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CORRESPONDENCE

Sir,

**Spontaneous involution of choroidal neovascularization secondary to rubella retinopathy: reply to Veloso, Costa, Oréfice, and Oréfice**

We have read with great interest the paper by Veloso, Costa, Oréfice, and Oréfice[1] about spontaneous involution of choroidal neovascularization secondary to rubella retinopathy. The authors have presented to the scientific literature a very interesting and unusual case of bilateral choroidal neovascularization in a 10-year-old girl, with apparent good ocular health since birth, until 4 weeks before presentation, when she developed retinal epithelium mottling in both eyes (probably from birth) and an active subfoveal choroidal neovascular membrane in the right eye and an involutional similar lesion in the left eye. These findings were attributed to the fact that her mother contracted rubella during pregnancy.

In Brazil, where this patient was probably seen by the authors, the main ocular findings among infants born from mothers who contracted rubella during pregnancy are congenital cataract or a complete congenital rubella syndrome, which includes systemic and ocular manifestations like retinopathy with retinal epithelium mottling (as described in the correspondence by the authors), congenital cataract, glaucoma, iris hypoplasia, strabismus, microphthalmos, corneal leucoma, and even eye atrophy. Systemic complications of the rubella syndrome can also cause neurological, auditive, and cardiologic impairments in many patients.[2,3] Previous article from Brazil related congenital cataract in 42.7% among patients with systemic infections and around 38% of those patients had cataract formation secondary to congenital rubella.[4]

The decision assumed by the authors in only performing periodical observations of the patient's natural evolution without using any of the available treatments for subfoveal choroidal neovascularization including photodynamic therapy was really a correct decision. The current knowledge among researchers on subfoveal choroidal neovascularization in young patients secondary to inflammatory diseases as occurs in ocular toxoplasmosis, histoplasmosis, progressive subretinal fibrosis, and other similar situations states that these lesions normally show a natural involution of the neovascular complex even without any treatment.[5] The reason for this remains unclear, as stated by the authors in the correspondence.

I wish to congratulate the authors for this really interesting contribution.

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Sir,

**Intravitreal triamcinolone and bevacizumab combination therapy for refractory choroidal neovascularization with retinal angiomatous proliferation**

A review of the literature showed no published cases using a combination of intravitreal triamcinolone acetonide (IVTA) and antivascular endothelial growth factor agents for choroidal neovascularization (CNV) with associated retinal angiomatous proliferation (RAP).[1–5] We present a case of RAP with a pigment epithelial detachment (PED) refractory to multiple treatment modalities, but which responded to the combination of intravitreal triamcinolone (Kenalog, Bristol-Myers-Squibb, Peapack, NJ, USA) and intravitreal bevacizumab (Avastin; Genentech, San Francisco, CA, USA).

An 80-year-old woman with bilateral AMD was referred for treatment of CNV. Her vision was 20/150 right eye (OD) and counting fingers at 5 feet left eye (OS). Clinical examination showed bilateral fibrovascular PEDs with overlying small coin-shaped geographic atrophy both eyes (OU). There was intraretinal haemorrhage and lipid associated with the PEDs. Fluorescein angiography and indocyanine green angiography showed leakage from minimally classic CNV with RAP lesions OU (Figure 1a and b). Optical coherence tomography (OCT) showed a PED, cystoid macular oedema (CME), and subretinal fluid OU (Figure 2).

Over the previous 9 months before referral, the patient had been treated with three sessions of verteporfin photodynamic therapy (PDT), the last combined with intravitreal triamcinolone. Over the next 20 months, she underwent multiple treatments, including two PDTs with IVTA, pegaptanib (Macugen, Eyetech, New York NY, USA) OD, four bevacizumab OD, two bevacizumab OS, one ranibizumab (Lucentis; Genentech, San Francisco, CA, USA) OD and three ranibizumab OS. After the third bevacizumab OD, the OCT showed a flat PED, vision improved to 20/70, and the PED remained flat without further treatments for 9 months. However, the CME in the left eye continued to worsen on OCT despite the last three ranibizumab injections (Figure 3a). After a discussion with the patient on combination therapies, intravitreal

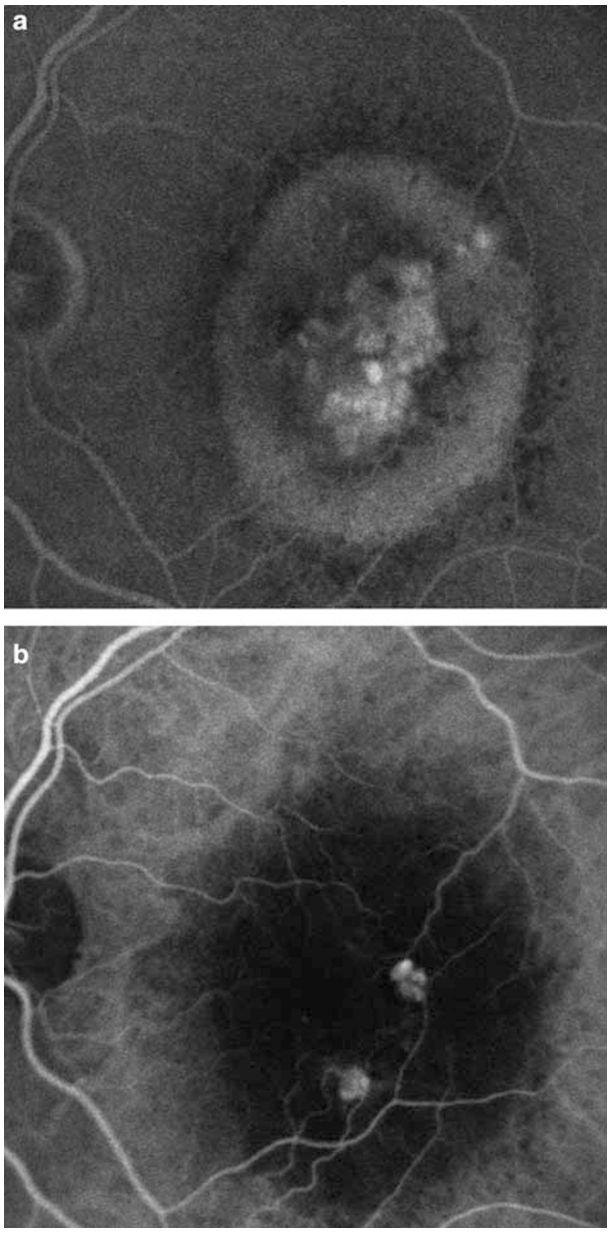


**Figure 1** (a) Left eye: Late-phase fluorescein angiogram showing minimally classic CNV. (b) Left eye: Indocyanine green angiogram showing the RAP lesions within CNV.

**Figure 2** Left eye: OCT showing a PED, cystoid macular oedema, and subretinal fluid with CNV.

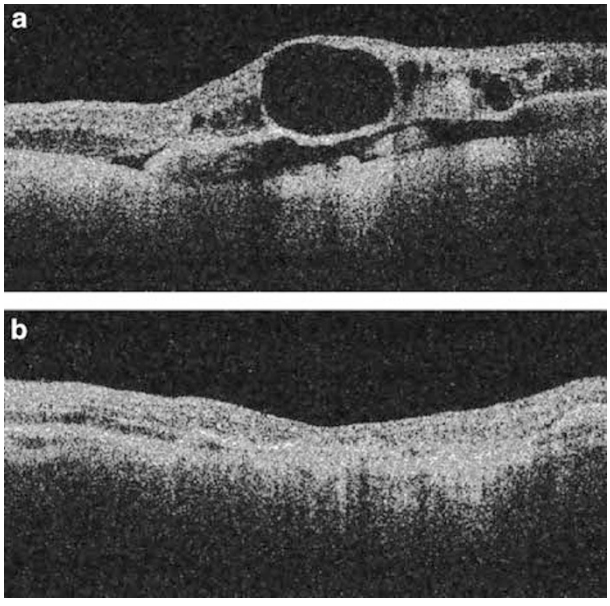


**Figure 3** (a) Left eye: Optical coherence tomography showing a pigment epithelial detachment, worse cystoid macular oedema, and subretinal fluid. (b) Left eye: Optical coherence tomography showing that the pigment epithelial detachment, cystoid macular oedema, and subretinal fluid have resolved after combination therapy with intravitreal triamcinolone and bevacizumab.

triamcinolone 2 mg (0.05 ml) and intravitreal bevacizumab 1.25 mg (0.05 ml) were given. This dose allowed us to give a single 0.1-ml injection without the need for a paracentesis. One month later, the CME had resolved and the PED was flat (Figure 3b). The vision remained at counting fingers due to the areas of geographic atrophy. This effect was maintained the last follow-up 5 months later with no adverse treatment effects.

Intravitreal bevacizumab and triamcinolone are used off-label as treatment options for CNV. This case illustrates that combination therapy with intravitreal triamcinolone and bevacizumab may be considered as an option for treatment of CNV.

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Sir,

**Bilateral basal cell carcinoma of the lower eyelids following radium treatment for blepharitis**

Approximately 5–10% of all skin cancers occur in the eyelid.[1] Basal cell carcinoma (BCC) is the most common malignant tumour of the eyelids. The most important environmental risk factor is repeated intense exposure to the sun, particularly second-degree solar burns during childhood.[2]

We present a case of bilateral lower lid BCC following therapeutic radium treatment for blepharitis in childhood.

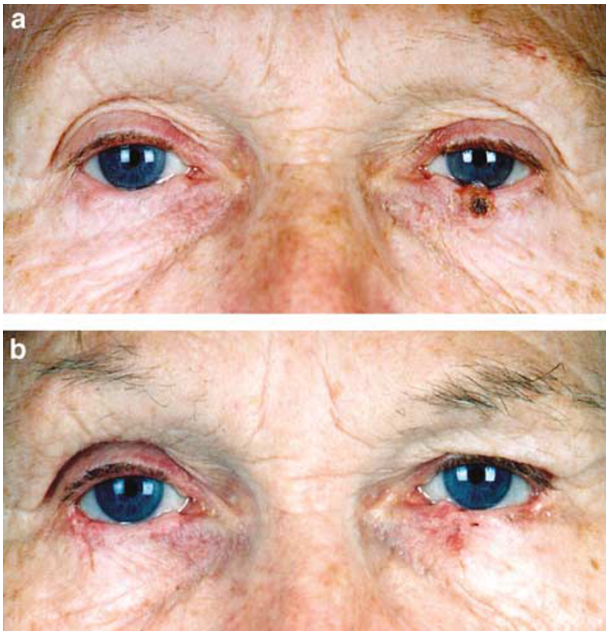


**Figure 1** (a) Preoperative photograph showing pearly ulcerated nodule on left lower lid and smaller nodule on lateral half of right lower lid. (b) Appearance at 10 days postoperatively after removal of sutures. Although there is still some residual erythema, there is a satisfactory cosmetic result.

*Case report*

A 71-year-old lady presented with a 2-year history of slow growing bilateral lower lid lesions. She had an ulcerated nodule with pearly raised edges on her left lower lid measuring 7 mm across and a smaller nodule on her right lower lid measuring 3 mm across (Figure 1a).

Her past ocular history was of particular interest. She had suffered from severe blepharitis in childhood and recalled a trial of radium irradiation for her condition at the age of 13. This took the form of six treatments over 6 weeks. 70 kv photons were used and she received 300 R in total (ie 50 R per treatment). The eyelid was held back and lead foil was used to protect the eye.

The clinical diagnosis was bilateral lower lid BCC. She underwent bilateral pentagon excision biopsies with a left semicircular flap. Histology confirmed nodulo-ulcerative BCC (Figure 2). All margins were clear of tumour.

The patient had an uneventful postoperative recovery and was satisfied with the cosmetic result. (Figure 1b).

**Comment**

Basal cell carcinoma is the most common malignant tumour of the eyelid. [3] The treatment of choice for

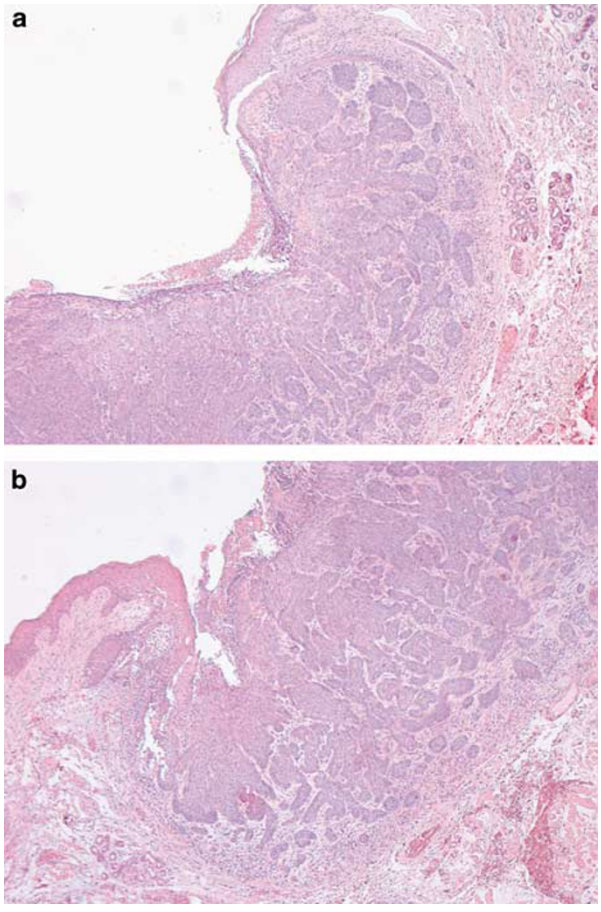


**Figure 2** Histological specimens of left (a) and right (b) lower lid excision biopsy specimens showing BCC with dilated glandular ducts and inflammation associated with ulceration.