chemosis in this case was at least partly due to the delay in the patient seeking medical attention. The swollen conjunctiva had prolapsed and its surface had become keratinised. Due to swelling of the orbital tissue and splinting between the eyelids the prolapsed conjunctiva was unable to return to its anatomical position.

Although the CT scan of the orbits was helpful in making the diagnosis, STIR (Short Tau Inversion Recovery) sequence MRI may have been more useful in this case as it gives an assessment of muscle water content in 'active' thyroid eye disease.[4] Active inflammation is associated with oedema within the tissues and will give a bright signal on the STIR sequence, which can be used to assess the degree of inflammation within the extraocular muscles.

The patient was treated with combined radiotherapy and medical immunosuppression as orbital decompressive surgery was declined. Claridge *et al.*[5] showed that early application of this combination therapy was more effective than either treatment alone in the management of active thyroid eye disease and also reduces the requirement for corrective surgery.

According to the NOSPECS classification the indication for treatment has been the severity of symptoms instead of the rate of progression of the disease. The soft tissue manifestations of Graves' disease have been apportioned a minor role in this clinical classification.[6] This is partly due to the difficulty in quantification of these findings, the absence of an associated immediate threat to vision and lack of specificity for Graves' disease especially when present unilaterally. However, the Mourits scoring classification is targeted more at assessing the rate of progression of thyroid eye disease and is helpful in predicting the therapeutic outcome of immunosuppressive therapy in Graves' ophthalmopathy.[7]

Our case illustrates that on occasions soft tissue changes can progress to cause considerable discomfort and interfere with vision.

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Sir,

Assessment of choroidal involvement in sarcoidosis with indocyanine green angiography

Ocular involvement is common in systemic sarcoidosis, anterior granulomatous uveitis being the most common finding.[1] Posterior segment involvement is generally found together with anterior segment signs.[1] We report a case with choroidal involvement only, in which sequential indocyanine green angiography (ICGA) helped to confirm the choroidal involvement and follow the resolution of the process.

Case report

A 52-year-old woman presented with erythematous papules on the face and extremities in June 1996. Biopsy of one papule revealed non-caseating granuloma, suggestive for sarcoidosis. The laboratory data, including serum angiotensin converting enzyme (ACE) level and chest radiograph, were unremarkable. Results of the ophthalmological examination were not significant. In January 1997, laboratory evaluation, gallium scintigraphy and thorax tomography were within normal limits.

In July 1997, increased serum ACE and urinary calcium levels were noted. Thorax tomography revealed increased reticular density and patchy consolidation. Ocular examination disclosed visual acuities of 20/20 in both eyes. There was a 5 × 8 mm nodule in the right lower eyelid. Anterior segments were quiet. A yellowish flat lesion was observed at the level of the choroid, temporal to the optic disc, in the right eye (Fig. 1, upper left). The retinal vessels appeared normal with no haemorrhages or exudates. There were no cells in the vitreous. Blind biopsy from the inferior forniceal conjunctiva revealed a non-caseating granuloma. Fundus fluorescein angiography (FFA) showed late staining hyperfluorescence corresponding to the lesion (Fig. 1, upper left). ICGA, performed with 25 mg of ICG using a Topcon 50 I/A (Topcon, Tokyo, Japan) camera coupled to an ImageNet (Topcon) system, demonstrated five patches of hypofluorescence starting very early and continuing for at least 20 min (Fig. 1, lower left). There was no significant hypo- or hyperfluorescence in late images (Fig. 1, lower right).

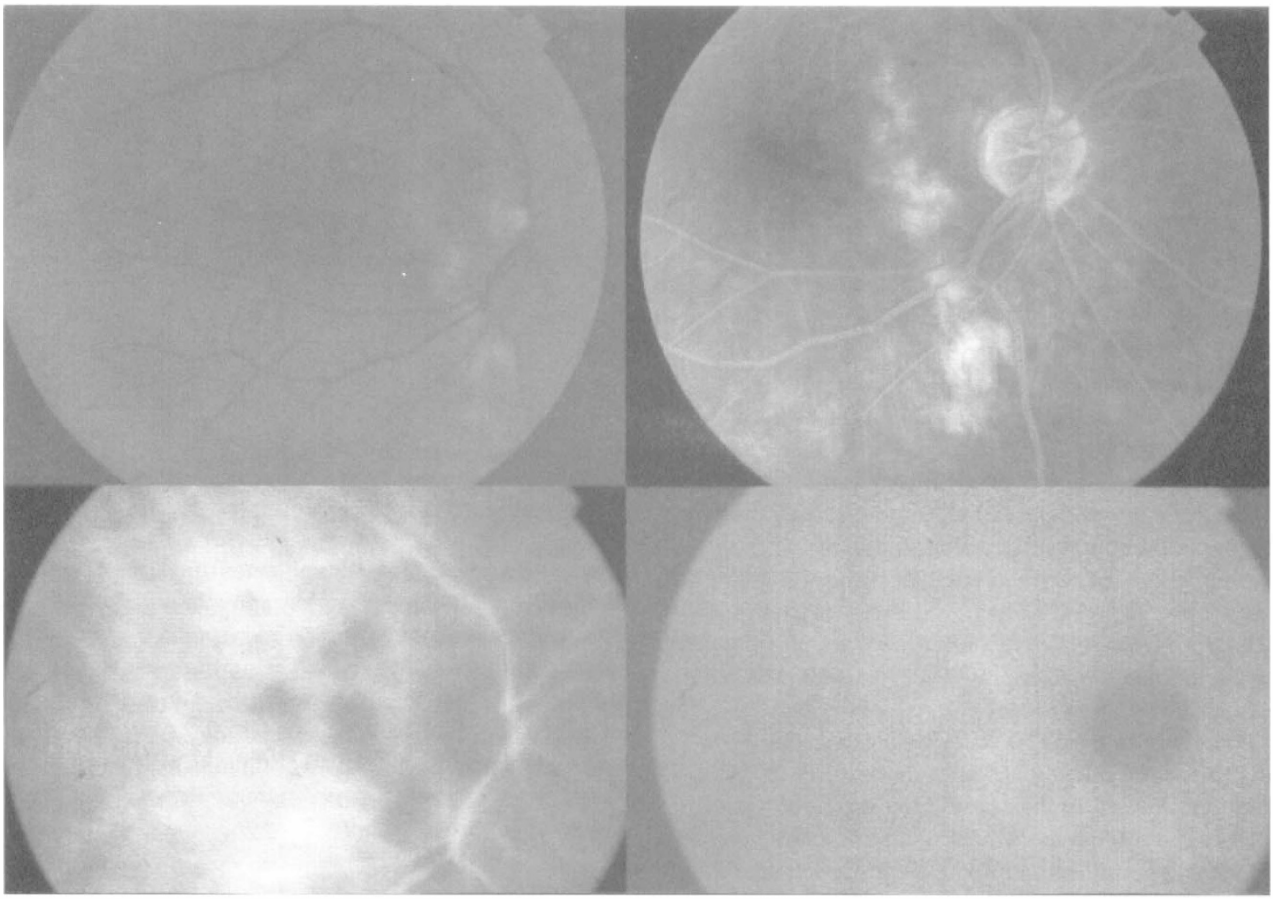

Fig. 1. *Upper left: Choroidal lesion temporal to the optic disc. Upper right: Fundus fluorescein angiography (FFA), late phase. There is late staining hyperfluorescence corresponding to the lesion. Lower left: Indocyanine green angiography (ICGA), mid-phase. Patches of hypofluorescence are seen. Lower right: ICGA, late phase. There is no significant hypo- or hyperfluorescence.*

With the transbronchial biopsy revealing non-caseating granuloma, the diagnosis of sarcoidosis was confirmed and systemic daily 40 mg prednisolone was instituted by the Pulmonary Medicine Department.

Funduscopic examination after 2 months of systemic therapy revealed very slight alteration in the lesion (Fig. 2, upper left), with no change on FFA (Fig. 2, upper right). On ICGA the early hypofluorescent patches were smaller in size, disappearing after 10 min, and were slightly hyperfluorescent in the very late images (Fig. 2, lower left and right). The nodule in the lid had disappeared. Thorax tomography was negative and serum ACE level was within normal limits.

Comment

Lesions at the level of retinal pigment epithelium (RPE) or choroid can be seen in 29-50% of cases with ocular sarcoidosis.[2] At the choroid, granulomas may be seen, which are generally multiple, bilateral and shallow yellow-white tumefactions with subretinal fluid. Choroidal granulomas are associated with intraocular inflammation at other sites, usually with the involvement of the overlying retina, giving the clinical appearance of chorioretinitis.[1,3,4] However, few isolated granuloma cases demonstrated an absence of retinal or vitreous inflammation, as in our case.[5,6]

The fluorescein angiographic appearance of active sarcoid lesions of the choroid has been described as early blocked choroidal fluorescence followed by gradually increasing fluorescence, with homogeneous staining of the lesion in the late phases.[5-7] There have been few reports in the literature describing the ICGA findings. In a recently reported case with multiple placoid areas of choroidal pallor, corresponding to the lesions, FFA showed no early masking but late staining hyperfluorescence and ICGA revealed a marked, sustained hypofluorescence.[8] Both the fluorescein and indocyanine green angiograms of our case were similar; with no early masking but late hyperfluorescence on FFA and hypofluorescence on ICGA. Active areas of choroidal inflammation due to other causes have also been reported to be seen on ICGA as areas of hypofluorescence.[9] Hypofluorescence on ICGA is either produced by blockage of underlying dye fluorescence by pigment, haemorrhage and other materials, or is secondary to a vascular filling defect.[10] Hypofluorescence in sarcoidosis may be due to focal choroidal infiltration with inflammatory cells which displaces the extravasated dye, due to choroidal vascular hypoperfusion, occlusion or both.

Hypofluorescence due to blockage may be seen either throughout the entire study or only on late-phase images.[10] In our case hypofluorescence was observed only in early to mid-phases of the angiogram. The

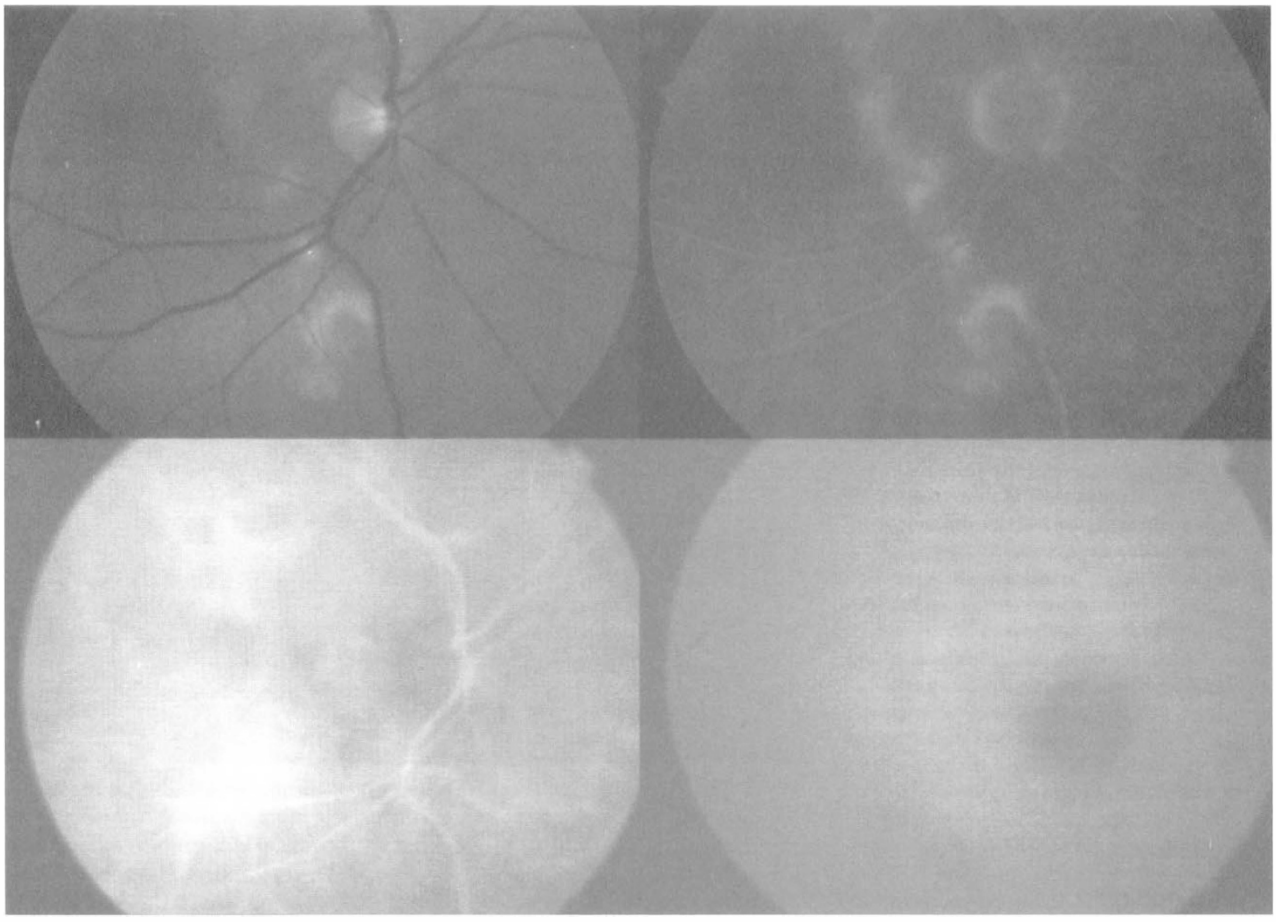

Fig. 2. *Upper left: No significant change is seen in the lesion after 2 months of therapy. Upper right: FFA, late phase. No significant change is seen after 2 months. Lower left: ICGA, mid-phase. Hypofluorescent patches are decreased in number and size. Lower right: ICGA, late phase. There is slight hyperfluorescence corresponding to the lesion.*

hypofluorescence was patchy, which may morphologically represent patches of choriocapillaris lobular infarcts.[11] These infarcts may be the result of increased resistance to flow at the inflammation area, resulting from partial closure of the vessels themselves, or increased resistance from external compression and infiltration of the perivascular space with inflammatory material. Another explanation is vasculitis of the choroidal arterioles resulting in obstruction and choriocapillaris non-perfusion. It is impossible to determine the exact cause without pathological examination; however, previous post-mortem studies revealed non-caseating granulomata within the choroid with closely associated lymphocytic vascular cuffing.[12]

In our case, ICGA seemed superior to FFA in the follow-up of choroidal involvement in sarcoidosis. There was no change on FFA after 2 months of therapy, and we have no proof that it will ultimately change; however, response to treatment was evident on ICGA as the hypofluorescent areas became smaller. It was possible to confirm the resolution of the process earlier with ICGA.

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