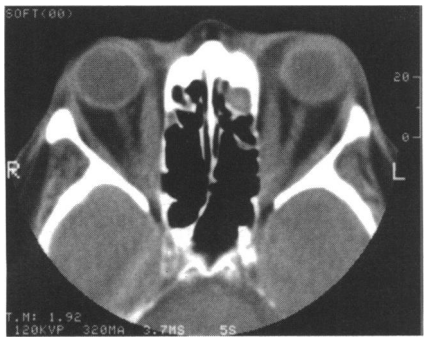

Figure 1 The computed tomography scan appearance of the orbit before treatment.

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 retropulsion 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 Duane[1] 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. Treament with amiodarone has also been shown to result in in the production of both antithyroid antibodies[4] 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.

S BANERJEE
C B JAMES
The Eye Unit, Stoke Mandeville Hospital, Aylesbury, Bucks

Correspondence to: Mr C B James.
Accepted for publication 15 April 1996

1 *Duane's clinical ophthalmology*. Vol 5. Baltimore: JB Lippincott, 1994.
2 Wilson JS, Podrid PJ. Side effects of amiodarone. *Am Heart J* 1991;**121**:158–71.
3 Fraunfelder FT. *Drug induced ocular side effects*. 4th ed. Philadelphia: Williams and Wilkins, 1996: 219–22.
4 Monteiro E, Galvao-teles A, Santos ML, Mourao L, Correia MJ, Lopo Tuna J, *et al*. Antithyroid antibodies as an early marker for thyroid disease induced by Amiodarone. *BMJ Clinical Research Edition* 1986;**292**:227–8.
5 Pichler WJ, Schindler L, Staubli M, Stadler BM, de Weck AL. Antiamiodarone antibodies: detection and relationship to the development of side effects. *Am J Med* 1988;**85**:197–202.

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 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.

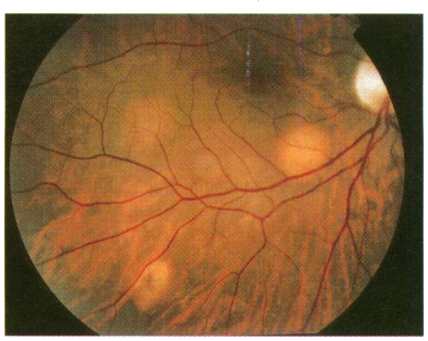

Figure 1 Fundus photograph of the right eye showing three orange choroidal metastases suggestive of carcinoid tumour.

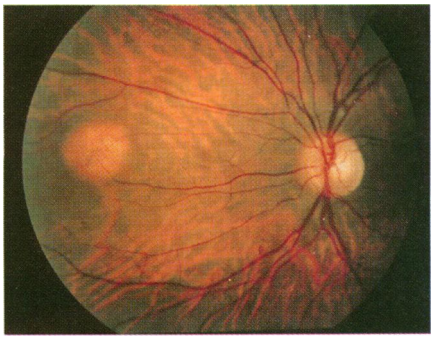

Figure 2 Fundus photograph of the left eye showing a single orange choroidal lesion nasal to the optic disc.

COMMENT

Choroidal metastases from carcinoid tumour are rarely described[1] and comprise approximately only 2% of intraocular metastases.[2]

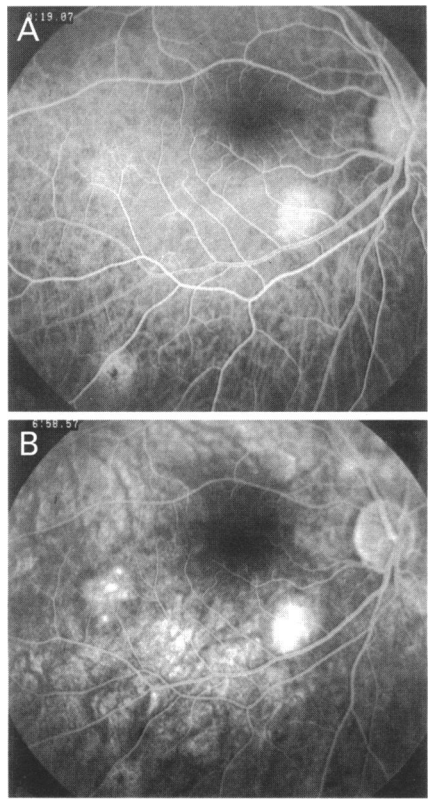

Figure 3 (A) Laminar venous phase fluorescein angiogram of the right eye showing moderate hyperfluorescence of the choroidal lesions. (B) Late phase fluorescein angiogram of the right eye showing continued hyperfluorescence of the choroidal lesions.

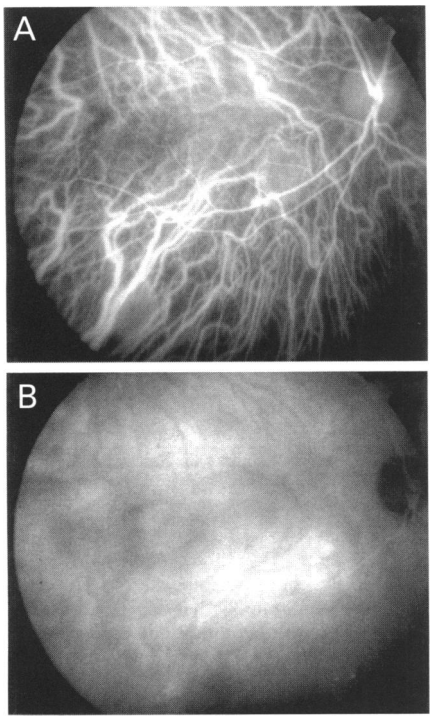

Figure 4 (A) Early frame indocyanine green angiogram of the right eye detailing mild homogeneous fluorescence of the choroidal lesions. (B) Late frame indocyanine green angiogram of the right eye showing isofluorescence of the three lesions and intrinsic dot hyperfluorescence of the largest tumour.

The few reported cases suggest they frequently originate from bronchus in contrast with orbital carcinoid metastases which may originate from the gastrointestinal tract.[3 4] They may have a characteristic orange appearance,[1 2] in contrast with most other choroidal metastases which have a creamy yellow colour.[1] The interval between diagnosis of the primary carcinoid tumour and uveal metastases may be as long as 15 years (mean 7 years) and in over 50% of patients the primary lesion is undiscovered at the time of ocular diagnosis.[2] Unlike breast and lung uveal metastases,[5] the mean survival after diagnosis of uveal carcinoid metastases approaches 3 years. The occurrence of orange choroidal lesions should raise the possibility of carcinoid metastases and prompt an intense search for a primary lesion, directed principally at the lung, or if the primary is known, for systemic recurrence elsewhere. At least 50% of cases are likely to have other metastases at the time ocular involvement is diagnosed.[1 4] In this case, however, systemic evaluation was unremarkable. A urinary 5HIAA was not performed as this had not been helpful in previous cases.[1] Close follow up is required to detect tumour activity, either growth or the development of subretinal fluid or haemorrhage. External beam radiotherapy (3000–4000 cGy) caused effective tumour regression in six of eight treated cases,[1 4] and is advocated if activity occurs.

Support provided by the Eye Tumor Research Foundation, Philadelphia, PA, USA.

PHILIP G HYKIN
Ocular Oncology Department and The Retina Service, Wills Eye Hospital, Thomas Jefferson University, Philadelphia, PA 19107, USA

JERRY A SHIELDS
CAROL L SHIELDS
PATRICK V DE POTTER
Ocular Oncology Department, Wills Eye Hospital, Thomas Jefferson University, Philadelphia, PA 19107, USA

WILLIAM H KEHRLI
Northeastern Eye Institute, Scranton, PA 18503, USA

Correspondence to: Jerry A Shields, MD, Ocular Oncology Department, Wills Eye Hospital, 9th & Walnut Streets, Philadelphia, PA 19107, USA.
Accepted for publication 25 June 1996

1 Shields JA, Shields CL. *Intraocular tumors: a text and atlas*. Philadelphia: WB Saunders, 1992;207–38.
2 Harbour JW, De Potter P, Shields CL, Shields JA. Uveal metastases from carcinoid tumor. *Ophthalmology* 1994;**101**:1084–90.
3 Archer DB, Gardiner TA. An ultrastructural study of carcinoid tumor of the iris. *Am J Ophthalmol* 1982;**94**:357–68.
4 Fan JT, Buettner H, Bartley GB, Bolling J. Clinical features and treatment of seven patients with carcinoid tumor metastatic to the eye and orbit. *Am J Ophthalmol* 1995;**119**:211–8.
5 Stephens RF, Shields JA. Diagnosis and management of cancer metastatic to the uvea: a study of 70 cases. *Ophthalmology* 1979;**86**:1336–49.