PostScript

LETTERS

Retinal pigment epithelial tear following intravitreal bevacizumab for choroidal neovascular membrane due to age-related macular degeneration

Retinal pigment epithelium (RPE) tears are an uncommon but visually damaging complication of pigment epithelial detachment (PED), caused by weak atrophic margins of chronic PED and contraction of sub-RPE fibrovascular scar tissue. RPE tears can occur spontaneously or following treatment of the choroidal neovascular (CNV) membrane. There have been reports of RPE tears following laser photocoagulation,¹ transpupillary thermotherapy,² photodynamic therapy with or without combined intravitreal triamcinolone³⁻⁴ and intravitreal Macugen.⁵ We report a case of a patient with PED with submacular haemorrhage and occult CNV who developed an RPE tear following intravitreal bevacizumab (Avastin).

Case report

A 74-year-old man presented with a 10-day history of diminished right visual acuity. The corrected visual acuities were 20/60, OD and count fingers, OS. Ophthalmoscopic examination revealed a submacular haemorrhage in the right eye and a disciform macular scar in the left eye. Fluorescein angiogram (FA) showed an actively leaking lesion nasal to an area of blocked hypofluorescence (fig 1A).

He underwent a pneumatic displacement of the subretinal haemorrhage with tissue plasminogen activator (Altipase 100 µg in 0.1 ml) and intravitreal perfluoropropane (C3F8) tamponade (100% C3F8 0.3 ml). He was examined 4 weeks later, when the haemorrhage had cleared. His visual acuity in the right eye at this stage was 20/80. FA showed an underlying occult CNV (fig 1B,D). Optical coherence tomography showed a small PED, with subretinal and intraretinal fluid and some subretinal fibrosis (fig 1C).

The patient then received an injection of intravitreal bevacizumab 1.25 mg to the right eye. Two weeks following the injection, his right visual acuity dropped to 20/320. Fundus examination now showed a well-demarcated, crescent-shaped area, in which the choroidal vessels were clearly visible. This area of RPE loss was also demonstrated on the FA showing a large window defect, flanked nasally by hypofluorescence indicating retracted and rolled-up RPE (fig 2B). The detached edge of the RPE was discernable on the optical coherence tomograph, which also showed an increased depth signal in the area of RPE loss (fig 2C).

Comment

RPE tears are thought to occur as a result of increased tangential stretching forces on weakened PE. They have been reported following both laser and injected treatments for CNV. In our case the presence of subretinal haemorrhage and early fibrosis clearly indicated some subretinal traction before the injection of Avastin. The use of a vascular endothelial

growth factor inhibitor may have caused regression of the neovascular tuft and an increase in tension. Although reports of RPE

tears following injectable treatments for CNV are rare, patients should be warned of this possible complication.

Figure 1 (A) Fluorescein angiography (FA) OD at baseline demonstrating an area of masking by subretinal haemorrhage with bright hyperfluorescence nasally consistent with choroidal neovascular (CNV) membrane. (B, D) Pre-Avastin early- and late-phase FA showing hyperfluorescence of an underlying occult CNV membrane. (C) Pre-Avastin optical coherence tomogram OD demonstrating a small pigment epithelial detachment (PED) with subretinal and intraretinal fluid.

Figure 2 (A) Colour fundus photograph OD 2 weeks after intravitreal bevacizumab showing a crescent-shaped area devoid of retinal pigment epithelium (RPE), temporal to the rolled edge of pigment epithelium. (B) Early phase fluorescein angiogram OD showing intense hyperfluorescence corresponding to the area of RPE loss flanked nasally by hypofluorescence due to retracted RPE. (C) Optical coherence tomogram OD 2 weeks after the injection of Avastin. Detached and retracted RPE is visible, as well as increased depth signal in the area of RPE loss (between arrows).

Shabeeba R Hannan,
Krishnappa C Madhusudhana, Andrew J Lotery,
Richard S B Newsom
Southampton Eye Unit, Southampton, UK

Andrew J Lotery
Human Genetics Division, University of Southampton,
Southampton, UK

Correspondence to: Dr R S B Newsom, Southampton
Eye Unit, Tremona Road, Southampton SO16 6YD, UK;
richard.newsom@suht.swest.nhs.uk

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Choroidal osteoma in association with Stargardt's dystrophy

Choroidal osteoma usually occurs in 20–30 year old, healthy, white women as a well-defined, unilateral (75%), solitary, yellow orange, slow-growing juxtapapillary lesion.¹ This is the first time that a choroidal osteoma has been associated with Stargardt's dystrophy.

Case report

A 16-year-old girl presented with bilateral reduction of distance visual acuity (6/24) since 2 years. Near vision, colour vision, stereoacuity and anterior segments of both eyes were normal. Amsler grid testing in the left eye revealed a blur of the grid superotemporal to the foveal fixation point. Stereoscopic funduscopy revealed bilateral Stargardt's maculopathy (foveolar pigment epithelial changes with parafoveal orange-coloured flecks; figs 1A,B), with an approximately 2.5 mm yellowish subretinal lesion situated 2-disc diameters below the left optic disc (fig 1B). Fundus fluorescein angiography (FFA) revealed macular and perimacular hypofluorescence (dark choroid) with hyperfluorescence and late staining of the lesion inferior to the left optic disc (figs 1C,D). A/B ultrasound echography demonstrated a corresponding 0.9 mm thick, 2.5 mm diameter lesion with high, homogeneous internal reflectivity and marked shadowing of tissue signals posterior to the lesion (fig 1E), characteristic of choroidal osteoma. Serum calcium phosphate and parathyroid hormone levels, electroretinogram, electro-oculogram and visual evoked potential tests were normal. Goldman's perimetry revealed bilateral central scotomas (fig 1F). FFA and B ultrasound scan were used to monitor the

patient for subretinal choroidal neovascular membranes (CNVM) or haemorrhage.

Comment

This is the first report of a choroidal osteoma occurring in a patient with Stargardt's maculopathy. The clinical and ultrasound features were characteristic for a choroidal osteoma as described above.

Choroidal osteomas present as unilateral yellow–white to orange–red juxtapapillary choroidal lesions, which can occasionally extend into the macula or involve both choroids.¹ The main differential diagnosis of choroidal osteoma is idiopathic sclerochoroidal calcification.

Sclerochoroidal calcification, common in 50–80 year old men, presents with bilateral (47% are unifocal), geographic, plaque-like or mildly elevated yellow–white fundal lesions along the retinal vascular arcades,^{2,3} with rare multiple grey scleral lesions that resemble the choroidal lesion.³

Choroidal osteomas, common in women, increase in size in 41% of cases, and can cause a reported secondary poor visual acuity ($\leq 6/60$) in 45% and 56% of eyes at the 5 and 10 years follow-up, respectively.⁴ Spontaneous resorption of the lesion can occur. CNVM, subretinal fluid and haemorrhages complicate 33% of cases. Quantitative CT scans have demonstrated occult lesions in 90% of fellow eyes of clinically

Figure 1 (A,B) Fundus photographs of right and left eyes revealing bilateral bull's eye maculopathy and left juxta papillary calcific lesion (arrow). (C,D) Fundus fluorescein angiography of right and left eyes. Bilateral dark choroids and hyperfluorescent juxtapapillary lesion in left eye. (E) B ultrasound scan reveals shadowing posterior to the highly dense lesion (A ultrasound scan: sound attenuation posterior to the lesion). (F) Perimetric central field defects. MD, mean deviation.