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Sir,

Metastatic Adenocarcinoma Presenting as an Isolated Ciliary Body Tumour

A 69-year-old man presented to the ophthalmology outpatient department complaining of a 3-month history of burning pain in his left eye. On examination visual acuity was 6/6 in both eyes. There was a brownish mass on the temporal iris, which, on gonioscopy, infiltrated the angle from 2 to 5 o'clock (Fig. 1). Intraocular pressure was 18 mmHg, right and left. On dilating the pupil there was a large mass in the ciliary body, which on ultrasound measured 14 mm × 14 mm with a thickness of 5 mm.

A presumptive diagnosis of malignant melanoma of the ciliary body was made. The possibility of a metastatic lesion was also considered. The patient was systemically well with no other signs or symptoms.

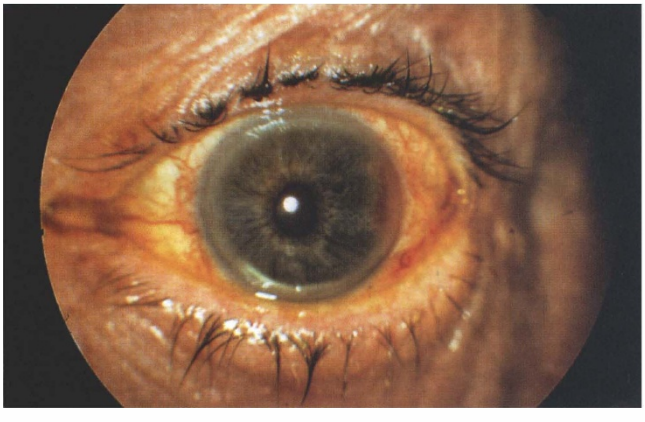

Fig. 1. *The left eye shows a brownish mass lesion on the temporal iris extending from 2 to 5 o'clock.*

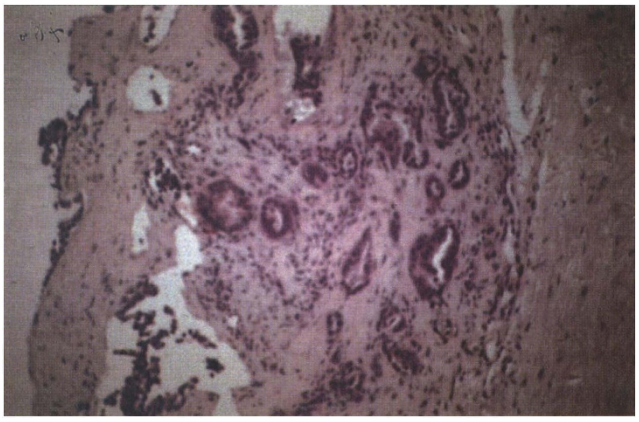

Fig. 2. *Pathology of the lesion reveals metastatic adenocarcinoma.*

A metastatic screen was negative. An excision biopsy of the lesion was performed. Local ruthenium plaque radiotherapy was then applied for 4 days, delivering a dosage of 11 400 cGy to a depth of 5.5 mm. Pathology revealed a mucin-secreting adenocarcinoma of likely lung or gastrointestinal origin (Fig. 2). A systemic investigation was negative for a primary lesion. The patient received a palliative course of chemotherapy. Clinically he deteriorated rapidly. He died 12 weeks later from pulmonary metastases, which were not present on initial examination. Permission for a post mortem was not obtained.

Discussion

This is a case report of a metastatic adenocarcinoma which presented as an isolated primary ciliary body tumour, for which no primary site was identified. Metastases to the iris are usually diffuse. Reported incidence of metastases to the eye varies between 4.9% and 7%.[1,2] The iris and ciliary body is a less common site and is involved in approximately 7.8% of cases metastasising to the eye.[3] The posterior choroid is most frequently involved – reportedly in 80% of cases.

Metastases to the eye generally present after the primary tumour has been identified. Unknown primary sites account for 11% of metastases to the eye.[4] Prognosis is poor with metastases, regardless of the origin of the primary lesion, median survival time being 13 months after diagnosis of iris metastases.[3]

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Sir,

Lipaemia Retinalis in a Premature Infant with Type I Hyperlipoproteinaemia

A premature infant who was found to have lipaemia retinalis due to hyperlipidaemia of unknown cause was recently reported.[1] We would like to report a premature infant with type 1 familial hyperlipidaemia[2] who developed lipaemia retinalis in association with stage 3 retinopathy of prematurity (ROP).

A premature infant born at 26 weeks gestational age (birth weight 960 g) was found to be markedly lipaemic at 37 weeks gestational age with a triglyceride level of 23.7 mmol/l (normal range 2–4 mmol/l) that rose to a peak of 78 mmol/l before being converted from expressed milk feeds to medium-chain triglyceride (MCT) milk. A diagnosis of hyperchylomicronaemia (Fredrickson type 1 hyperlipoproteinaemia) was made. There was no family history of any lipid disorder and both parents had normal lipid profiles.

Screening for ROP was undertaken from 32 weeks gestational age. Initially, stage 1 ROP in zone III was present in both eyes.[3] At 37 weeks, 2 clock-hours of stage 3 ROP in zone III was observed in the left eye.

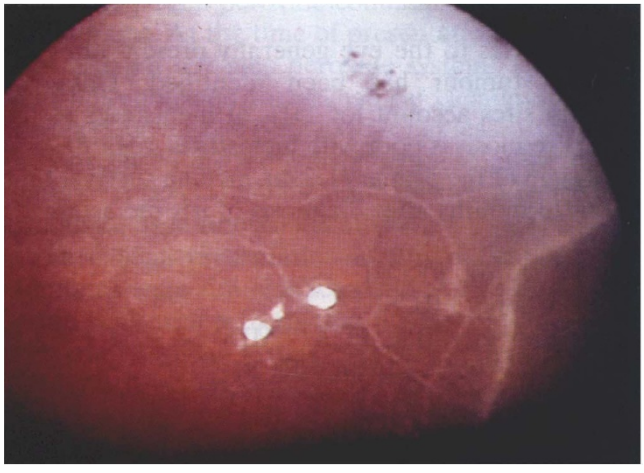

Fig. 1. *Fundus photograph of the left eye at 37 weeks gestational age showing lipaemia retinalis and ROP stage 3 in zone III.*

The retinal vessels remained of normal calibre and there was no 'plus' disease. At 40 weeks the retinal vessels were noted to be uniformly pale pink in appearance (Fig. 1). The plasma triglyceride level at this time was 59 mmol/l. The lipaemia retinalis and ROP both resolved leaving normal retinal vasculature by 43 weeks.

The baby's triglycerides fell to normal on a 25% fat diet made up of 87% MCT lipids. Mother was able to introduce breast feeds at night with MCT lipids by day, with satisfactory growth and triglycerides in the range 5–7 mmol/l. At 2 years he is well, still on the low fat diet but has a mild delay in development and normal triglycerides.

Lipoprotein lipase deficiency, which is the commonest cause of familial hyperchylomicronaemia,[4] was excluded by enzyme assay of post-heparin plasma. It is likely that the patient was deficient in the lipoprotein lipase cofactor apoprotein C-II, but this assay was not available.

Lipaemia retinalis was discovered as an incidental finding during screening for ROP. While it is possible to speculate that an increased plasma viscosity might predispose to abnormal development of the retinal vasculature, the infant was at high risk of ROP in any event and such cases generally resolve spontaneously. Furthermore, in the fully developed retina angiographic evidence suggests that retinal perfusion in lipaemia retinalis is normal.[5]

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