

Radiation Retinopathy/Maculopathy: A Case Report

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A 29-year-old male presented to OPD with complaint of decreased vision in Left eye. About 6months back he was diagnosed with Myxofibrosarcoma of the left zygomatic bone. Patient underwent radiation therapy for the tumor. 3 months after the last session of radiotherapy, patient experienced decreased vision in left eye. VA was 6/6 OD and 6/36 OS. Anterior segment examination revealed madarosis, trichiasis and conjunctival congestion of left eye. Fundus examination of the left eye showed cotton wool spots, intraretinal hemorrhages and macular edema. Patient was given intravitreal injection Bevacizumab (Avastin) 1.25mg/0.05ml in the left eye. After 4 weeks of the injection VA was improved to 6/9 in left eye. Patient was asked to have a monthly follow-up. On the 5th follow-up vision was again reduced to 6/18 OS. OCT revealed increased macular thickness. Patient was again advised intravitreal injection Bevacizumab. 3 injections were given monthly. Vision improved to 6/6 OS and patient is still on follow-up for 18 months. Anti-VEGF Bevacizumab is effective in the treatment of radiation associated macular edema.

Key Words: Radiation retinopathy Bevacizumab, intraretinal hemorrhages.

Radiation has been used therapeutically for the treatment of neoplastic lesions for over 100 years. Stallard first described ocular complications following radiation therapy in 1933¹. Radiation retinopathy can occur following either external-beam or local plaque therapy. The disease process is slow and onset often occurs months or years after the initial exposure to radiation². Characteristics of radiation retinopathy include cotton wool spots, retinal microaneurysms, intraretinal hemorrhages, macular edema, telangiectasia, exudates, perivascular sheathing, optic disc edema and atrophy. The clinical features of radiation retinopathy resemble that of diabetic retinopathy. The underlying pathology is also the same i.e. microangiopathy leading to micro vascular leakage and occlusion. The primary cause of vision loss is usually due to exudative or ischemic maculopathy.

CASE DESCRIPTION

A 29-year-old male presented to OPD with complaint of decreased vision in Left eye. The past history

revealed that he was diagnosed with Myxofibrosarcoma of the left zygomatic bone about six months back. The tumor was indenting the left orbital floor. Patient underwent Intensity - Modulated Radiation Therapy (IMRT) for the tumor. Total of 30 sessions of radiotherapy was given over 1 month period with the dosage of 25 Greys. According to the patient left eye could not be covered during the therapy as the orbital floor had to be irradiated. 3 months after the last session of radiotherapy, patient

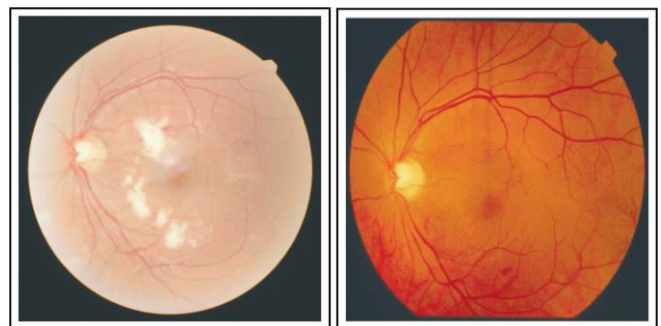


Fig. 1: Pre Treatment.

Fig. 2: Post Treatment.

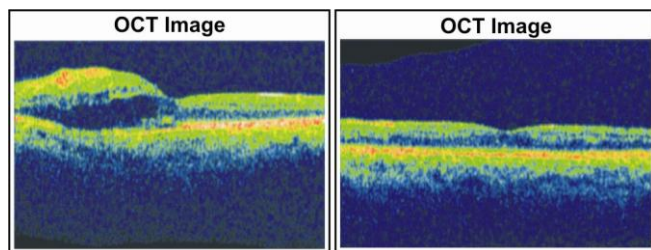


Fig. 3: Pre-Treatment.

Fig. 4: Post Treatment.

experienced decreased vision in left eye. On examination VA was 6/6 OD and 6/36 OS. Anterior segment examination revealed madarosis, trichiasis and conjunctival congestion of left eye. Conjunctival congestion was due to the dry eye caused by disruption of the goblet cells of conjunctiva. Fundus examination of the left eye showed cotton wool spots, intraretinal hemorrhages and macular edema. Fundus photos were taken and OCT was done which confirmed macular edema of 600 μ m and subretinal fluid accumulation (Fig. 1, 3). Patient was given intravitreal injection Bevacizumab (Avastin®) 1.25 mg/0.05 ml in the left eye. After 4 weeks of the injection VA improved to 6/9 in left eye. OCT was done which showed partial resolution of macular edema. Macular thickness was reduced to 370 μ m. Patient was given topical Nepafenac eye drops 4 times a day and was asked to have a monthly follow-up. The visual acuity remained stable for 8 weeks and then the patient was lost to follow-up. Five months after patient again presented to our OPD with complaint of decreased vision. Visual acuity was reduced to 6/18 OS. OCT revealed increased macular thickness of about 500 μ m. Patient was again advised intravitreal injection Bevacizumab. Three injections were given on a monthly basis. Post treatment vision improved to 6/6 OS with complete resolution of macular edema, intraretinal hemorrhages and cotton wool spots (Fig. 2). OCT shows normal macular thickness (Fig. 4). Patient is still on follow-up for 18 months and vision is stable.

DISCUSSION

The development of radiation retinopathy has many variables. It depends on total dose of the radiation, fraction numbers and size as well as the location. 35 Greys has been accepted as the upper limit of the safe dose. However cases of radiation retinopathy have been described with doses lower than this as in our case (25 Greys). Many studies have been performed to understand the pathophysiology of the disease. Egbert

et al, described the pathological changes in 1980. They demonstrated that there is thickening of the arteriolar and capillary walls due to deposition of the fine fibrillary material within and outside the wall³. They proposed that occlusion occurs due to the narrowing of the vessel lumen³. In 1987 Irvine et al reported focal loss of capillary endothelial cells and pericytes resulting in edema in 11 primates after radiation therapy⁴. They postulated that as more capillaries become incompetent, retinal ischemia follows. Retinal ischemia leads to neovascularization and finally neovascular glaucoma⁴.

Several treatment modalities have been used for the treatment of radiation retinopathy. Hyken et al, studied the effect of focal laser on radiation induced macular edema. He concluded that though there is modest improvement in visual acuity and resolution of macular edema in the initial 6 months of the treatment, no significant difference in visual acuity was found at 2 year follow-up compared to observation group⁵. Different other techniques have been proposed for the treatment. This includes the use of oral pentoxifylline, intravitreal triamcinolone, verteporfin photodynamic therapy, pan - retinal photocoagulation for proliferative disease and hyperbaric oxygen^{6,7,8,9}. All of these treatment modalities have not proven to have long lasting effects on visual acuity and macular edema. With the advent of new modalities targeting vascular endothelial growth factor (VEGF), these agents have been used for the treatment of maculopathy and also for proliferative radiation retinopathy. Three main agents have been used in previous studies, including Bevacizumab, Ranibizumab and Pegaptanib sodium. Studies have shown that Anti-VEGF therapy was associated with initial decrease in capillary permeability¹⁰. It is evidenced by resolution of hemorrhages and exudates. Finger et al concluded that continuous therapy with anti-VEGF agents produces a preserved vision and sustained response for up to 10 years¹¹. In our case also there was initial decrease in the capillary permeability and macular edema, which was proven by clinical examination and OCT. After the initial decrease in edema, there was again gradual increase in the macular edema and vascular permeability as evidenced by increased retinal hemorrhages and OCT. Regular monthly dose of intravitreal Bevacizumab for 3 months provided a sustained response, which is maintained even after 18 months. Though results of Anti-VEGF agents are promising in the treatment of radiation retinopathy,

yet long term multi – center collaborative efforts will be needed to design the clinical trials with sufficient statistical power to evaluate the safety, efficacy, and role of these emerging pharmacotherapeutic agents.

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