Case report

Rapid “epiretinal membrane” development following intravitreal bevacizumab for Coats’ disease

Andrew W. Kam, Michelle Hui, Svetlana Cherepanoff, Adrian T. Fung

Abstract

Purpose: To report a case of rapid “epiretinal membrane” (“ERM”) development following intravitreal bevacizumab for juvenile Coats’ disease.

Observations: A 7-year-old boy was followed for four years with asymptomatic stage 2 Coats’ disease in his left eye. At age 11, he developed symptomatic cystoid macular edema. Argon laser photocoagulation to the leaking aneurysms failed to improve his vision, which had symptomatically declined to 20/30. Four-months after laser, a single injection of intravitreal bevacizumab was given. Rapid development of an “ERM” was noticed on his first post-injection follow-up at 4 weeks. By 8-weeks post-injection the visual acuity had deteriorated to 20/400. 25 + gauge pars plana vitrectomy with “ERM” peeling was performed, with recovery of vision to 20/30 at the 4 months post-operative visit.

Conclusions and importance: Intravitreal bevacizumab may induce rapidly progressive “ERM” in patients with juvenile Coats’ disease.

1. Introduction

Coats’ disease is a sporadic, typically monocular disorder characterised by retinal telangiectasias (Stage 1) which can progress to lipid exudation (Stage 2) and retinal detachment (Stages 3–5) in late disease.1–3 It has a strong male predominance (3:1) and although it may be recognised at any age, most cases present within the first decade of life.5,6

Management depends on disease severity. Laser photocoagulation and/or cryotherapy to the telangiectatic vessels is recommended when lipid exudation threatens the macula. In recent times, anti-vascular endothelial growth factor (VEGF) agents have also been used as an adjuvant to treat Coats’ disease. This is based on the observation that VEGF is known to be markedly elevated in eyes affected by Coats’ disease.1–4 We describe a potential adverse effect - rapid development of “epiretinal membrane” (“ERM”) following a single injection of intravitreal bevacizumab in a patient with Stage 2 juvenile Coats’ disease. Informed consent for this report was provided by the patient and his parents.

2. Case report

A 7-year-old boy with good general health was referred with telangiectatic vessels, aneurysms and intraretinal lipid temporal to his left fovea confirmed on optical coherence tomography scans (OCT, Fig. 1A). The rest of the ocular examination including the right eye was normal and given the male gender, age and clinical findings a diagnosis of Stage 2 Coats’ disease was made. Since he was asymptomatic and the visual acuity was 20/20, the patient and his parents opted for close observation.

The patient was followed twice a year for three and a half years, during which time his disease remained stable. However, at four years he started to notice blurring of his vision which had dropped to 20/30 due to development of cystoid macular edema (Fig. 1B). Fundus fluorescein angiography was attempted but aborted due to difficulty with cannulation. Argon laser photocoagulation was applied to the
temporal aneurysms and affected retina but four months later following this there was still no resolution of the edema and his vision had worsened to 20/60 (Fig. 1C). An OCT raster through the fovea showed trace thickening of the internal limiting membrane/“ERM”. A single injection of intravitreal bevacizumab (Avastin; Genentech, San Francisco, CA, 1.25mg/0.05mL) was given under topical and sub-conjunctival anaesthesia.

At the 4-week post-injection follow-up visit the vision had dropped to 20/150 and a dense “ERM” was noted at the macula (Fig. 1D). At 8 weeks post-injection the vision had deteriorated to 20/400 with further progression of the “ERM”. The central foveal thickness had increased from 333μm at the time of the bevacizumab injection to 579μm. The patient underwent 25-gauge pars plana vitrectomy with “ERM” and internal limiting membrane (ILM) peeling using trypan blue dye (0.06%). The posterior hyaloid was found to be extremely thickened. Supplemental endolaser was applied to the temporal and inferior aneurysms, telangiectasias and retina. Interestingly, histopathological analysis demonstrated highly folded, paucicellular membrane favouring ILM. Four months post-operatively the macular edema had largely resolved with improvement of vision back to 20/30 and reduction in central foveal thickness to 270μm (Fig. 1E).
therapy to be associated with ERM development and progression.8 In our case, following surgical intervention, the patient’s vision recovered to 20/30 after 4 months. Although management of ERM via vitrectomy and membrane peeling in the setting of Coats’ disease has been reported rarely,6,7 it was felt that expedient management would likely prevent the permanent macular changes that may be seen if ERMs are left untreated for longer periods.6,7 This timely intervention is likely to have prevented more severe visual acuity deterioration in our patient.

In summary, we report a case of rapid macular “ERM” development following intravitreal bevacizumab for juvenile Coats’ disease. Caution is advised when considering anti-VEGF agents for the management of this disease.

Patient consent

The patient and his parents provided written informed consent for the report.

Funding

No funding or grant support.

Conflicts of interest

The following authors have no financial disclosures: AWK, MH, SC, ATF.

Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ajoc.2018.06.002.

References

1. Ramasubramanian A, Shields CL. Bevacizumab for Coats’ disease with exudative retinal detachment and risk of vitreoretinal traction. Br J Ophthalmol. 2012;96:356–359.
2. Shields JA, Shields CL. Review: coats disease: the 2001 LuEsther T. Mertz lecture. Retina. 2002;22:80–91.
3. Sigler EJ, Randolph JC, Calzada JD, Wilson MW, Haik BG. Current management of Coats disease. Surv Ophthalmol. 2014;59:30–46.
4. Rishi P, Rishi E, Uparkar M, et al. Coats’ disease: an Indian perspective. Indian J Ophthalmol. 2010;58:119–124.
5. Shields JA, Shields CL, Honavar SG, Demirci H, Cater J. Classification and management of Coats’ disease: the 2000 proctor lecture. Am J Ophthalmol. 2001;131:572–583.
6. Kumar P, Kumar V. Vitrectomy for epiretinal membrane in adult-onset Coats’ disease. Indian J Ophthalmol. 2017;65:1046–1048.
7. Shukla DMS, Chakraborty SMS, Behera UCMS, Kim RDNB. Vitrectomy for epimacular membrane secondary to adult-onset Coats’ disease. Ophthalmic Surg Las Im. 2008;39:239–241.
8. Ghaseem Falavarjani K, Nguyen QD. Adverse events and complications associated with intravitreal injection of anti-VEGF agents: a review of literature. Eye (Lond). 2015;27:787–794.
9. Martinezena J, Romano MR, Heimann H, et al. Intravitreal bevacizumab for retinal vein occlusion and early growth of epiretinal membrane: a possible secondary effect? Br J Ophthalmol. 2011;95:391–395.
10. Zhang Q, Qi Y, Chen L, et al. The relationship between anti-vascular endothelial growth factor and fibrosis in proliferative retinopathy: clinical and laboratory evidence. Br J Ophthalmol. 2016;100:1443–1450.
11. Chu SJ, Zhang ZH, Wang M, Xu HF. Effect of bevacizumab on the expression of fibrosis-related inflammatory mediators in ARPE-19 cells. Int J Ophthalmol. 2017;10:566–571.
12. Zhang M, Chu S, Zeng F, Xu H. Bevacizumab modulates the process of fibrosis in vitro. Clin Exp Ophthalmol. 2015;43:173–179.