LETTERS TO THE JOURNAL

Sir,

Leopard Spot Retina

A 64-year-old man was referred with a 1-year history of distortion of central vision in his right eye and difficulty in dark adaptation. Two years previously he had developed generalised lymphadenopathy with a white count of 22.5 $\times 10^9$/l (85% lymphocytes) and chronic lymphatic leukaemia was diagnosed. General well-being and a stable white count precluded the initiation of chemotherapy.

On examination his visual acuity was 6/9 in either eye; anterior segments and intraocular pressures were normal, with a clear and quiet vitreous. The posterior poles were likened to leopard spots, with extensive focal areas of hyperpigmentation and pallor (Fig. 1). The optic nerve heads and peripheral retinae were normal. Fluorescein angiography demonstrated hyper- and hypofluorescent areas corresponding to the retinal pigment epithelial abnormalities; the retinal and choroidal vasculature was normal (Fig. 2). B-scan ultrasound revealed thickened choroid at the posterior poles (Fig. 3). Results of computed tomographic scanning of the brain and orbits were within normal limits. The magnetic resonance scan showed abnormal enhancement of both the optic nerves and choroid consistent with leukaemic infiltration, but with no central nervous system involvement. The electro-oculogram (EOG) was markedly subnormal; the electroretinogram (ERG) was small but within normal limits, with the final threshold elevated in dark adaptation. Colour vision testing revealed a severe defect especially in the blue/yellow axis. Despite the apparent localisation to the posterior pole the abnormal EOG and reduced ERG suggest widespread dysfunction of the retinal pigment epithelium.

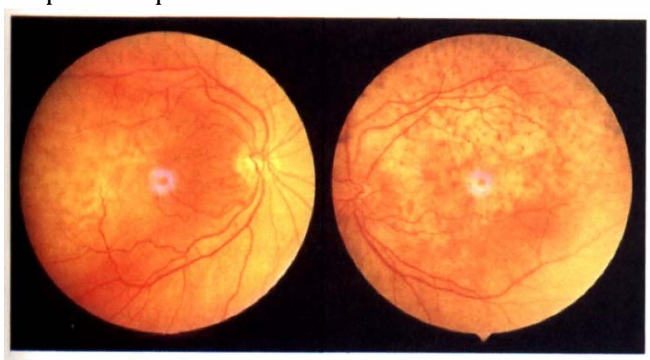

Fig. 1. *Bilateral leopard spot retina. Leukaemic retinal pigment epitheliopathy at the posterior poles. Note the central flash artefact.*

In view of the ocular findings the patient was started on chlorambucil 10 mg daily, which was rapidly discontinued following the onset of uncontrollable nausea. Oral steroids were instituted which lowered the white cell count. Twelve months later, 2 years after the onset of symptoms, the patient retained good visual acuity of 6/12 right 6/6 left, with little change in the fundal appearance.

Discussion

Different series report leukaemic ocular involvement at autopsy being between 28% and 80%.[1] Schachat *et al.* found 42% of patients had clinical signs of leukaemia affecting their eyes.[2] These were mostly due to thrombocytopenia, anaemia and hyperviscosity; only 3% showed direct evidence of ocular infiltration.

Leukaemic pigment epitheliopathy is a rare form of ocular involvement. It was first described by Clayman *et al.* in 1972, who likened the fundal appearance to that of a leopard skin.[3] It has been well described in children who had acute leukaemia and received chemotherapy.[3–6] The visual acuity was reduced in the cases in which it was recorded. Optic nerve infiltration and vitritis were found in most cases.[3–6] Underlying choroidal infiltration was present in the eyes available for histopathology.[3,5]

Toxic or ischaemic effects of the underlying choroidal infiltration have been suggested as a cause of the epithelial changes, and it would seem likely in this case.[3,5,6] It has been identified histologically in the cases where pathology was available. Leukaemic choroidal infiltration has been found to be most marked at the posterior pole in cases of leukaemic pigment epitheliopathy and other eyes with

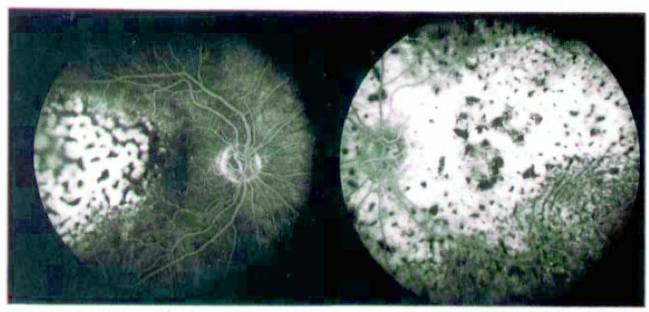

Fig. 2. *Fluorescein angiography demonstrates hyper- and hypofluorescent areas at the posterior poles.*

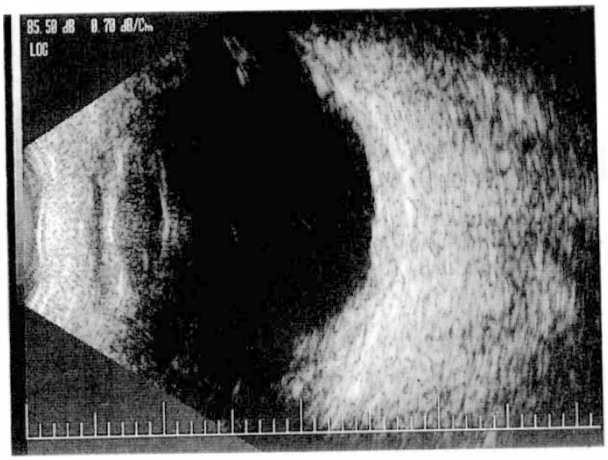

Fig. 3. *B-scan ultrasound of the right eye shows thickening of the choroid at the posterior pole.*

ocular involvement.[3,5,7] This distribution corresponds to the pattern of the retinal pigment epithelial changes observed – although it does not explain why the epitheliopathy is so rare. Additional factors such as anaemia and drug toxicity have been postulated. However, our patient maintained a normal haemoglobin level and had not received any treatment. This suggests the underlying choroidal infiltration was probably the most important factor in our case.

The case presented differs from those previously reported in that it occurs in an adult with untreated chronic lymphatic leukaemia. It also appears to be a more benign form with well-maintained vision and no clinical signs of optic nerve disease or vitreal infiltration.

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Sir,

Salmonella Endophthalmitis in an Infant with Presumed Retinopathy of Prematurity

Endogenous endophthalmitis is a rare complication of salmonellosis and has been reported in several immunocompromised hosts and infants.[1] Six cases of endophthalmitis attributed to *Salmonella* species have been reported; *S. typhimurium* was isolated in three.[1,2]

A male child born at 34 weeks gestation was placed on oxygen therapy for 1 month. At age 4 months his mother noted bilateral leucocoria. He became febrile with nausea and vomiting at age 8 months. Rehydration and unknown systemic antibiotic treatment were instituted at that time through a local clinic 3 days prior to hospitalisation.

On admission his temperature was 39°C and the right eye had marked periorbital swelling, proptosis, and corneal melting with a total hypopyon. The diagnosis was fulminant endophthalmitis of the right eye, possibly secondary to acute necrosis from an advanced retinoblastoma, based on the history of leucocoria and observation of proptosis/cellulitis. The left eye had a shallow anterior chamber, an irregular pupil, and a retrolental membrane compatible with retinopathy of prematurity, grade V.

Significant haematology test results were: white blood count of 30 700, 49% polys, 2% bands, 39% lymphs and 10% monos. Blood, stool and conjunctival cultures grew no pathogens. Chest roentgenogram results were normal. Computed tomography of the orbits revealed periorbital soft tissue swelling, optic nerve thickening, and a small soft tissue mass superior to the distal optic nerve in the right eye; the left eye had evidence of a long-standing retinal detachment consistent with retinopathy of prematurity. Ultrasound examination of the right eye revealed a diffuse opacification of the vitreous without calcification, suggestive of endophthalmitis with obliteration of retinal detail; the left eye showed a total funnel-shaped retinal detachment compatible with retinopathy of prematurity.

The right cornea perforated spontaneously 8 hours after admission despite administration to the right eye of topical fortified drops of cephazolin 50 mg/ml and gentamicin 14 mg/ml every hour. The right eye was enucleated 12 hours after admission due to this perforation and the possibility of a necrotic intraocular tumour. Cultures of intraocular contents grew *Salmonella* serogroup B sensitive to cephazolin. Histopathological examination revealed an acute, suppurative panophthalmitis with necrosis obscuring retinal detail—neither confirming nor excluding retinopathy of prematurity. There was no evidence of tumour. The subarachnoid space of the optic nerve and

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