Case of recurrent vitreous hemorrhage in Eales disease managed successfully with Intravitreal Bevacizumab.

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Abstract

A 23 year old male with past history of recurrent uncontrolled vitreous hemorrhage in both eyes, undergone vitrectomy and laser photocoagulation in both eyes presented with sudden diminution of vision in right eye since 2 days. On examination his BCVA was 20/ 2000 and 20/30 in right and left eye respectively. Indirect ophthalmoscopy showed fresh vitreous hemorrhage with no fundus view in right eye, B scan ultrasonography of right eye was suggestive of the same with no evidence of retinal detachment. Patients right eye was injected with 0.05 ml (1.25mg) bevacizumab (Avastin) intravitreally. At 1 month follow up his vision had recovered to 20/30 in right eye. A fundus fluorescein angiogram did not show any evidence of neovascularization. On 3 month and 1



Fig. 1: Red free photograph of left eye fundus post injection Avastin showing chorioretinal atrophic scar along superior arcade.

year follow up there was no recurrence of neovascularization and vision has been stable in both eyes. We highlight the use of intravitreal bevacizumab in management of recurrent vitreous hemorrhage in Eales disease.

Introduction

Eales disease is an idiopathic obliterative vasculopathy that usually involves the peripheral retina of young adults. In 1880, Henry Eales first described it in healthy young men with abnormal retinal veins and recurrent vitreous hemorrhages. Clinical findings are characterized by ischemic areas in the retinal periphery, followed posteriorly by micro aneurysms, dilatation of capillary channels, tortuosity and sheathing of neighboring vessels, and recurrent spontaneous vitreous hemorrhages.1,2 It is a diagnosis of exclusion, as many



Fig 2: FA picture of left eye post injection Avastin showing no evidence of neovascularization.

other retinal disorders can mimic Eales disease, especially conditions of retinal inflammation or neovascularization such as retinal vein occlusion and proliferative diabetic retinopathy. The pathophysiology of Eales disease is mostly unknown. It is believed to be a primary, inflammatory disorder of the walls of peripheral retinal vessels, namely the shunt vessels. This often leads to vascular occlusions, peripheral neovascularization, and vitreous hemorrhage. Vascular endothelial growth factor (VEGF) is established to be the primary mediator for the microvascular abnormalities seen at the junction of perfused and nonperfused zones of the retina.3,4

Several studies regarding the management of Eales disease in various stages have been reported. Yet, in the absence of a randomized controlled clinical trial, a definite guideline is not available. Management options include the following: observation, medical (oral steroids), photocoagulation, and vitreoretinal surgery. A particular patient may require one or more of the above modalities of treatment, as quite frequently the other eye has a different stage of the disease. Often, multiple or repeated treatments fail to control the disease or its recurrence. Bevacizumab is a humanized monoclonal antibody that inhibits VEGF and is effectively used as a treatment modality in variety of ocular conditions such as neovascular age-related macular degeneration, retinal vein occlusion and proliferative diabetic retinopathy. We highlight the use of intravitreal bevacizumab in management of recurrent vitreous hemorrhage in Eales disease.

Case Report

A 23 year old male presented with complaint of sudden diminution of vision in right eye since 2 days. He had a past history of recurrent uncontrolled vitreous hemorrhage in both eyes for which he had undergone vitrectomy and laser photocoagulation in both eyes 2 years back and repeat vitrectomy with Endolaser in right eye 1 year back. Postoperatively he had good visual recovery. On examination his BCVA was 20/2000 and 20/30in right and left eye respectively. Anterior segment examination in both eyes revealed no significant abnormality. The Intraocular Pressure in both eyes was within normal limits. Examination with indirect ophthalmoscopy showed fresh hemorrhage in the vitreous cavity precluding any fundus view in right eye, whereas left eye showed old photocoagulation scars in the peripheral retina. B scan ultrasonography of right eye was suggestive of the same with no evidence of retinal detachment.

We then injected 0.05 ml (1.25mg) bevacizumab (Avastin) intravitreally in right eye with repeat examination after 1 month. At 1 month follow up his vision had recovered to 20/30 in right eye. A fundus fluorescein angiogram did not show any evidence of neovascularization Fig 2. On 3 month and 1 year follow up there was no recurrence of neovascularization and vision has been stable in both eyes.

Discussion

Pathogenesis of Eales disease involves extensive retinal nonperfusion zones leading to release of VEGF resulting in neovascularization. This may explain the beneficial effect of bevacizumab in the resolution of vitreous hemorrhage and stabilization of the disease process. However, long term studies using antiangiogenic agents in Eales disease need to be done to establish it as a definitive treatment modality.

This case report may give a new direction to the management of neovascularization and its sequale in Eales disease, a quite common entity in Indian subcontinent.

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