margin of the macular hole. The surgical induction of PVD and the release from the traction due to the hyaloid membrane let the macular hole close. We suggest that the macular hole and retinal detachment were associated with the vitreoretinal traction phenomenon in the staphyloma.

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Hirokazu Ohashi
Department of Ophthalmology
Tenri Yorozu Hospital
Nara, Japan

Ryoji Yamakawa
Department of Ophthalmology
Kurume University School of Medicine
Kurume, Japan

Hirokazu Ohashi, MD ✉
Department of Ophthalmology and Visual Sciences
Graduate School of Medicine
Kyoto University
54 Shogoin Kawaharacho Sakyo-ku
Kyoto 606-8507, Japan

Tel: +81 75 751 3250
Fax: +81 75 752 0933
e-mail: oha@kuph.kyoto-u.ac.jp

Sir,

Indocyanine green angiographic findings in serpiginous choroidopathy: evidence of a widespread choriocapillaris defect of the peripapillary area and posterior pole

We report a case of early serpiginous choroidopathy in which the indocyanine green angiogram revealed a more extensive defect of the choriocapillaris than was suspected on the clinical and fluorescein angiographic features alone. We discuss the possible implications of this finding for understanding both the aetiology and the subsequent progression of the disease.

Case report

A 56-year-old Caucasian woman with a history of serpiginous choroidopathy affecting the right eye presented with a 2 month history of visual disturbance in her previously unaffected left eye. At presentation her visual acuity was 6/60 N48 right eye, 6/9 N6 left eye. Fundus examination of the left eye revealed an area of chorioretinal geographic atrophy at the posterior pole which involved the lower half of the fovea (Fig. 1). There was in addition an isolated, active lesion at the inferotemporal disc margin. Examination of the right eye revealed an old geographic scar with no evidence of active disease.

Fluorescein angiography (FA) of the left eye revealed a hypofluorescent lesion which, in the late phases of the angiogram, acquired a hyperfluorescent edge – findings typical of an inactive serpiginous lesion (Fig. 2). FA also confirmed the presence of a small area of active disease inferotemporal to the disc. An indocyanine green (ICG) angiogram of the left eye revealed a well-demarcated filling defect within the choriocapillaris and medium-sized choroidal vessels under the area of retinal pigment epithelium (RPE) atrophy (Fig. 3). There was, in the early phases of the angiogram, the additional finding of an extensive area of choriocapillaris hypoperfusion which extended throughout the peripapillary area and macula of the left eye (Fig. 3). This perfusion defect was far more extensive than the preceding clinical examination and FA had suggested.

Comment

Serpiginous choroidopathy is a chronic, progressive, usually bilateral disease of the RPE and choriocapillaries whose natural history is of recurrent relapses and subsequent visual loss.[1] The aetiology of serpiginous choroidopathy is unknown, but it has been hypothesised that it is caused by an inflammatory-induced closure of the choroidal vascular bud.[2–4] Although this hypothesis appears to explain many of the clinical and FA features of the disease, the debate as to whether serpiginous choroidopathy is a primary inflammatory disease of the choriocapillaris, or a primary inflammatory disease of the RPE which secondarily affects the choriocapillaris, has yet to be resolved.

ICG angiography may help answer this question, but to date little has been published on the ICG findings in serpiginous choroidopathy. To our knowledge there are

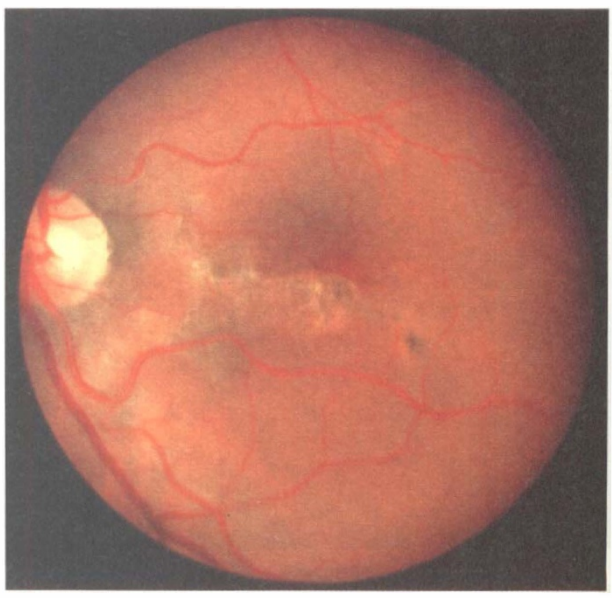
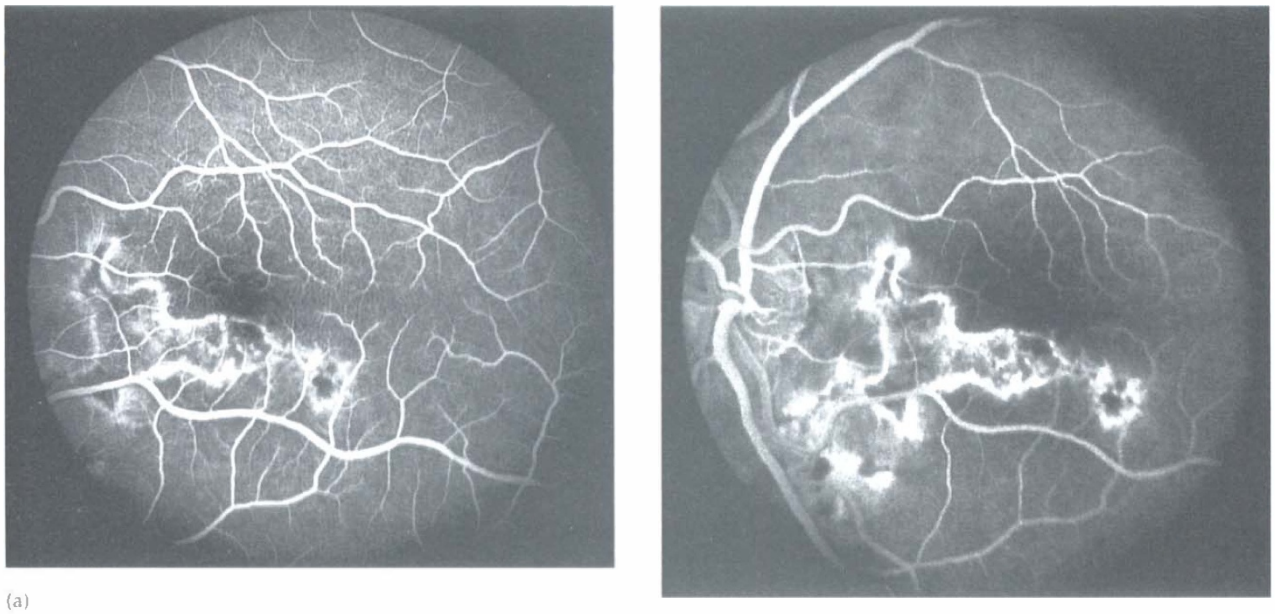


Fig. 1. *Colour fundus photograph of the left eye. The well-demarcated area of chorioretinal geographic atrophy is clearly seen.*



(a) (b)

Fig. 2. *Early (a) and late phase (b) of the fluorescein angiogram peformed in the left eye. The area of geographic choroidal atrophy which radiates from the disc involving the lower half of the fovea is clearly seen. Note the typical hyperfluorescent edge that the serpiginous lesion acquires in the late phase of the angiogram.*

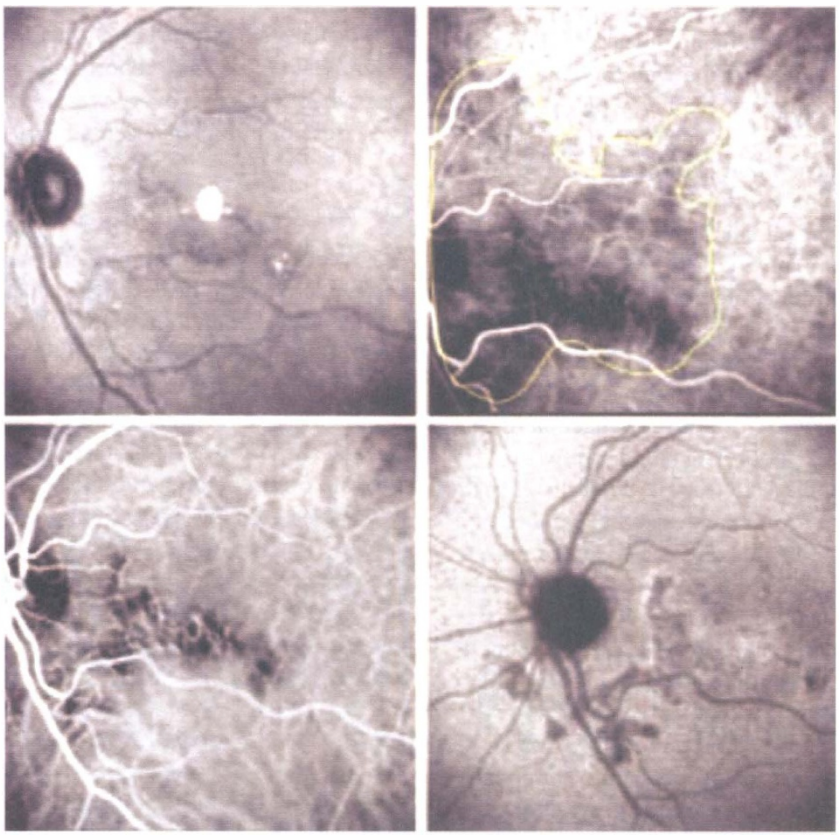


Fig. 3. *Indocyanine green (ICG) angiogram of the left eye. Top row left: Posterior pole of the left eye viewed before ICG was injected. Top row right: Early phase of the angiogram. The intense filling defect corresponding to the atrophic scar is clearly seen. Note also the more subtle but extensive choriocapillaris perfusion defect surrounding the atrophic scar (as outlined by the contour lines). Bottom row: Mid-phase and late phases of the angiogram. The intense perfusion defect under the atrophic area is clearly visible. The subtle choriocapillaris defect is no longer discernible.*

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.

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D.M. Squirrell ✉
R.M. Bhola
J.F. Talbot
Department of Ophthalmology
Royal Hallamshire Hospital
Glossop Road
Sheffield S10 2RX, UK
Tel: +44 (0)114 2713056

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