

Choroidal Neovascular Membrane (EYE) Treatment using Optical Fibre and LASER



Rudransh Pandey, Samadrita Bhattacharjee, Sanika Liya Sunil, Simran Nair, Jabeena A

Abstract: At the point when the eye experiences choroidal neovascularization, scar, or both, at the hour of treatment, the pace of repeat was 65% versus 33% if the concerned eye had no choroidal neovascularization, scar, or both. Normally a visual sharpness of four lines is lost when individual eyes had choroidal neovascularization, scar, or both, versus one line when the individual eye had no choroidal neovascularization or mark. Photocoagulation of choroidal neovascularization using LASER in patients experiencing angioid streaks can cause the ending of the choroidal neovascularization and adjustment of visual keenness. Treatment of choroidal neovascular membranes (CNVM) can differ depending on the concerned disease. Treatment consists of thermal laser treatment, anti-VEGF drugs, photodynamic therapy (PDT). Contingent upon the advancement of the illness, the individual may get at least one of these therapeutic treatment. Laser treatment is generally done as an outpatient strategy. It happens in the eye specialist's office or at the hospital. The LASER beam utilized in this treatment is a focused light having high energy.[5] It obliterates the abnormal blood veins, anticipating further spillage, dying, development. After the laser treatment, vision may turn out to be more obscured than previously. Be that as it may, frequently it will balance out inside half a month. The treatment brings about a perpetual scar, making a lasting vulnerable side. This came about vulnerable sides, may be perceptible in the field of vision. Generally in the LASER treatment anomalous veins are demolished. In any case, patients regularly need re-treatment inside three to five years.

Keywords: Choroidal Neovascularization Membrane, Angioid Streaks, Visual Acuity, Proangiogenic Mediators, Pathogenesis.

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I. INTRODUCTION

Choroidal neovascularization (CNV) optional to horrendous choroidal breakage is an uncommon condition that is commonly identified with aberrant choroidal break pursued by nonpenetrating closed globe obtuse injury. Break of Bruch's film, the fiery procedure, and the subsequent irregular creation of proangiogenic arbiter clarifies it's pathogenesis.[1] Reportedly it shows up between one month and four years after visual injury.

Notwithstanding, not many papers record the result of direct harm to the choroid following intraocular remote body (IOFB) injury in open globe damage, and the most incessant inconvenience in these cases are the improvement of proliferative vitreoretinopathy prompting complex retinal separation. The motivation behind this paper is to portray an unordinary instance of post-horrible CNV

auxiliary to coordinate choroidal burst as the aftereffect of a metallic IOFB, with accentuation on the counter vascular endothelial development factor (VEGF) dosing routine.

Choroidal neovascularization (CNV) is one of the prominent causes of focal visual loss in all age groups.[1] The visual loss is especially uproarious in instances of subfoveal neovascularization. The pervasiveness of visual impairment is lower in kids than adults. It has been seen that visual deficiency is progressively prevalent in kids due to much higher disability-balanced life years. Besides, a blind kid faces extensive difficulties in educational and passionate advancement.

Studies revealed that advantage of laser photocoagulation to choroidal neovascularization optional to angioid streaks is as yet questionable. Review of records of about 20 patients and 24 eyes with prominent choroidal neovascularization secondary to angioid streaks who received treatment between 1982 and 1991 in eminent Institute revealed that the choroidal neovascularization was extrafoveal in 18, juxtafoveal in around two and three had subfoveal followed by one having indeterminate.[6] As a result lines of visual acuity change was average about; -2.5 to 3, -2.6 at 6 followed by -2.4 at 12 and -4.8 lines at 24 months.

There are some conspicuous contrasts in CNV among youngsters and grown-ups: first, the uncommonness of macular degeneration and nearsighted fundus changes (the two most normal reasons for grown-up beginning CNV) at a youthful age; second, lack of calcification and thickening of Bruch's membrane (which is otherwise frequently observed among adults) and third, presence of single subretinal indevelopment destinations,

not at all like grown-up cases in which different indevelopment locales are common.[2] All these components may make the regular course, forecast, and treatment results progressively great among young people. Fig.1 shows an AMD affected eye of 64-year-old female, treatment of intravitreal injection of an anti-VEGF agent showed full thickness macular hole related to contraction of CNV and rapid resolution of macular edema. Treatment performing vitrectomy with CNV removal improved visual acuity and macular hole found closed.

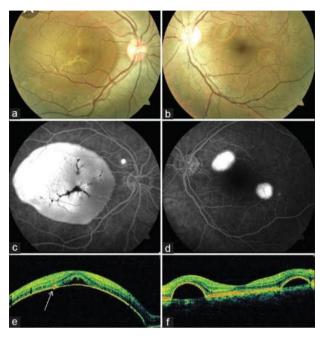


Fig. 1: Expulsion of choroidal neovascular film for a situation of macular gap after enemy of VEGF treatment for age-related macular degeneration

Courtesy; https://images.app.goo.gl/ZgjjRz8JcojFc7cYA

II. CLINICAL PRESENTATION

Blurred distance and distorted near vision, are the most frequent symptoms. Patients may likewise complain of metamorphopsia or a scotoma. Be that as it may, ordinarily they volunteer no side effects or offer just dubious visual protests. Detailed examination of the macula with stereoscopic slit lamp biomicroscope using either 90 or 78 D lens or a corneal contact lens is essential to evaluate the subtle clinical finding of CNVM. It appears as a dirty graygreen color lesion, deep to the retina. The gray-green appearance is believed to be due to the hyperplastic response of RPE. It may be accompanied by subretinal hemorrhage and/or lipid, serous or hemorrhagic PED, neurosensory exudate detachment, macular edema, and cystoid macular changes.

III. PROPOSED METHODOLOGY

a. Retinal Pigment Epithelial Detachment (Rpe)

Serious Pigment Epithelial Detachment PED appears as orange-yellow round, the oval or bean-formed rise of the RPE with smooth, raised surface contours. Some resorb or flatten spontaneously leading to RPE disturbances including geographic atrophy. PED in younger patients generally has a benign course. However, one third to one half of the serous PED in patients >50 years of age will develop CNVM with severe visual loss. Certain characteristics of PED are associated with increased risk of developing CNVM. They are old age (>65 years), associated sensory detachment, larger PED size (>1-disc diameter) and notching. Blood and lipids within or surrounding PED imply the presence of CNVM. Similarly, chorio-retinal folds under PED also indicate the presence of CNVM.

RPE tears are generally described as a complication related with PED, with or without laser photocoagulation. Tears are usually located at the intersection of the joined and disengaged RPE; it happens when the liquid powers from the hidden CNVM outstretch the sub-RPE space. As the RPE tears, the free edge of the RPE is moved back and moves toward the fibrovascular tissue.

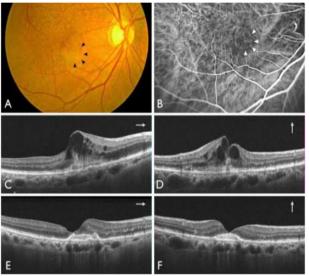


Fig 2: Recurrence of CNVM with different types of surface of ocular surfaces.

Courtesy; https://images.app.goo.gl/5S2eh6kE1nAd1zNo7

b. Laser treatment

Once extrafoveal, well defined CNVM is diagnosed, prompt treatment should be given. The goal of treatment is to close the neovascular lesion and prevent severe visual loss rather than to restore normal visual acuity. Before treatment, a detailed discussion about the disease, treatment goal, side effects such as permanent scotoma, corresponding to treatment and long-term follow up with repeat angiography should be emphasized.

Laser treatment is also used to treat histoplasmosis usually used as an outpatient procedure.

As we can see in Fig 2, patients with symptomatic peripapillary choroidal neovascularization (CNVM) who got beginning bevacizumab treatment pursued by warm laser and bevacizumab blend treatment.





Intravitreal bevacizumab infusions were directed to patients until the sores were well-characterized. Warm laser removal was then regulated and an extra bevacizumab infusion was allowed following 1 week. [4]

The thermal laser specifically (532-nanometer diode laser) was applied to the CNVM, and 200 microns past the worldly side of the sore until retinal brightening happened. The optic nerve was not involved in the treatment. Treatment was administered according to the Macular Photocoagulation Study (MPS) guidelines. Within one week of laser treatment, additional bevacizumab was injected. Patients were then evaluated for treatment success and safety by OCT at one month and deliberately checked for any indications of repeat at expanding interims of about a month and a half, two months, ten weeks, and afterward at regular intervals, for at least 15 months. Pre- and postoperative values for visual acuity and Central Macular Thickness CMT were compared using the Wilcoxon rank-sum test. Analyzing statistics p-value of less than 0.05 was considered significant.

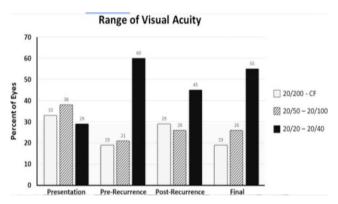


Fig. 3: Statistical representation of Age related Macular Degeneration ARMD with different causative factors.

Courtesy; https://images.app.goo.gl/j2H9fR3zb5VsmWCA8

IV. RESULT ANALYSIS

Research have shown that median visual outcomes have improved from 20/50 to 20/30 (p=0.0232). The mean visual improvement observed was three lines.[4] Despite proper laser treatment, the recurrence of CNVM does occur. By the end of five years, recurrence of CNVM has been observed in 54% of laser-treated eyes, and 80% of recurrence occurred within one year of treatment. Recurrence of CNVM was responsible for most of the visual deterioration seen in the treatment group.[3] The average visual acuity in eyes without recurrence was 6/12 at one year and 6/15 at three years following the treatment. Interestingly, the average visual acuity in eyes with recurrences was 6/37.5 at one year and 6/75 at three years following treatment.

V. RISK FACTORS

Age related Macular Degeneration ARMD is a multifactorial disorder with various causative elements harming the macula and results in a typical appearance that we perceive clinically as ARMD, as should be obvious in , Fig 3. Hazard elements ensnared in clinical and research center investigations incorporate drusen - photic injury,

antioxidant and vitamin/mineral deficiency.[3] The neovascular type of ARMD characterized by CNVM occurs in older patients who are more likely to show evidence of non-specific cardiovascular problems. Hypertension and smoking are associated with the development of neovascular ARMD as well as with recurrence of CNVM following laser photocoagulation. Laser treatment cannot fix histoplasmosis. It diminishes the spreading of blood vessels which will create more problems to the individual's vision.[5] If after the treatment blood vessels returns, more laser surgery will be needed.

VI. CONCLUSION

People with loss of vision or poor eye sight was related to macular damage/rupture or baseline VA. Choroidal neovascularization is related with choroidal rupture, macular degeneration due to age, photocoagulation scars etc. The scar/spot or atrophy after the surgery may also cause reversal visual disease.

Choroidal rupture may be direct, indirect or at the particular spot though indirect is most common. They can be isolated but cause multiple ruptures.

In this paper, we analyzed the possible and convenient treatments and the risk factors associated with the comeback of CNV in the patients.

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