only two previously reported cases and these formed part of a series describing the ICG findings in various choroidal inflammatory diseases.[5] Although the angiograms were not reported in detail, in both cases the serpiginous lesion was described as being continuously hypofluorescent on ICG and the area of hypofluorescence was described as being more extensive than had been suspected from clinical and FA examination. This observation was also true in the case we describe and our findings therefore support the hypothesis that, in serpiginous choroidopathy, the true extent of the choriocapillaris defect can only be accurately assessed with ICG angiography. In addition to supporting this hypothesis, the case we describe also gives us a new insight into the aetiology of the disease. The RPE overlying much of the area of hypoperfusion we observed in the early phases of the ICG appeared normal. This is an important observation for it suggests that serpiginous choroidopathy is not a primary inflammatory disease of the RPE that secondarily affects the choriocapillaris, but rather that the primary target 'organ' of serpiginous choroidopathy is the choriocapillaris itself.

The management of serpiginous choroidopathy remains controversial. Whilst triple immunosuppression may arrest the active phase of the disease,[6] the disease often relapses after cessation of treatment and the ultimate prognosis is poor.[7] That the choriocapillaris defect may be more extensive than clinical and FA examination suggests may go some way to explaining these observations. It would also explain why the disease often recurs at sites apparently isolated from the original lesion. It is well recognised that choriocapillaris perfusion has to drop markedly before it becomes visually significant to the patient.[8] This may explain why our patient's vision is relatively well preserved despite the extensive lesion demonstrated on ICG angiography. It may be, therefore, that serpiginous choroidopathy does not become clinically manifest until the level of choriocapillaris perfusion drops below a certain threshold which correlates with critical ischaemia of the RPE and outer retina. Without ICG angiography the extent of any choriocapillaris defect, particularly early in the disease, will be underestimated. Any recurrence of the disease would then threaten an already compromised choriocapillaris circulation still further, risking infarction of the dependent RPE and outer retina. ICG angiography, by revealing whether an apparently small area of serpiginous choroidopathy is associated with a more widespread defect of the choriocapillaris (particularly if the choriocapillaris under the fovea is involved), may help persuade the clinician to commence and then continue aggressive immunosuppressive therapy early in the disease in an attempt to prevent future visual loss.

References

1. Weis H, Annesley W, Shields J, Tomer T, Christopherson K. The clinical course of serpiginous choroidopathy. Am J Ophthalmol 1979;87:133–42.
2. Laatikainen L, Erkklia H. A follow up study on serpiginous choroiditis. Acta Ophthalmol 1981;59:707–18.
3. Wu J, Lewis H, Fine S, Grover D, Green R. Clinicopathologic findings in a patient with serpiginous choroiditis and treated choroidal neovascularisation. Retina 1989;9:292–301.
4. King DG, Grizzard WS, Sever RJ, Espinoza L. Serpiginous choroidopathy associated with elevated factor VIII – von Willebrand factor antigen. Retina 1990;10:97–101.
5. Van Liefferinge T, Sallet G, De Laey J. Indocyanine green angiography in cases of inflammatory chorioretinopathy. Bull Soc Belge Ophtalmol 1995;25:73–81.
6. Hooper PL, Kaplan H. Triple agent immunosuppression in serpiginous choroiditis. Ophthalmology 1991;98:944–52.
7. Jones NP. Geographic choroidopathy. In: Uveitis: an illustrated manual. Oxford: Butterworth-Heinemann, 1998:331–5.
8. Kinyoun J, Kalina R. Visual loss from choroidal ischaemia. Am J Ophthalmol 1986;101:650–6.

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Sir,

Visual recovery following intraocular infiltration of gentamicin

Intraocular gentamicin in non-therapeutic doses leads to severe visual loss.[1] We report what is probably the first case where visual recovery was seen after injection of a toxic dose of intraocular gentamicin.

Case report

A 35-year-old woman underwent pars plana lensectomy and vitrectomy, for posteriorly dislocated cataract. At the end of surgery, due to hypotony, the surgeon decided to re-form the globe. The assistant handed a syringe with 20 mg of gentamicin sulphate and 2 mg of dexamethasone for subconjunctival injection, assuming that the surgery was completed. The syringe had been filled up 5 min beforehand and was correctly labelled. While injecting, the surgeon noticed a 'schlieren' effect in the anterior vitreous and detected the mistake. Gentamicin injected was approximately 10 mg and dexamethasone 1.0 mg. Immediate vitreous lavage was started. The 4 mm infusion cannula was replaced after cutting the suture at the lower sclerotomy site and balanced salt solution (BSS) was allowed to flow in. The superior sclerotomy suture was also cut to allow free flow of fluid. The time elapsed between intravitreal injection and the start of BSS washout was 3 min. Vitreous lavage was done for 45 min using 750 ml of BSS. After closure, subconjunctival cefazoline 100 mg was given.

Next day the visual acuity with +10.00 DS was 20/100. The anterior chamber showed trace flare and cells with mild corneal oedema. The macula had no evidence of haemorrhage, infarction, detachment or

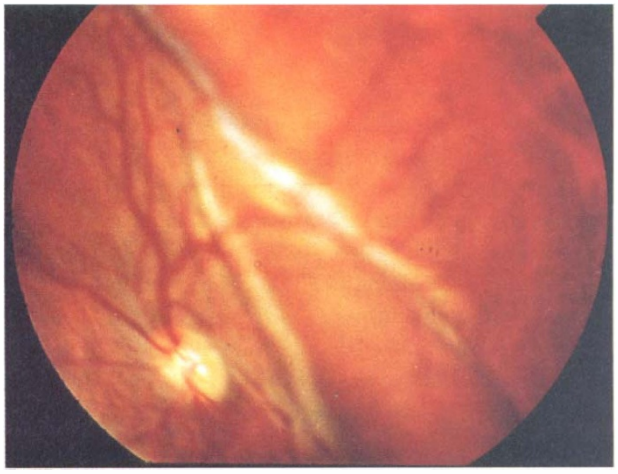

Fig. 1. *Fundus photograph of the left eye showing a large chorioretinal fold due to hypotony. Note the clear media and absence of any choroidal or retinal detachment.*

oedema. The patient was discharged on prednisolone acetate 1%, cyclopentolate hydrochloride 1% and ciprofloxacin 0.3% eye drops, all 4 times a day.

She reported 4 days after surgery with painful visual loss for 8 h. Visual acuity was counting fingers at 1 m. There was a collapsed, tender globe with only trace flare and cells in the anterior chamber. Siedel's test was negative. Fundus evaluation showed folding of the retina along with the ocular coats due to severe hypotony; the fold was passing through the macula (Fig. 1). The patient was diagnosed to have severe hypotony, possibly due to ciliary body inflammation.

She was given intravenous methylprednisolone 500 mg followed by oral prednisolone (1 mg/kg body weight per day) and topical prednisolone acetate 1% eye drop 1 hourly, with cyclopentolate hydrochloride 1% 6-hourly. After 48 h, visual acuity improved to 20/50. Medication was tapered over the next 6 weeks. Three months later her best-corrected visual acuity was 20/30 with a near vision of N6 and the intraocular pressure was 14 mmHg. The retina was normal except for an RPE depigmented line in the macula, at the site of the previous retinal fold (Fig. 2).

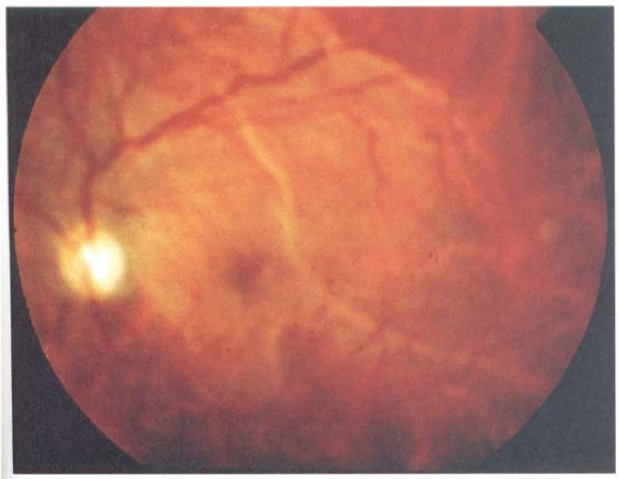

Fig. 2. *Fundus photograph 3 months later showing a depigmented line across the macula at the site of the previous retinal fold.*

Comment

The non-toxic dose of intravitreal gentamicin has been established to be 200–400 μg in rabbits[2] and 400 μg in primates.[3] Higher doses cause retinal haemorrhage, oedema and vascular occlusion followed by optic atrophy, pigmentary retinal changes and neovascular glaucoma.[1] The primary site of gentamicin toxicity is the retinal pigment epithelium (RPE)[3] and toxicity is seen more in albino eyes than in pigmented eyes of rabbits.[4] The higher level of pigmentation in our patient from South India could contribute to the lower risk of toxicity. Though we have not done an electroretinogram (ERG) her functional and clinical recovery are remarkable. The severe, painful hypotony could be attributed to the prolonged flow of fluid through the ciliary body region leading to inflammation and a ciliary shut-down. We are not aware of any reports of effects of gentamicin on the ciliary body, to understand whether the drug itself had a role to play.

Retinal whitening, cherry red spot, retinal haemorrhages and ERG changes were seen within 5 min of a 10 mg injection of gentamicin in primates.[5] The very short time elapsed (less than 5 min) between accidental injection and copious vitreous lavage in our patient was due to the fact that the equipment, the eye and the surgeon were already prepared for the exercise of vitreous lavage, and the mistake was detected while performing the injection. Except for one of the previous 5 cases reported by McDonald *et al.*,[6] the surgeon realised the mistake only when informed by the assistant. To prevent such mishaps, McDonald *et al.*[6] suggested precise labelling of all solutions and the drawing up of antibiotics only when they are needed; both these practices were adhered to in our patient. While injecting the intravitreal injection slowly in the anterior vitreous, with the needle bevel up,[6] we noticed schlieren. We would like to re-emphasise that the presence of 'schlieren' (subtle wavy lines that develop when clear fluids of different viscosities mix) while injecting antibiotics should alert the surgeon to a possible mistake. Since toxicity with intraocular gentamicin occurs within 5–10 min,[5–7] this complication should be managed by *immediate* vitreous lavage, before onset of action of the drug.[7] The efficacy of this procedure is probably shown here in a human eye for the first time.

References

1. Snider JD III, Cohen HB, Chenoweth RG. Acute ischemic retinopathy secondary to intraocular injection of gentamicin. In: Ryan SJ, Dawson AK, Little HL, editors. Retinal diseases. Orlando, FL: Grune and Stratton, 1985:227–32.
2. Peyman GA, May DR, Ericson ES, Apple D. Intraocular injection of gentamicin: toxic effects and clearance. Arch Ophthalmol 1974;92:42–7.
3. D'Amico DJ, Libert J, Kenyon KR, Hanninen LA, Caspers-Velu L. Retinal toxicity of intravitreal gentamicin: an electron microscopic study. Invest Ophthalmol Vis Sci 1984;25:564–72.
4. Zemel E, Lowenstein A, Lei B, Lazar M, Perlman I. Ocular pigmentation protects the rabbit retina from gentamicin-induced toxicity. Invest Ophthalmol Vis Sci 1995;36:1875–84.

5. Brown GC, Eagle RC, Shakin EP, Gruber M, Arbizio VV. Retinal toxicity of intravitreal gentamicin. Arch Ophthalmol 1990;108:1740–4.
6. McDonald HR, Schatz H, Allen AW, Chenoweth RG, Cohen HB, Crawford JB, *et al*. Retinal toxicity secondary to intraocular gentamicin injection. Ophthalmology 1986;93:871–7.
7. Peyman GA. Discussion to: retinal toxicity secondary to intraocular gentamicin injection. Ophthalmology 1986;93:871–7.

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Sir,

Concentric annular macular dystrophy

Concentric annular macular dystrophy is a very rare condition which was first described by Deutman in 1974.[1] The main features are bull's eye maculopathy, perifoveal circular pigment epithelial atrophy and dyschromatopsia. As far as we are aware this is the first case reported in a British journal.

Case report

A 49-year-old Asian woman was referred to the Eye Department following a routine visit to an ophthalmic optician who noted an abnormal macular appearance. At presentation the patient had no visual complaints but on direct questioning did admit to having slight difficulty seeing under dark conditions. There was no history of chloroquine ingestion. Her father was under treatment for primary open angle glaucoma. No other family members had visual problems.

The corrected visual acuity by Snellen chart was right eye 6/9 and left eye 6/6. Anterior segment examination and intraocular pressure were normal. There was a bilateral bull's eye macular appearance (Fig. 1). The optic disc and retinal blood vessels were normal, as were the perifoveal and peripheral fundus. Colour vision by Ishihara testing was normal. Visual fields by computerised perimetry (Humphrey Full Field 120 Point Program) were full.

Fluorescein angiography revealed bilateral circular perifoveal areas of mottled hyperfluorescence with a few areas of hypofluorescence (Fig. 2). There was no leakage but late staining was observed (Fig. 3). The macular area itself did not have any remarkable features on angiography.

Electrodiagnostic testing found normal photopic and scotopic electroretinograms (ERG). The electro-oculogram (EOG) was slightly subnormal. Arden ratios were RE 1.40 and LE 1.37. Flicker fusion frequency was greater than 30 Hz in both eyes.

Comment

In 1974, Deutman reported four cases of a macular dystrophy with a bull's eye appearance clinically, but with angiographic and electrodiagnostic features differing from other macular dystrophies.[1] He named this condition benign concentric annular macular dystrophy. In each case there was a characteristic macular appearance consisting of central hyperpigmentation surrounded by a hypopigmented ring which itself was surrounded by a darker pigmented halo. All patients had normal or near normal vision with mild defects in colour vision.

In Deutman's series, all patients belonged to the same family and a pattern of autosomal dominant inheritance with variable expressivity was seen. In those patients who were affected at a younger age there were more

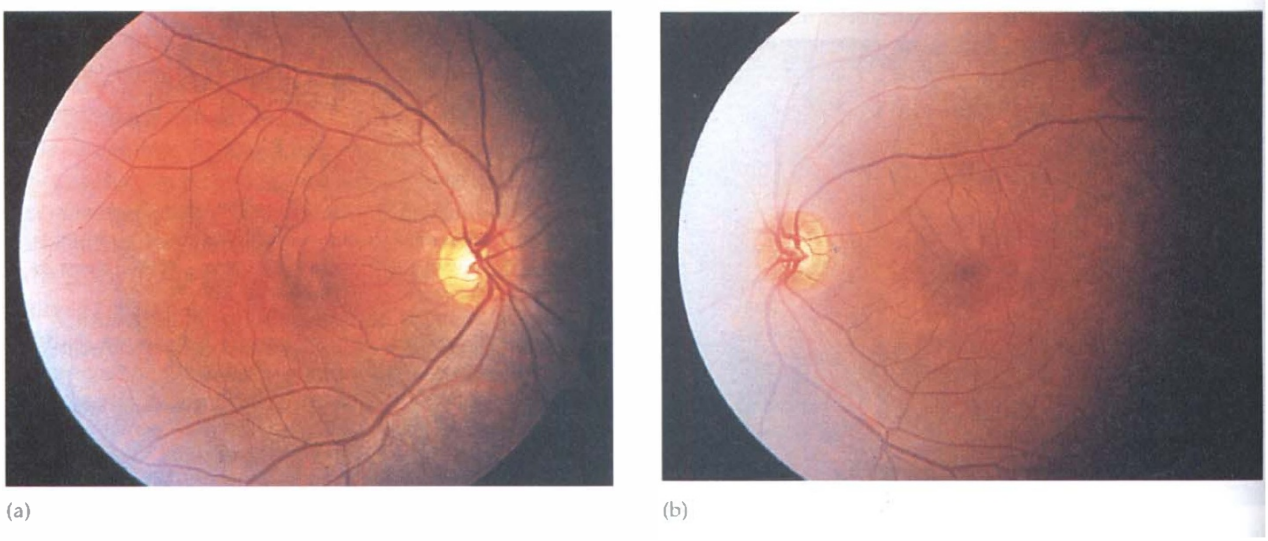

Fig. 1. *(a) Right fundus. (b) Left fundus.*