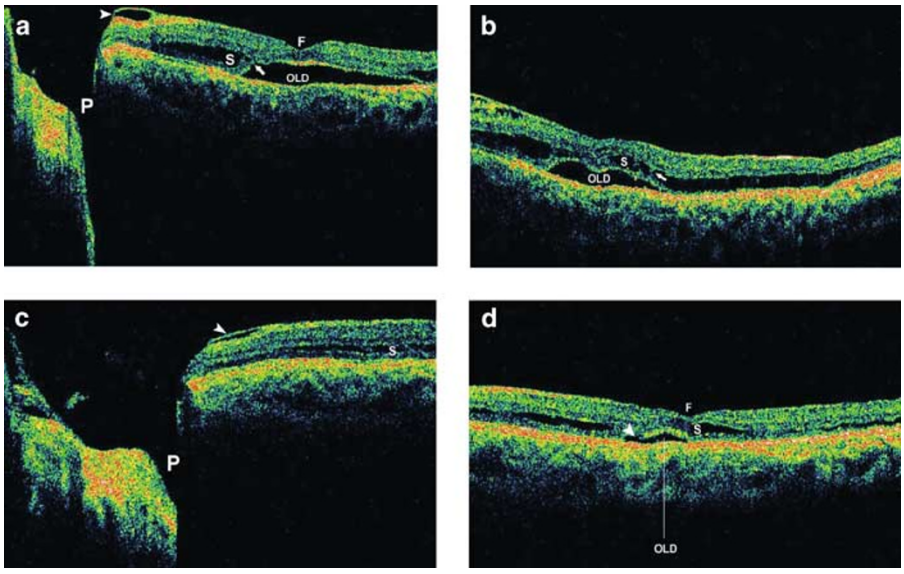

Figure 3 (**a**) Linear OCT scan of the patient at presentation, through the disc and the fovea showing the optic nerve pit (P) and the nerve fibre layer cyst (white arrow head). F denotes the fovea. Also seen are the inner layer schisis (S) communicating with the pit and the OLD in a subfoveal location. An outer layer hole is seen as a discontinuity in the nasal margin of the OLD (white arrow). (**b**) Linear OCT scan of the patient at presentation, through the fovea shows the schisis (S) extending subfoveally. The white arrow depicts the bridging septae characteristic of the schisis. (**c**) Linear OCT scan at follow-up after 1 month, through the disc and the fovea showing the optic nerve pit (P) and the schisis (S). The nerve fibre layer cyst has considerably decreased in size (white arrow head). (**d**) Linear OCT scan at presentation after 1 month, through the fovea shows the persistence of the schisis (S) underneath the foveola (F). The OLD has also decreased in size and has shifted to a nasal parafoveolar location. The outer layer hole is less obvious and is seen to have come closer to the retinal pigment epithelium (white arrow head).

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Sir,

Neovascular glaucoma and sarcoidosis

Neovascular glaucoma (NVG) occurs when new fibrovascular tissue proliferates onto the anterior chamber angle obstructing the trabecular meshwork. Retinal ischaemia is thought to be the main stimulus.[1] Sarcoidosis can lead to retinal ischaemia and neovascularisation in the setting of ischaemic vasculitis.[2] Uveitis without retinal ischaemia is a rare cause of NVG. We report the first case of NVG secondary to panuveitis of sarcoidosis in the absence of retinal ischaemia.

Case report

A 62-year-old woman with known sarcoidosis was referred from the uveitis clinic with a 2-week history of bilateral elevated intraocular pressure (IOP). A mediastinal lymph node biopsy had confirmed the diagnosis of sarcoidosis approximately 1 year prior to presentation to our clinic. Her systemic sarcoidosis required high-dose oral prednisone to control the disease. Since she suffered from prednisone-induced diabetes as well as other side effects, her treatment with prednisone was

usually tapered quickly. At the time of presentation, she was on 1 mg of prednisone and 12.5 mg of methotrexate. Over the month prior to presentation, she had experienced increased shortness of breath with a recurrence of photophobia and floaters, especially in the left eye. She had normal serum glucose levels without treatment for diabetes. Her past ocular history is of a chronic low-grade pan-uveitis with numerous flare-ups that required sub-tenons dexamethasone injections. She had been on timolol 0.5% b.i.d. OU for the past year for mildly elevated IOP bilaterally (20–25 mmHg).

On examination, her visual acuities were 6/9 right and 6/36 left. Her IOP measured 28 mmHg OD and 48 mmHg OS. Anterior segment examination revealed granulomatous inflammation with anterior chamber cells and flare (left greater than right). There was moderate rubeosis iridis on the left and neovascularisation of the anterior chamber angle on the left. Bilateral vitritis was present, more marked on the left. Fundus examination revealed cup:disc ratio of 0.2 right and 0.3 left, with widespread previously documented sarcoid-related chorioretinal lesions bilaterally (Figure 1). There was no evidence of diabetic retinopathy or retinal vein occlusion. A fluorescein angiogram was performed and revealed no evidence of retinal ischaemia (Figure 2). She was treated with oral prednisone 60 mg, and 2 weeks later had a Baerveldt tube shunt with a Vicryl tie and Sherwood slit inserted into her left eye. Postoperatively, her IOP was 10 mmHg OS. Her oral prednisone was tapered over the subsequent 4 weeks. She represented with a scleritis over the left bleb, which settled with an increase to her prednisone dose.

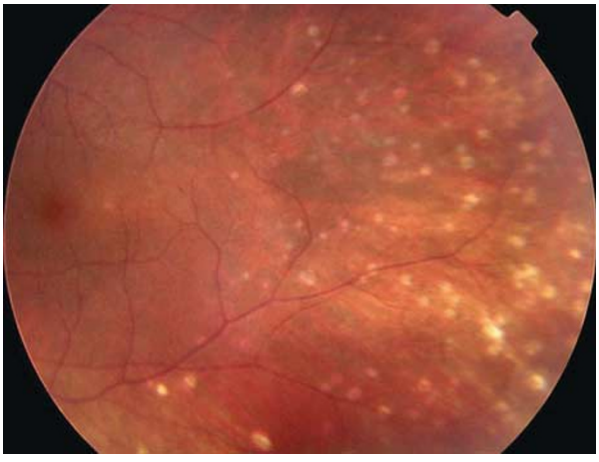

Figure 1 Fundus photograph from the left eye demonstrating sarcoid-related chorioretinal lesions

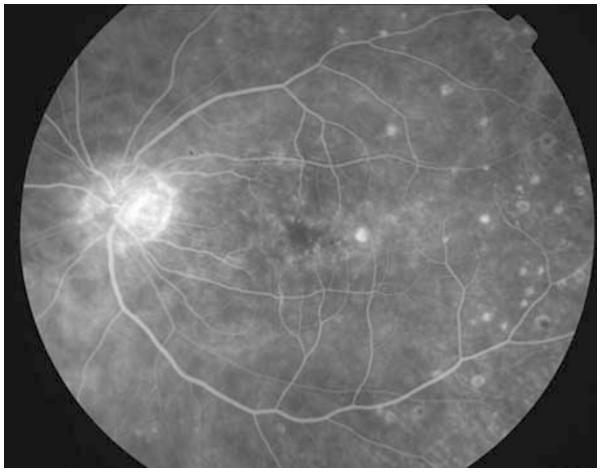

Figure 2 Late-phase fluorescein angiogram from the affected left eye of a patient with NVG showing no evidence of retinal ischaemia.

Comment

Sarcoidosis is a systemic granulomatous disorder characterised by the presence of multinucleated giant cells, epithelioid cells, and activated T-lymphocytes. This case of NVG in sarcoidosis represents, to the best of our knowledge, the first case in the English literature where rubeotic glaucoma has been seen to occur in the absence of retinal ischaemia demonstrated by fluorescein angiography. It is well documented that the stimulus for the vast majority of cases of NVG is retinal ischaemia. Brown *et al*[1] suggested retinal ischaemia is the underlying pathology in more than 97% of cases of NVG. Of the three cases of NVG secondary to uveitis in their review of 208 patients, two were vitreous wick syndrome and one was squamous cell carcinoma invading the anterior chamber. The fundus was not visualised in two cases, and therefore retinal ischaemia could not be completely excluded. Hoskins suggested 11% of cases of NVG were secondary to uveitis.[3] Many of these cases have subsequently been considered to be ocular ischaemia with an anterior chamber reaction rather than uveitis. However, uveitis and anterior segment ischaemia, particularly in the presence of chronic iridocyclitis, Fuch's uveitis syndrome, scleritis, and carotid occlusive disease have been implicated in NVG and rubeosis.[4,5] We would like to propose an alternative mechanism for how sarcoid-related uveitis, in the absence of retinal ischaemia, may lead to NVG.

The mechanism by which sarcoidosis usually results in elevated IOP is granulomatous inflammation or 'trabeculitis' and fibrosis around Schlemm's canal. However, sarcoidosis is known to produce factors that result in angiogenesis. Endothelial cell proliferation and neoformation of a diffuse capillary network have often

been found within and adjacent to sarcoid granulomas.[6] Meyer *et al*[7] have shown that macrophages from bronchoalveolar lavage in patients with active pulmonary sarcoid produce higher levels of angiogenic cytokines than do macrophages from normal patients, patients with other lung disorders, or those with inactive sarcoidosis. This was corroborated by Tolnay *et al*[6] who identified the increased transcription and production of vascular endothelial growth factor (VEGF) in activated alveolar macrophages in patients with pulmonary sarcoidosis. Sarcoidosis has been documented to result in microangiopathic lesions in various other organs,[8] where activated monocytes and macrophages have upregulated transcription of VEGF. Tripathi *et al*[9] have shown that VEGF levels are elevated in the aqueous of patients with other causes of NVG. These findings would support the idea of ocular granuloma formation and subsequent production of VEGF being associated with new vessel proliferation in sarcoid-related uveitis.

The chronic, aggressive granulomatous inflammation seen in our patient with sarcoidosis may have provided the angiogenic substrate to induce iris and angle neovascularisation in the absence of retinal ischaemia. We recommend careful iris and gonioscopic examination for the presence of new vessels in those with chronic inflammation secondary to sarcoidosis whose elevated IOP has been put down to secondary open angle glaucoma on the basis of inflammation or steroid response.

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Sir,

Spontaneous resolution of a choroidal mass

A case of spontaneous regression of a choroidal mass is presented.

Case report

An 80-year-old male was referred to the Ocular Oncology service from a tertiary referral centre in March 2003 with a 2-month history of floaters. He had been noted to have a large choroidal lesion in the superior periphery of the left eye (Figure 1a). On ultrasonography, the lesion demonstrated choroidal thickening with low internal reflectivity, suggestive of choroidal melanoma (Figure 1b). Past ocular history included bilateral uncomplicated phacoemulsification with the left eye surgery in October 2001. He was suffering from chronic obstructive pulmonary disease and had a myocardial infarction in 1972 and was on aspirin, inhalers, lansoprazole, and frusemide. There was no history of systemic malignancy.

On examination, 4 weeks after referral, visual acuity was 6/9 in both eyes. Anterior segment examination was normal with bilateral pseudophakia and an intraocular pressure of 14 mm Hg in both eyes. Dilated fundus examination of the right eye was normal. Fundus examination of the left eye showed normal optic disc and macula with areas of widespread retinal pigment epithelial (RPE) disturbance in the superior quadrant,