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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Accepted for publication 24 May 1996

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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.

Figure 1 The computed tomography scan appearance of the orbit before treatment.

One week later the patient complained of horizontal diplopia. On examination visual acuities were unchanged, but the conjunctiva was diffusely inflamed and chemosed bilaterally. Colour vision was reduced on both sides on testing with Ishihara plates (9/15 right and 8/15 left). There was no proptosis but the eyes felt hard to repulsion on both sides. The intraocular pressures were now 34 mm Hg and 32 mm Hg, increasing to 40 mm Hg and 41 mm Hg on upgaze. A diagnosis of dysthyroid eye disease was made and the patient treated with acetazolamide 250 mg three times daily and prednisolone 80 mg daily.

Thyroid function tests, full blood count, erythrocyte sedimentation rate, urea and electrolytes were all normal. A computed tomography scan showed marked increase in size of all the extraocular muscles but particularly the medial rectus on both sides (Fig 1) with crowding of the orbital apex suggestive of dysthyroid eye disease. Over the next week there was gradual improvement in both symptoms and clinical signs. Steroids were gradually reduced and at his last visit 4 weeks after the episode the visual acuities had returned to 6/6 right and 6/6 left with all Ishihara plates seen on both sides. The chemosis had resolved and there was a full range of extraocular movements.

COMMENT

Dysthyroid eye disease is described by Duane¹ as an organ specific autoimmune disease with a tenuous link to thyroid disease. It is characterised histologically by inflammation, oedema, and secondary fibrosis and clinically by proptosis, eyelid oedema and retraction, conjunctival chemosis, extraocular muscle abnormalities, and optic neuropathy. The aetiology is thought to be an autoimmune process possibly due to an altered antigen on a follicular cell that stimulates antibody production which cross reacts with extraocular tissue.

Alternatively, the primary problem may be a deficiency in suppressor T cell function and a breakdown of the immune system's ability to recognise self tissue. Extraocular tissue is thus recognised as foreign and an immune response mounted against it.

Amiodarone is a class III antiarrhythmic and chemically a benzofuran derivative. It is extensively plasma protein bound and is structurally similar to thyroxine. Amiodarone has been reported in the medical literature to cause both hypothyroidism and hyperthyroidism^{2,3} which ophthalmologists should be aware of. Treatment with amiodarone has also been shown to result in the production of both antithyroid antibodies⁴ and anti-amiodarone antibodies. Previous studies have shown a positive correlation between the presence of anti-amiodarone antibodies and the development of amiodarone induced side

effects suggesting an immunological basis of these side effects.⁵ These ocular side effects may occur in the presence of normal or increased thyroid function.

We suggest that amiodarone induced an exacerbation of thyroid eye disease in our patient already suffering from hypothyroidism either by acting as a stimulus for the production of antibodies that cross reacted with extraocular tissue or by the direct stimulation of T cells and a breakdown of the self recognition process.

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Accepted for publication 15 April 1996

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Carcinoid tumour metastatic to the choroid

EDITOR.—Uveal metastasis is believed to be the most common form of intraocular malignancy. Rarely, however, do features of a metastatic lesion indicate the primary origin of the tumour. A case of multiple metastatic uveal carcinoid tumour is presented, in which the characteristic orange colour of the lesions was suggestive of the nature of the primary tumour.

CASE REPORT

A 23-year-old white woman presented with a 1 month history of bilateral floaters. Eight years earlier, a left pneumonectomy with adjuvant radiotherapy had been performed for bronchial carcinoid tumour with mediastinal lymph node involvement. She had since remained well with no evidence of systemic recurrence. The visual acuity was 20/20 in both eyes and the anterior segments were normal. In the right fundus there were three well circumscribed orange choroidal lesions in the posterior pole (Fig 1) and one inferonasal to the optic disc. In the left fundus, a similar orange choroidal lesion was identified nasal to

Figure 1 Fundus photograph of the right eye showing three orange choroidal metastases suggestive of carcinoid tumour.

Figure 2 Fundus photograph of the left eye showing a single orange choroidal lesion nasal to the optic disc.

the optic disc (Fig 2). Fluorescein angiography showed hyperfluorescence of the lesions in the arterial and laminar venous phases that persisted through the late stages (Fig 3A and B). Indocyanine green angiography revealed hyperfluorescence of the lesions in early and late frames with late dot hyperfluorescence (Fig 4A and B). B scan ultrasonography documented high internal reflectivity and a maximum thickness of 1 mm. A diagnosis of multiple metastatic uveal carcinoid tumour was made. Systemic evaluation, including physical examination, liver enzymes, chest x ray, and brain magnetic resonance imaging showed no evidence of metastases elsewhere and she was visually asymptomatic. The lesions were carefully observed and remained unchanged at 12 months' follow up.

COMMENT

Choroidal metastases from carcinoid tumour are rarely described¹ and comprise approximately only 2% of intraocular metastases.²

Figure 3 (A) Laminar venous phase fluorescein angiogram of the right eye showing moderate hyperfluorescence of the choroidal lesions. (B) Late phase fluorescein angiogram of the right eye showing continued hyperfluorescence of the choroidal lesions.