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Proprietary interests: None.

Informed consent: Full informed consent has been obtained from the patient concerned for publication of his clinical details and facial photographs.

Sir,

An unusual macular involvement in pathological myopia

Eye (2003) 17, 654–656. doi:10.1038/sj.eye.6700416

Pathological myopia is one of the leading causes of blindness in the world. Progressive macular degeneration is the major complication and causes visual loss. These eyes generally have refractive errors greater than 6 dioptres, and the clinical findings are related to increased axial length.¹

The degenerative changes in pathological myopia initially involve the choriocapillaris, Bruch's membrane, and retinal pigment epithelium (RPE).² Central visual function may be affected by breaks in Bruch's membrane, choroidal neovascular membranes and associated haemorrhages, serous and/or haemorrhagic detachments of the retinal pigment epithelium.

Herein, an unusual macular involvement in a case with pathological myopia is discussed with fluorescein angiography and optical coherence tomography (OCT) findings.

Case report

A 21-year-old healthy woman presented complaining of worsening visual acuity, especially in the right eye. The review of her medical story indicated that she began to wear glasses for high myopia when she was 5 years old, and she had been wearing contact lenses for 4 years. Her parents were first cousins and nobody was suffering from any eye disease in her family.

On February 1998, ophthalmic examination revealed that her best corrected visual acuity was RE 8/10 (-13.0 ($-2.0 \times 20^\circ$)) and LE 7/10 (-13.50 ($-3.75 \times 170^\circ$)). Anterior and posterior examinations were normal. On

June 2001, ophthalmological examination demonstrated a visual acuity of 6/10 in the right eye and 8/10 in the left eye with contact lenses. External examination, pupils, intraocular pressure and slit-lamp examination were unremarkable in both eyes. Fundus examination of the right eye demonstrated a small white lesion centred on the fovea (Figure 1a).

Fluorescein angiography demonstrated a horizontal ovoid zone of hyperfluorescence surrounding a nonfluorescent centre in all phases in the right eye (Figure 1b). In the left eye, a similar appearance of the macula was obtained, but it was more indistinct than the right eye.

Horizontal and vertical OCT images obtained through the fovea demonstrated a sharply defined, full thickness defect of the highly reflective (red) band corresponding to the retinal pigment epithelium and choriocapillaris. An area of increased reflectivity was observed beneath this defect. The overlying retinal layers were normal (Figure 1c).

There were pigment epithelial defects in the left macular area, and OCT scans did not demonstrate any abnormality (Figure 2a,b).

A 30 Hz flicker response of ERG reflecting right macular function showed lower amplitude in comparison to the left eye. EOG responses were subnormal in both eyes (the Arden ratio was 1.40 OD and 1.52 OS) without significant light rise and dark trough.

Visual field examination was unremarkable. Axial length was 25.71 mm in the right eye and 25.91 mm in the left eye.

Discussion

Patients with progressive elongation of the eye develop thinning of the choroid and retinal pigment epithelium in the macular area. This may be associated with the development of tilting of the optic disc, peripapillary chorioretinal atrophy, posterior staphylomata, areas of atrophy in pigment epithelium and choroid, and lacquer cracks.³ Progressive macular degeneration is the major cause of visual loss. Ruptures of Bruch membrane, subretinal neovascular membranes, haemorrhages, and serous detachment can occur in the macular area.

A rapid loss of central vision is usually caused by exudative and haemorrhagic macular detachment overlying areas of choroidal neovascularization. This may occur adjacent to a lacquer crack, in an area of geographic atrophy of RPE, or often in an area of generalized attenuation of RPE and choroid. The new vessels are small and located close to the central macular area. They characteristically appear as a faint grey

Figure 1 (a) Unusual macular lesion in the right eye. (b) Fluorescein angiography shows bull's eye maculopathy. (c) Horizontal OCT scan through the macula shows the defect in the RPE-choriocapillaris complex with increased reflectivity below.

semitranslucent plaque with a hyperpigmented border in slit-lamp fundus examination. If associated with subretinal bleeding, a small, relatively round mound of RPE proliferation may develop as the blood clears and this may obscure the subretinal membrane from view.⁴ In

Figure 2 (a) Absence of macular foveolar reflex in the left eye and (b) normal OCT scan.

young myopes, the neovascular tuft exhibits an early hyperfluorescence and late leakage confined to the neovascular membrane borders. In elderly patients, subretinal neovascularization exhibits more leakage, resulting in the extension of dye beyond the boundaries of the neovascular net that was delineated in the early dye transit.

Fuchs' spot is a raised, circular, pigmented lesion that frequently develops in the macula of 10% of people with pathological myopia. The bilateral involvement of Fuchs' spot is estimated to be 40%, and the second eye may be involved in 5 years.⁵ The characteristic appearance of Fuchs' spot is produced by hyperplasia of the retinal pigment epithelium over a subretinal membrane. A depigmented atrophic area usually surrounds this lesion. Fluorescein angiography shows central hypofluorescence because of pigment epithelial hyperplasia and hyperfluorescence of the RPE atrophy surrounding this lesion.

Localized serous detachment may be caused by a macular hole or a minute, round retinal hole that may be in the paramacular area in myopic patients with

posterior staphylomata and must be differentiated from exudative detachment of the macula.

The macular lesion in our case was a small, round, hole-like lesion with a yellow base. Fluorescein angiography showed hypofluorescence in the centre and hyperfluorescence surrounding it, and this was typical for bull's eye maculopathy. Considering the macular lesions in pathological myopia, this lesion may be a sequela of haemorrhage (Fuchs' spot?) that blocked fluorescence in the centre. There is an RPE atrophy that enables the choroidal fluorescence surrounding the lesion. However, biomicroscopy and OCT scans did not support these findings. Horizontal and vertical optical coherence tomography images obtained through the fovea demonstrated a full thickness defect of the highly reflective band corresponding to the RPE and choriocapillaris. An area of increased reflectivity was observed beneath this defect. The overlying retinal layers were normal. According to this appearance on OCT, our interpretation was a colobomatous defect of RPE and choriocapillaris in the foveal area. Fluorescein angiography findings were in accordance with this lesion. The central hypofluorescent area corresponds to the area of choriocapillaris and RPE atrophy, while the surrounding hyperfluorescence corresponds to the RPE defect only (window defect). If this lesion were a Fuchs' spot, on OCT scans, we would expect to find an irregular RPE and choriocapillaris layer with RPE atrophy surrounding it.

With these findings, we conclude that this is an unusual atrophic macular lesion of pathological myopia having different features with different diagnostic examinations. We intend to follow up this patient in order to evaluate the prognosis.

Acknowledgements

The authors do not have any proprietary interest.

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Sir,

Late onset group B streptococcus endophthalmitis associated with conjunctival filtration bleb

Eye (2003) **17**, 656–658. doi:10.1038/sj.eye.6700445

Endophthalmitis is a well-recognised late complication of glaucoma filtration surgery. The causative organisms differ from those of acute postoperative endophthalmitis. We present the first case description of late bleb-related endophthalmitis caused by *Streptococcus agalactiae* or group B Streptococcus (GBS).

Case report

A nondiabetic 86-year-old woman presented to the emergency eye service in 2001 with a 1-day history of painful, inflamed left eye associated with marked reduction in visual acuity. She was known to have bilateral advanced primary open-angle glaucoma, which had been treated with bilateral trabeculectomies without antifibrotic agents (right 1998, left 1999), and she had been bilaterally pseudophakic for 12 months. Recently, she had undergone treatment for right cystoid macular oedema. At 2 weeks before presentation, her visual acuities were 6/36 (right) and 6/24 (left), and a slow leak was detected in her left conjunctival surgical bleb. The anterior chambers were deep.

At presentation, her visual acuities were counting fingers (right) and perception of light (left). The left conjunctiva was severely injected. The bleb was opaque with marked corneal oedema and intense fibrino-cellular reaction in the anterior chamber. The intraocular