these events. In this case, the oedema initially underwent complete resolution following a subtenons injection of triamcinolone. The pathophysiology of this ocular manifestation in this condition is unknown.

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Sir,

Indocyanine green angiography in diffuse unilateral subacute neuroretinitis

Diffuse unilateral subacute neuroretinitis (DUSN) is a clinical syndrome caused by a subretinal nematode, initially described by Gass *et al*. [1] DUSN affects one or, occasionally, both eyes and usually results in severe loss of vision if left untreated. This disorder has two distinct stages. The early stage manifests inflammatory signs such as vitritis, papillitis, retinal vasculitis, and recurrent crops of evanescent yellow-white outer retinal lesions.[1] The late stage is characterized by progressive visual loss, optic atrophy, retinal vessel narrowing, and focal or diffuse retinal pigment epithelium (RPE) degeneration.[1]

Fluorescein angiography (FA) characteristics have already been reported in some cases of early and late-stage DUSN.[1] To our knowledge, this report describes for the first time the indocyanine green angiography (ICG-A) characteristics of a patient with early and late-stage DUSN.

Case report

A 34-year-old man was referred to our Retina Clinic complaining of acute loss of vision of 7 days duration. His best-corrected visual acuity was 20/40 in the right eye (RE) and 20/20 in the left eye (LE). Slit-lamp examination of the anterior segments was unremarkable. Intraocular tension was within normal limits in both eyes. Fundus examination of the RE showed a pattern of multiple deep, 100–600 μm, yellow-whitish lesions situated near the infero-temporal vascular arcade. Some discrete yellow-white dots were also observed in the superior half of the fundus (Figure 1a). The posterior vitreous showed a mild inflammatory response. The optic disk and the retinal vessels were apparently healthy. The fundus of the LE was normal.

FA of the RE showed that the lesions masked fluorescence early during the study, and there was a significant hyperfluorescence in the late stages. Some late hyperfluorescence was also seen on the optic disc.

ICG-A of RE revealed that the lesions initially masked fluorescence. Most of these hypofluorescent dark spots were already visible in the early phase of the angiogram (Figure 1b), became more sharply delineated in the intermediate angiographic frames, but just a few spots remained hypofluorescent in the late frames (Figure 1d). The lesions seen in the initial phases of the ICG-A corresponded to those seen ophthalmoscopically. A fuzzy hyperfluorescence at the macular level, which started at the initial phases of ICG-A, persisted in the late frames of the angiogram.

We performed the following examinations: complete blood cell count, FTA-ABS, PPD, and chest X-ray. All results were normal. The diagnosis of DUSN was proposed, but after many visits we were not able to find the worm. Within 1 month, the corioretinal lesions became more evident in the superior half of the fundus and the patient's visual acuity dropped to 20/100 (Figure 1d). After 6 months, the worm was eventually located (Figure 2-b) and destroyed by laser photocoagulation. However, the patient's visual acuity was 20/400. Ophthalmoscopy performed at this time revealed round hypopigmented lesions throughout the posterior pole as well as diffuse RPE degeneration, mild optic disk atrophy and discrete

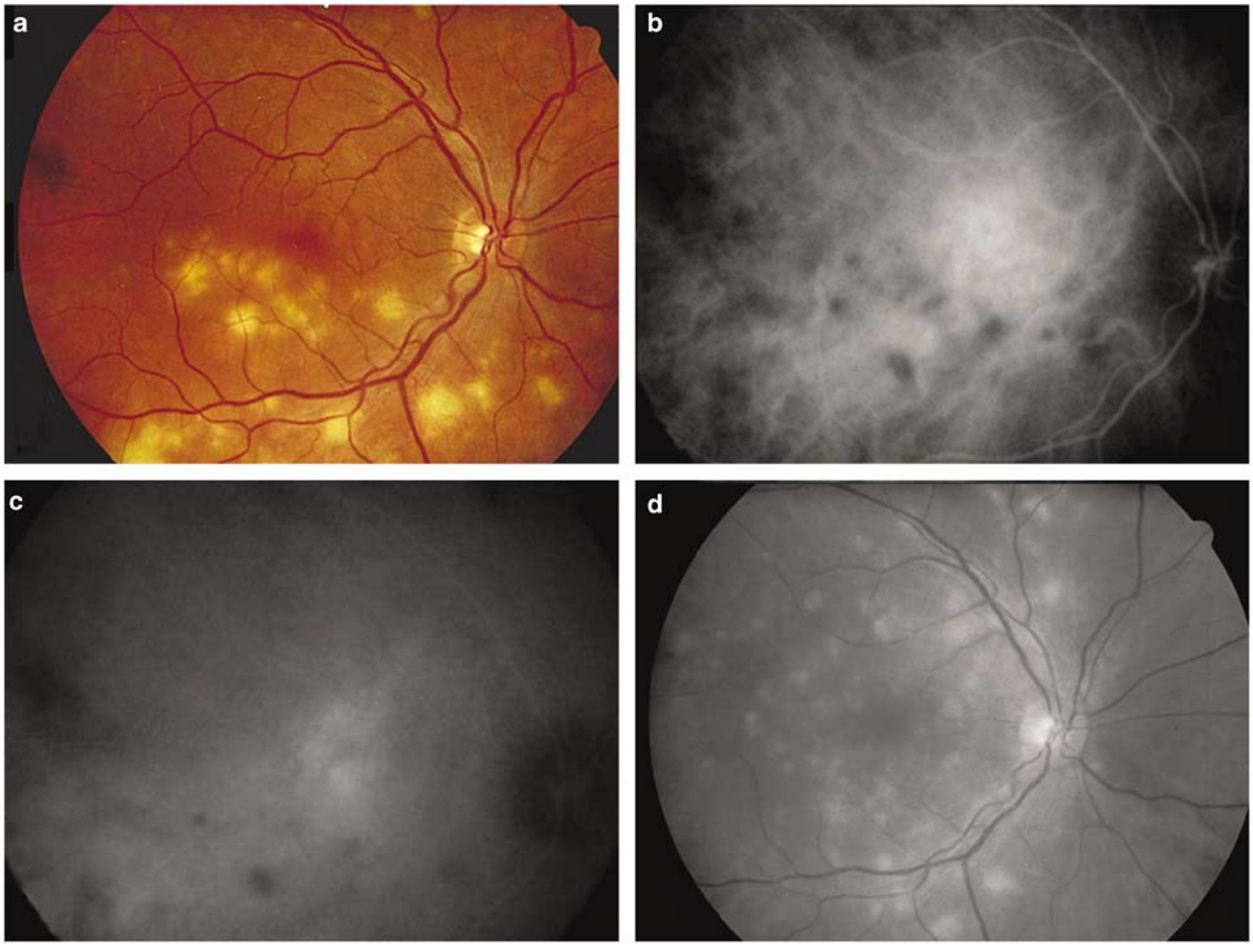

Figure 1 Early-stage DUSN. (a) The affected eye revealed multiple yellow-white subretinal lesions at the posterior pole. (b) Early-phase ICG-A shows hypofluorescence of the lesions. (c) Late-phase ICG-A reveals few hypofluorescent dots and a fuzzy hyperfluorescence in the macular region. (d) After 1 month, the superior subretinal lesions increased in number and became more evident.

narrowing of the retinal vessels (Figure 2a). ICG-A revealed several dark spots located at the posterior pole. A few spots remained hypofluorescent throughout the examination. An area of hyperfluorescence in the macular region was observed in the intermediate phases of the exam, and persisted up to the late phases (Figure 2c and d).

Comment

It has been suggested that the pathogenesis of DUSN appears to involve a local toxic tissue effect on the outer retina caused by the worm by-products left behind, as well as a more diffuse toxic reaction affecting both the inner and outer retina. [2] Nevertheless, our ICG-A features suggest that the choroid is also involved in early-stage DUSN. Choroidal infiltration, which prevented normal choroidal ICG impregnation, probably was the physiopathogenic explanation for the hypofluorescent dark spots seen in the affected eye of our patient. However, additional choriocapillaris nonperfusion may also have been present, which would explain the corresponding hypofluorescence seen on FA in some of the lesions. The dark spots present in the initial phase of ICG-A were seem to either disappear or persist in the late phase of the exam. Hypofluorescent dots persisting in the late phase were interpreted as full-thickness lesions allowing no ICG diffusion, whereas dots becoming isofluorescent in the late phase were interpreted as partial-thickness lesions progressively surrounded by ICG fluorescence.
A late hyperfluorescence located in the macular region was interpreted as vasculitis with leakage from choroidal vessels. Interestingly, this region appeared relatively unaffected on fundus biomicroscopy and on FA.

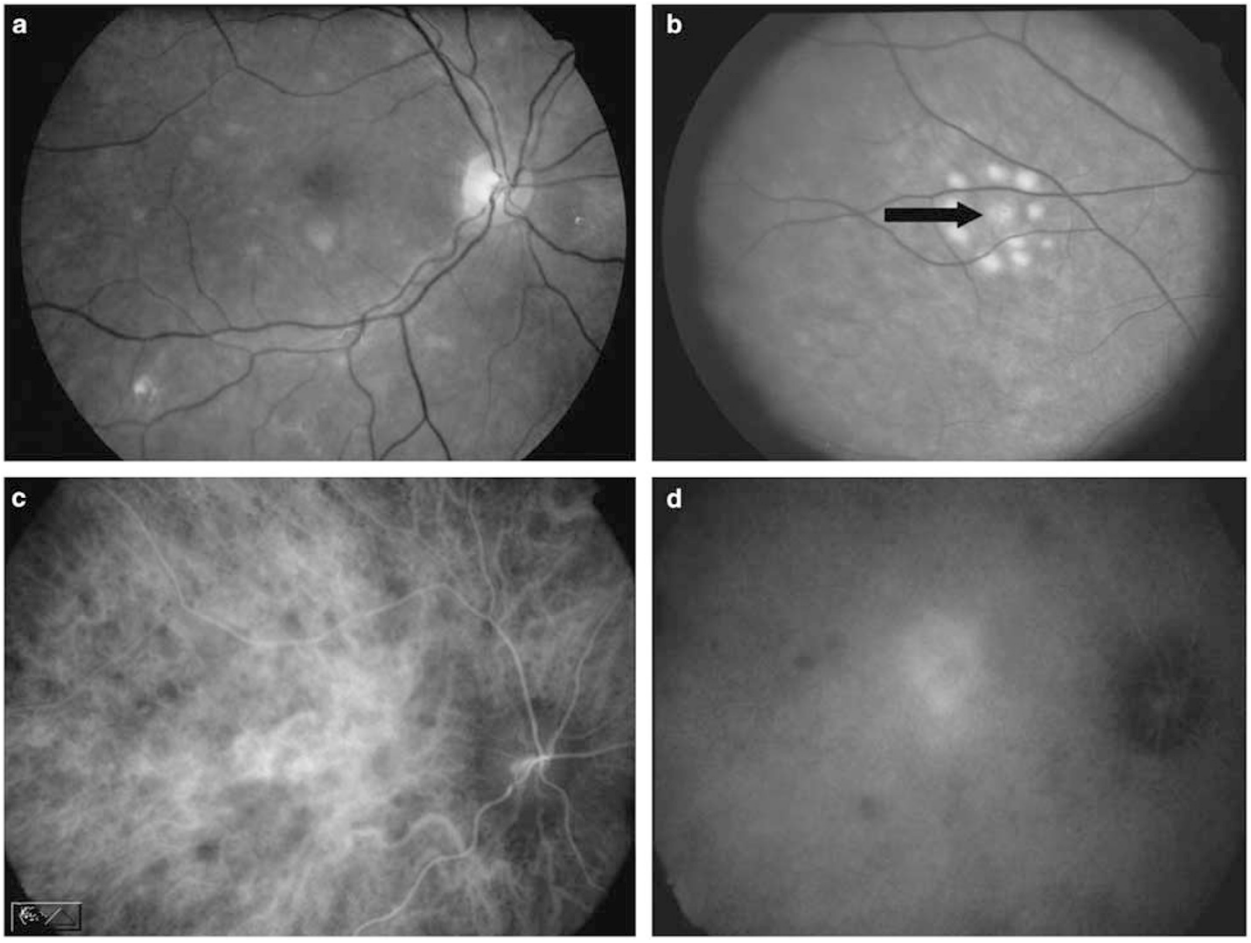

Figure 2 Late-stage DUSN. (a**)** Observe many round hypopigmented lesions throughout the posterior pole as well as mild optic disk atrophy, discrete narrowing of the retinal vessels, and diffuse RPE degeneration. (b) The located worm, surrounded by laser spots. (c**)** Early and (d) late-phase ICG-A revealed hypofluorescent spots and an area of hyperfluorescence in the macular region.

In late-stage DUSN, ICG-A revealed dark spots, which were interpreted as fibrosis (stromal scars) or chorioretinal atrophy in areas of previous choroidal inflammatory infiltration. The persistent hyperfluorescence in the macular region, now also observed in the FA study, was interpreted as being due to degenerative changes of both RPE and choriocapillaris, secondary to previous vasculitis at that site.

We know that the ICG-A features showed in this case of DUSN were nonspecific for the disease. Similar findings have been identified in other posterior uveitis, such as Vogt–Koyanagi–Harada syndrome, sympathetic ophthalmia, tuberculosis, and birdshot chorioretinopathy.[3,4] However, we believe that the additional findings on ICG-A add to the understanding and extent of the disease, demonstrating a degree of choroidal involvement not previously suspected.

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Sir,

Decreased vision as the initial presenting symptom of disseminated prostatic disease

Soft tissue solitary choroidal masses of the eye as the initial presenting symptom of disseminated metastatic disease is rare. We present the case of a 52-year old who presented with visual symptoms relating to a prostatic adenocarcinoma primary, who responded clinically with external beam radiotherapy. The presentation and infrequency of such presentations is discussed.

Case report

A 52-year old presented with a 1-week history of a painless decrease in visual acuity in his right eye, of gradual onset. Past ocular history was remarkable for anisometropic amblyopia of the left eye. Examination revealed an unaided Snellen visual acuity of 6/60 right and 6/18 left. Anterior segment examination was normal. Fundal examination revealed a solitary elevated solid mass lesion superiorly with an overlying exudative retinal detachment, involving the macula (Figure 1). On systems review, the patient revealed a 6-week history of low back pain and nocturia and right-calf pain for 5 days.

B-scan ultrasound examination revealed a 5 mm (high) × 13 mm homogenous mass with choroidal excavation (Figure 2). A metastatic screen with CT imaging revealed extensive systemic metastases including a retropharygeal mass, para-aortic adenopathy, bilateral hydronephrosis, bony lytic secondaries in lumbar vertebrae 3 and sacroiliac joints with an osteolytic appearance from a presumed prostatic carcinoma primary (Figures 3a–d). Dopplers of the right lower limb confirmed a deep vein thrombosis secondary to pelvic compression. Serology revealed an elevated prostate-specific antigen (PSA) at 104 ng/dl, and renal impairment with a creatinine of 135. Liver function tests revealed an elevated alkaline phosphatase. Clinical examination (urology) revealed a T4 prostatic carcinoma. Ultrasound-guided biopsy confirmed prostatic adenocarcinoma on histopathology, with a Gleeson rating of 9.

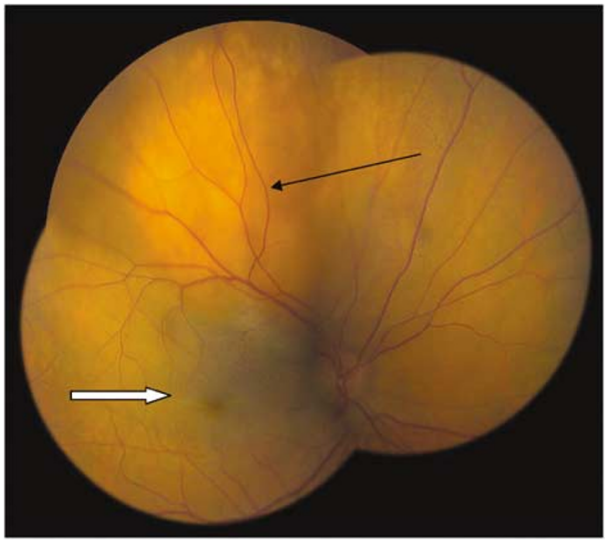

Figure 1 Superior lesion (black arrow) with overlying exudative retinal detachment involving the macula (white arrow).

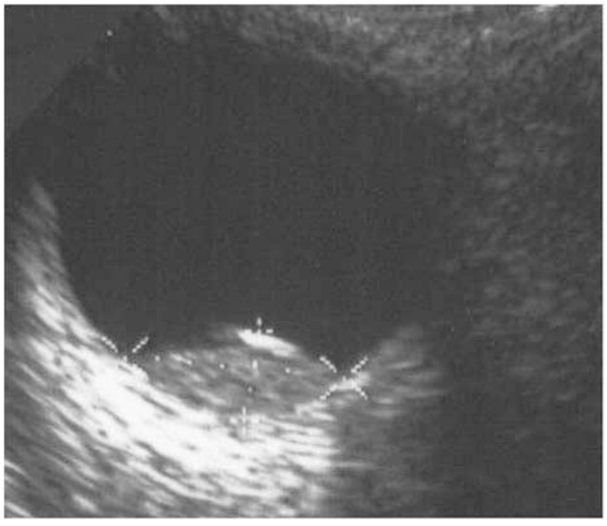

Figure 2 B-scan ultrasound showing an elevated, solid, homogenous mass with choroidal excavation with calcified area in the anterior pole of the lesion.