**Intravitreal bevacizumab in choroidal neovascularization associated with Best’s vitelliform dystrophy**

Dear Editor,

We read with interest the report by Rishi et al.\(^1\) We wish to report our experience in a similar but younger patient with Best’s disease complicated by choroidal neovascularization (CNV), being treated by intravitreal bevacizumab (IVBe).

A 10-year-old boy presented to a tertiary eye care hospital in north India with reduced vision in the right eye of 2-week duration. Best-corrected visual acuity (BCVA) was 20/200 in the right eye and 20/25 in the left eye.

Fundus examination of the right eye revealed a large, hypopigmented, egg yolk-like subfoveal lesion with fresh subretinal hemorrhage [Fig. 1a]. These clinical findings were suggestive of the choroidal neovascular membrane (CNVM) in the right eye. Left eye fundus examination also revealed a hypopigmented, egg yolk-like lesion at the fovea, characteristic of the vitelliform stage of Best’s disease. Fundus fluorescein angiography (FFA) revealed early hyperfluorescence with intense late leakage confirmatory of the CNVM in the right eye, along with hypofluorescence corresponding to the subretinal hemorrhage [Fig. 1b]. Optical coherence tomography (OCT) of the right eye confirmed the presence of the subretinal fluid (SRF), cystoid macular edema, disorganization of the retinal pigment epithelium–choriocapillaris complex corresponding to the CNVM [Fig. 1c].

Electro-oculogram showed Arden’s ratio to be 1.11 in both the eyes (normal is more than 1.85).

He was treated with 1.25 mg/0.05 ml IVBe after obtaining an informed (written) consent from the parents. At 6-week follow-up, BCVA in the right eye improved to 20/100. Fundus examination of the right eye revealed regression of CNVM with marked resolution of subretinal hemorrhage. OCT revealed markedly reduced SRF and increased fibrosis of CNVM as compared to the previous visit [Fig. 2]. A second IVBe was given at 6 weeks following the first one. His BCVA improved to 20/25 at 6 weeks after the second injection and was maintained till 24-month follow-up. Fundus photograph, FFA, and OCT demonstrated regression of the CNV, resolution of subretinal hemorrhage, and SRF [Fig. 3a–c]. BCVA also improved to almost normal without any recurrence during further follow-up (2 years).

As discussed by Rishi et al., CNVM is a rare complication of Best’s disease in children.\(^1,2\) Various treatment modalities for CNV in Best’s disease have been reported in the form of observation,\(^3\) photodynamic therapy, laser photocoagulation, and IVBE with triamcinolone.\(^4,5\) There is an obvious lack of consensus regarding the management of this condition. The most important limitation of laser photocoagulation is being not suitable for subfoveal CNV. On the other hand, PDT with verteporfin with or without intravitreal triamcinolone acetonide though effective, is limited by its high cost.

Leu et al. has reported favorable results of bevacizumab in a 13-year-old boy with Best’s disease with CNV.\(^4\) Cakir et al. administered combination therapy of bevacizumab and triamcinolone and reported favorable results without any adverse events.\(^5\)
We fully agree with Rishi et al. that IVBe as a monotherapy is a promising, cost-effective, and most suitable modality of treatment in this disorder with a potential for improvement in visual acuity, although with inherent risks associated with intravitreal injections.

In our case, the vision improved with two injections and was maintained till 2-year follow-up without any adverse events.

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