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"INTRAVITREAL DEXAMETHASONE AN EFFECTIVE TREATMENT IN CASES OF RECURRENT MACULAR OEDEMA IN CRVO"

Ophthalmology

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ABSTRACT

Retinal vein occlusion is second most common retinal vascular disease after diabetic retinopathy. Macular oedema secondary to RVO is the second most common retinal vascular disease after diabetic retinopathy. Various treatment options have been mentioned in literature i.e., Anti VEGF injections, retinal laser, corticosteroids, vitrectomy, etc. We here with report a case of CRVO with recurrent macular edema even after 2 monthly doses of intravitreal ranibizumab injections who responded very well by single intravitreal injection of dexamethasone implant (Ozurdex). Till 3 months post injection of single dexamethasone implant, visual acuity was stable at 6/12 with maintaining of central foveal thickness below 250 microns without requiring of any other further mode of treatment. So we conclude that dexamethasone intravitreal implant is a very good effective mode of treatment in such cases of CRVO with recurrent macular edema.

KEYWORDS

CRVO, Dexamethasone implant, macular edema

INTRODUCTION

Retinal vein occlusion (RVO) is thought to result from a thrombotic event or vessel wall pathology[1] and significantly reduces vision.[2] The prevalence of RVO is estimated at 5.20 cases per 1000 people[3], and macular oedema secondary to RVO is the second most common retinal vascular disease after diabetic retinopathy.[3] Branch retinal vein occlusion (BRVO) and central retinal vein occlusion (CRVO) are the two major types of RVO and are named based on the location of the venous occlusion.[4]

Case Summary

A 59 years old female presented in eye outpatient department with the chief complaints of diminution of vision in right eye for 20 days. Patient was a known hypertensive and was on treatment for last 30 years. She also had history of angina pain 4 years back for which she took treatment that time.Systemic examination was within normal limits.On ocular examination, both eyes were pseudophakic and best corrected visual acuity in Right eye was finger counting 4 meters and in Left eye was 6/9. Pupillary reaction was normal in both eyes at time of presentation. Patient also complained of metamorphopsia. Intraocular pressure by applanation tonometry was normal in both eyes at the time of presentation.Fundus examination revealed multiple superficial haemorrhages with macular oedema in right eye while it was normal in left eye. On optical coherence tomography (OCT) cystoid macular oedema was found with neurosensory detachment (Figure 1). Therefore on above mentioned findings, diagnosis CRVO with macular oedema was made in right eye.

An injection of anti-VEGF (ranibizumab) was given intravitreally in right eye. After 2 week unaided visual acuity in right eye was 6/60 with significant reduction in Central macular thickness from 760 to 280. (Figure 2)

At 1 month follow up, there was recurrence of macular oedema on OCT although the visual acuity remained unchanged. Thus, 2nd dose of intravitreal injection ranibizumab was given(Figure 3) but macular oedema persisted.

In view of persistence of macular oedema, intravitreal dexamethasone implant (Ozurdex) was planned. After 1 week of steroid implant, vision improved to 6/12 and central macular thickness decreased to 275 microns. However, Intra Ocular Pressure (IOP) was raised which was controlled with antiglaucoma medicines. Antiglaucoma medicines were gradually withdrawn after 2 weeks.Gonioscopy was also done to rule out NVI and NVA.

On follow up at 1 month and 3 months, central macular thickness was found 240 micronson OCT and IOP was within normal range. Patient's BCVA remained stable without need of any further treatment. (Figure 4)

DISCUSSION

Retinal vein occlusion (RVO) was first described in 1855 by Liebreich and in 1878 by Michel[5], who indicated that RVO was a complication of systemic vascular status that can be observed on the fundus of the eye. Disease was named as Apoplexia retinae. This type of occlusion was later described as the central retinal vein occlusion (CRVO). Leber, in 1877, and Oeller, in 1896, described branch retinal vein occlusion (BRVO)[6,7] Retinal vein occlusion is second most common retinal vascular disorder after diabetic retinopathy. Studies in overall populations showed that its prevalence varies from 5.2 to 16 per 1,000[8].

RVO is more prevalent in men than women and it is more frequent in older people, over 65 years of age[8] and BRVO is four times more common than CRVO[8].In our case we have a 58 years old female with history of hypertension and ischaemic heart disease diagnosed with unilateral CRVO.

CRVO results from thrombosis of the central retinal vein when it passes through the lamina cribrosa.[9,10] It is classically characterised by disc oedema, increased dilatation and tortuosity of all retinal veins, widespread deep and superficial haemorrhages, cotton wool spots, retinal oedema and capillary non-perfusion in all four quadrants of the retina. In less severe forms the disc oedema may be absent. A previous CRVO may show evidence of optic disc and retinal collaterals, a telangiectatic capillary bed and persistent venous dilation and tortuosity, perivenous sheathing, arteriolar narrowing, and macular abnormalities (chronic macular oedema and retinal pigment epithelial changes).

Hypertension, diabetes, and open angle glaucoma are major known risk factors. Occasionally, patients with clotting disorders, dysproteinemias, and vasculitis can also develop CRVOs. Subsequent to the inciting event, approximately 16% of patients developed neovascularization of the iris and/or angle leading to neovascular glaucoma which is difficult to manage. The main risk factor in the development of neovascularization is the extent of ischemia.

Apart from all other routine investigations, visual fields, relative afferent pupillary defect (RAPD), color doppler imaging, electroretinography (ERG), visual acuity play an important role in differentiation of ischemic from non-ischemic central retinal vein occlusion[20].

Treatment

Corticosteroid therapyis given but the exact mechanism of action is unknown. It is thought that steroids work by targeting various inflammatory pathways and decreasing expression of VEGF, reducing vascular permeability, stabilizing endothelial tight junctions, and decreasing macular oedema.Currently, triamcinolone acetonide and dexamethasone are the two steroid preparations used to treat macular oedema associated with central retinal vein occlusion (CRVO).

Thepostulated mechanism for macular oedema is up-regulation of VEGF in response to retinal ischemia secondary to increased resistance to venous blood flow. The reported intravitreal levels of VEGF-A are higher in CRVO than in any other ischemic retinal vascular disease. VEGF increases vascular permeability, leading to macular oedema and development of neovascularization. Multiple anti-VEGF medications have also shown to be effective in reducing oedema.

Laser photocoagulation

CVOS evaluated the efficacy of prophylactic PRP in eyes with 10 or more disc areas of retinal capillary nonperfusion, confirmed by fluorescein angiography, in preventing development of 2 clock hours of iris neovascularization or any angle neovascularization or whether it is more appropriate to apply PRP only when iris neovascularization or any angle neovascularization occurs. Argon green laser usually is used. Laser parameters should be about 500-µm size, 0.1-0.2 second duration, and power should be sufficient to give medium white burns. Laser spots are applied around the posterior pole, extending anterior to equator. They should be about 1 burn apart and total 1200-2500 spots.

If ocular media is hazy for laser to be applied, cryoablation of the peripheral fundus is performed. About 16-32 transscleral cryo spots are applied from ora serrata posteriorly.CVOS evaluated the efficacy of macular grid photocoagulation in preserving or improving central visual acuity in eyes with macular edema due to central vein occlusion (CVO) and best-corrected visual acuity of 20/50 or poorer. Macular grid photocoagulation was effective in reducing angiographic evidence of macular edema, but it did not improve visual acuity in eyes with reduced vision due to macular edema from CVO.

Chorioretinal venous anastomosis

Chorioretinal venous anastomosis[11-15] can be performed by creating an anastomosis to bypass the site of venous occlusion in the optic disc.

Radial optic neurotomy

Radial optic neurotomy (RON) [16,17,18,19,20,21]is a new surgical technique in which a microvitreoretinal blade is used during pars plana vitrectomy to relax the scleral ring around the optic nerve. The central retinal artery and vein passes through the narrow openings of the cribriform plate in the optic disc.

Vitrectomy

A vitrectomy[22] is a technique in which the vitreous is surgically removed along with removal of the posterior hyaloid.

Some studies have shown that a vitrectomy may be beneficial for macular oedema due to CRVO. One theory is that a vitrectomy may relieve traction on the macula and, thus, reduce macular oedema. According to another hypothesis, removing the vitreous will also remove cytokines and VEGF associated with a venous occlusive event; thus, the stimulus for macular oedema will be reduced.

CONCLUSION

Hence, it is suggested that in CRVO cases,recurrent macular oedemaafter multiple intravitreal anti-VEGF injections,can be treated with intravitreal dexamethsone implant whichresults in significant reduction of macular oedema and improvement of BCVA.

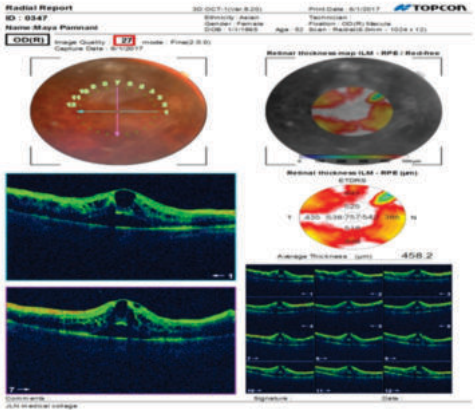

Figure 1:OCT showing cystoid macular oedema (CME) in CRVO

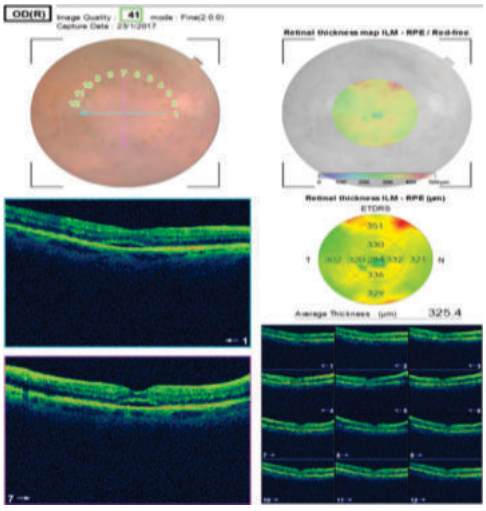

Figure 2: Resolution of CME after first intravitreal anti-VEGF injection at 2 weeks follow up

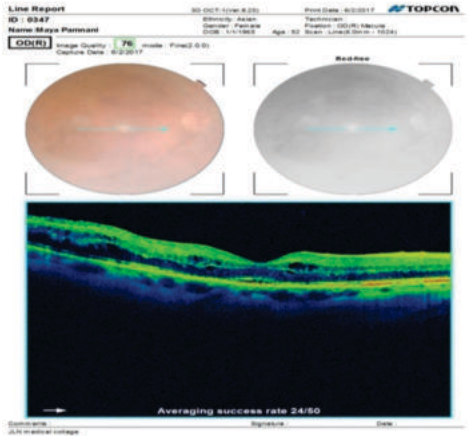

Figure 3: Recurrence of CME at 1 month follow up after intravitreal anti-VEGF injection

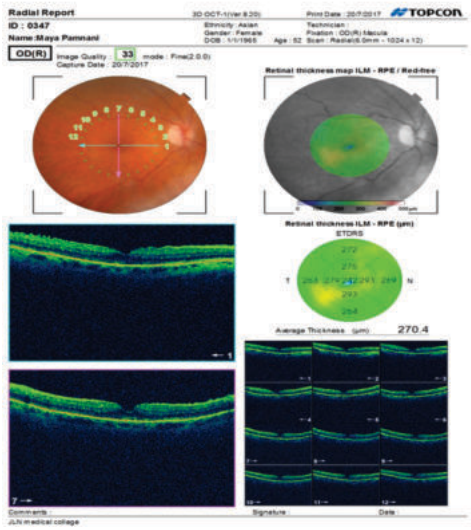

Figure 4: Resolution of CME after intravitreal steroid implant at 1 month

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