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Editorial

A Brief Editorial on Clinical Presentation and Treatment of Radiation Retinopathy Following Plaque Brachytherapy of Choroidal Melanoma

Editorial

Treatment of choroidal melanoma with radiation plaque brachytherapy (Iodine, (I^{125}) avoids enucleation, salvages the globe, preserves the vision, achieves better local tumor control and prevents metastasis [1,2]. However, it can lead to complications such as keratitis, iris neovascularisation, cataract, radiation optic neuropathy, retinopathy, maculopathy and vitreous hemorrhage. Radiation retinopathy (RR) is one of the expected, common complications following external beam radiation, plaque brachytherapy of choridal melanoma, retinoblastoma, choroidal metastasis and orbital tumors.[1,2] The common risk factors in the development of RR are posterior choroidal melanoma nearer to the fovea (< 3 mm from fovea), large choroidal tumors (base > 10mm), tumors limited to the choroid with no anterior uveal involvement, high radiation dose, use of radioisotope Iridium (Ir^{192}) over I^{125}, co-existing diabetes mellitus, hypertension, and use of radiosensitisers (such as chemotherapy) [1-3]. The severity and incidence of radiation retinopathy depends on size and location of the tumor, type of radiation used, method of delivery, total radiation dose and the fractionation scale.

Early studies by Collaborative Ocular Melanoma Study (COMS) for the treatment of choroidal melanoma used the radiation dose of 85 Gy followed by dose adjustments of upto 75 Gy (depending on apical thickness, basal dimensions and tumor location). Radiation retinopathy can occur in more than half of the patients receiving > 65 Gy. RR occurs at a mean interval of 26 months following plaque treatment, but can occur even after 10 to 15 years of radiation treatment [6-8].

Non-proliferative radiation retinopathy is characterized by microaneurysms, telengiectasia, retinal hemorrhages, hard exudates and infarcts. Proliferative radiation retinopathy has above findings along with the presence of retinal or optic disc neovascularisation, choroidal neovascularization, retinal artery/vein occlusion and vitreous hemorrhage. Histopathologically in RR, there is damage to the retinal vascular endothelium with relative sparing of the pericytes, subsequent damage to the inner retinal layers and retinal pigment epithelium. The unequivocal loss of endothelial cells leads to retinal vascular occlusion thereby causing irreversible vision loss [1-4].

Treatment of radiation retinopathy remains challenging. Photodynamic therapy, laser photocoagulation, oral pentoxyphylline, intravitreal steroid therapy and hyperbaric oxygen are used in the past with limited response [4-9]. Laser photocoagulation limited the progression of retinal neovascularisation and edema in patients with RR [8]. Focal argon laser photocoagulation regresses the radiation induced vasculopathy and pan retinal laser photocoagulation prevents radiation induced neovascular glaucoma. Intravitreal anti-vascular endothelial growth factor (VEGF) agents decrease the vascular permeability and inhibit the formation of abnormal new vessels. Anti-VEGF therapy involves continuous intravitreal injections in 1 to 3 month intervals with dosages of 1.25 mg/0.05 mL, 2.0 mg/0.08 mL of bevacizumab and/or of 0.5 mg/0.05 mL or 2.0 mg/0.05 mL of ranibizumab. They are useful to suppress radiation-induced neovascular glaucoma, radiation maculopathy, and optic neuropathy. Frequent intravitreal anti-VEGF injections resolved the retinal/macular edema, regressed the retina/choroidal neovascularization. Studies have reported a short term, temporary improvement in the vision and changes in the retina/macular architecture on optical coherence tomography with anti-VEGF agents [9]. Pars plana vitrectomy with silicon oil injection can form a shield to prevent injury to the normal ocular structures at the time of placement of plaque brachytherapy [10].

Radiation retinopathy continues to be leading cause of vision loss following radiation brachytherapy of choroidal melanoma. Timely intervention with frequent intravitreal injections prevents the progression of RR with preservation of the vision. Advanced and new radiation techniques in future might reduce the incidence of radiation retinopathy. Additionally studies are required on the safety, tolerability and efficacy of the anti-VEGF agents to prevent long-term complications of radiation of retinopathy.

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