Eye (2008) 22, 978–981

www.nature.com/eye

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.

References

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Eye (2008) **22,** 978; doi:10.1038/sj.eye.6703037; published online 23 November 2007

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