examination of the eye (Fig 1) was performed and routine stains used. There was a large spindle B type melanoma originating from the anterior choroidal area with only occasional mitotic figures. There was no evidence of extrascleral extension or necrosis. The retina was totally detached with plasma cells in the vitreous cavity. Bruch's membrane was intact. Immunocytochemistry was performed. The retina had a polyclonal plasma cell infiltrate, with occasional lymphocytes and macrophages. The plasma cells were concentrated in the inner retinal area around blood vessels. Staining of the plasma cells for immunoglobulins was maximal for IgG and IgM and there was also granular staining of retinal capillary basement membranes for both IgG and IgM. There were no neutrophils suggesting that complement was not activated. There was minimal photoreceptor degeneration consistent with the detached retina. The choroid and lens were normal.

At the 2 week follow up appointment after enucleation there was a dramatic improvement in the inflammatory signs with no keratic precipitates or anterior chamber cells and only a few vitreous cells. The new vessels inferiorly in the right fundus had stopped proliferating and were regressing. At review 1 month later there were no inflammatory cells present in the vitreous or anterior chamber and fibroglial remnants only from the new vessels with no active periphlebitis (Fig 2). There has been no recurrence to date.

COMMENT

The pathogenesis of the inflammatory signs in this case is interesting and there are no other reports of choroidal melanoma in association with a bilateral retinal vasculitis.

There were features consistent with sarcoidosis but non-diagnostic and the histological findings were not consistent with sarcoid. Oral corticosteroids had no effect on the inflammatory signs. These decreased, however, after enucleation despite a rapid withdrawal of the steroids.

The finding of a plasma cell infiltrate in the retina is unusual and differs from the predominantly lymphocytic infiltration described in retinal vasculitis.⁴ The significance of this is

not known but the presence of plasma cells suggests an immunologically mediated process.

Choroidal melanomas are rare in young adult males and retinal vasculitis is also uncommon. The temporal association between these two conditions in this patient would suggest that they were linked and the improvement in the inflammatory signs after enucleation suggests that the melanoma was implicated in their aetiology. It can be postulated that in this case there was cross reactivity between the melanoma and various retinal antigens or, alternatively, that the melanoma enhanced retinal antigen presentation to the immune system, resulting in altered antiretinal autoimmunity and a subsequent retinal vasculitis.

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Amiodarone and dysthyroid eye disease

EDITOR,—The antiarrhythmic drug amiodarone is known to have ophthalmic side effects including the formation of corneal microdeposits and, more rarely, optic neuritis. Among systemic side effects are hypothyroidism and hyperthyroidism. We present a case in which amiodarone appears to have been responsible for an exacerbation of dysthyroid eye disease.

CASE REPORT

A 75-year-old man presented with a 1 week history of reduced right visual acuity and pain in both eyes which was worse on eye movement. He also noticed that colours were less clear with the right eye. Chronic open angle glaucoma had been diagnosed in 1990 and treated with pilocarpine 4% and Betoptic 0.5%. There was a medical history of hypothyroidism and ischaemic heart disease. Two months before the onset of ocular symptoms he had developed an arrhythmia which was treated with amiodarone 200 mg three times daily.

On examination his visual acuity was 6/9 right and 6/6 left. Extraocular movements were full. No defect in colour vision was found in either eye on testing with Ishihara plates. His visual fields were full. The pupils were miosis. The conjunctiva was not inflamed and anterior examination was unremarkable save for elevated intraocular pressures (29 mm Hg right and 23 mm Hg left). The optic discs were asymmetrically cupped with a cup/disc diameter of 0.6 right and 0.4 left. The discs were not swollen and funduscopy was otherwise unremarkable.