# CLINICAL FEATURES, FUNDUS FLUORESCEIN ANGIOGRAPHY FINDINGS, OPTICAL COHERENCE TOMOGRAPHY FINDINGS AND VISUAL OUTCOME AFTER TREATMENT WITH INTRAVITREAL BEVACIZUMAB IN PARAFOVEAL TELANGIECTASIA

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This is to certify that the dissertation **"CLINICAL FEATURES, FUNDUS FLUORESCEIN ANGIOGRAPHY FINDINGS, OPTICAL COHERENCE TOMOGRAPHY FINDINGS AND VISUAL OUTCOME AFTER TREATMENT WITH INTRAVITREAL BEVACIZUMAB IN PARAFOVEAL TELANGIECTASIS"** entitled is a bonafide work done by **Dr. K. PIOUS**, Postgraduate student in M.S. (Ophthalmology) during APRIL 2010 to MARCH 2012, under our direct supervision and guidance, at our institute, in partial fulfilment for the award of M.S. Degree in Ophthalmology of the Tamilnadu Dr. M.G.R. Medical University, Chennai.

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# CONTENTS

## ABSTRACT

**AIM:** To evaluate the clinical features, fundus fluorescein angiographic (FFA), optical coherence tomographic (OCT) findings and to study the effect of intravitreal bevacizumab in type 2 parafoveal telangiectasia (PFT).

**PATIENTS AND METHODS:** In this prospective, interventional case series, patients with clinical diagnosis of PFT were divided into 3 groups: eyes with PFT alone (group 1), eyes with PFT with cystoid macular edema (CME) (group 2) and eyes with PFT with Choroidal neovascular membrane (CNVM) (group 3). Eyes with PFT with CME and CNVM received intravitreal bevacizumab (2.5mg/0.1ml). Main outcome measures studied were reduction in macular thickness and improvement in visual acuity.

**RESULTS:** Eighty eyes of forty patients (14 males and 26 females), ranging in age from 40 to 70 were included in the study. Eyes with PFT alone showed no change in visual acuity or macular thickness during follow up. Eyes with PFT with CME showed improvement in visual acuity ($p<0.001$) and in macular thickness ($p<0.001$). Eyes with PFT with CNVM showed improvement in macular thickness after bevacizumab ($p<0.001$) but not in visual acuity.

**CONCLUSION:** Intravitreal bevacizumab is effective in management of eyes with PFT with CME. In eyes with PFT with CNV, there may be not be improvement in vision after bevacizumab despite reduction in macular thickness.

Key words: Bevacizumab, Cystoid macular edema, Choroidal neovascular membrane, Parafoveal telengectasia

# INTRODUCTION

The term "retinal telangiectasis" was first used by Reese to describe the developmental vascular anomalies seen in patients with Coats' disease and Leber's miliary aneurysms.[1] The telangiectatic vessels are characterized by localised, congenital abnormal segments of blood vessels that result in leakage. The telangiectasis may be localised only to the capillaries of the parafoveal region, known as Parafoveal Telangiectasis, and is characterized by the microaneurysmal and saccular dilation and capillary nonperfusion of the parafoveal capillaries. The more generalised form is known as Coats' disease and is characterized by the telangiectatic retinal vessels that cause massive subretinal and intraretinal exudation.

Parafoveal telangiectasis was first described by Gass who observed that the parafoveal retinal capillaries only showed telangiectasia; fluorescein angiography was required to diagnose this cases.[2] since the capillaries affected are confined to the juxtafoveal region. Telangiectasis of parafoveal capillaries could either be idiopathic or secondary to a number of retinal vascular disorders or systemic diseases. The idiopathic form further could be either (a) developmental which is usually seen unilaterally in males (spectrum of Coats' disease); or (b) acquired which tends to present bilaterally without any gender predilection. These are further subdivided according to the revised classification of Gass and Blodi.[3] Type 2 parafoveal telangiectasia is the form most commonly encountered.

Parafoveal Telangiectasis is classified [3] as:

- Group1A - Unilateral congenital juxtafoveal telangiectasis;
- Group1B - Unilateral idiopathic juxtafoveal telangiectasis;
- Group 2A - Adult, bilateral, idiopathic, acquired juxtafoveal telangiectasis;
- Group 2B - Juvenile ,occult, familial, idiopathic, juxtafoveal telangiectasis;
- Group 3A – Occlusive, idiopathic, juxtafoveal retinal telangiectasis; and
- Group 3B – Occlusive, idiopathic, juxtafoveal retinal telangiectasis associated with central nervous system vasculopathy.

Patients typically present in the fifth and sixth decades of life with mild blurring of vision in one or both eyes which may progress, primarily from foveolar atrophy or subretinal neovascularization.[4] Biomicroscopy reveals symmetric blunting of the foveal reflex with a mild grayish appearance of the parafoveal retina, minimal serous exudation, and no lipid deposition.[4,5] In addition, glistening white or yellow-white crystalline deposits may be noted in the superficial parafoveal retina in approximately 40% of patients.[6] In some patients, a small, yellow lesion, 1/3 disk diameter in size, develops within the foveal avascular zone and appears to represent an inner retinal cavitation on evaluation by optical coherence tomography (OCT). [4,5]

**Histopathology**

The histopathological features of IJFT IIA have been described. In a landmark histopathological report, Green *et al*.[7] did not find telangiectasis of the retinal vessels but, instead, thickening of retinal capillaries from marked proliferation of the basement membrane in a multilayered configuration, and narrowing of the caliber of the lumen. Degeneration of pericytes, and occasionally endothelial cells, was also described, not only in the affected juxtafoveal area, but also to a lesser degree throughout the retina.

Cellular debris from degenerated endothelial cells and pericytes, and multimembranous lamellar lipid were entrapped between layers of capillary basement membrane. Localized endothelial defects in the temporal parafoveal area were seen, and the authors postulated that these were the sites at which fluorescein diffused into walls and became trapped by the thickened capillary wall. Intracellular and extracellular edema, especially prominent in the inner retinal layers was observed. Retinal capillary proliferation into the outer retina as far as the photoreceptor layer was also noted.[7]

Another histopathological report of a more advanced case with SRNV corroborated these findings and demonstrated in addition (1) dilation and proliferation of retinal capillaries into the outer retinal layers and into the subretinal space with retinochoroidal vascular anastomosis and (2) intraretinal migration of the RPE along the course of the telangiectatic vessels.[8]

All these histological features corroborate the clinical findings described by Gass *et al*., and although they suggest that the juxtafoveal retinal capillaries are the primary tissues involved in this condition, [10,11,12] they also support what Gass has postulated, i.e., that capillary dilation and "telangiectasis" does not occur until later in the process.[9]

**Pathophysiology**

The precise pathogenesis of IJFT IIA is controversial. In their earlier report and based on the angiographic findings, Gass and associates initially proposed a primary role of the retinal capillaries.[13] The altered capillary wall associated with metabolic alterations and increased endothelial permeability, incite chronic nutritional damage to the retina, particularly the Müller cells.[14] Further changes in the outer capillary bed

would cause an alteration in the pattern of venous outflow and the formation of right-angled venules. The nutritional deprivation of the middle retinal layers would lead to degeneration and atrophy of the outer retina including the photoreceptors which would cause the gradual visual loss and permit subretinal and intraretinal pigment epithelial migration and proliferation to form black plaques in the vicinity of the right-angled venules. Photoreceptor atrophy would also permit the proliferating capillaries to invade the subretinal space causing SRNV and the latter may develop anastomosis with the choroidal vessels.[10,11,15] In later years however, Gass commented that IJFT IIA "is not primarily a leaky retinal blood vessel disease," but rather "the primary abnormality may reside in one or both of the parafoveolar retinal neural or Müller cells" since he observed that telangiectatic capillary dilation does not occur until later in the process, and the loss of central vision is due to photoreceptors atrophy in the absence of macular edema.[9,14]

Although, the clinical pathologic report by Green *et al*.[12] showed intracellular and intercellular edema primarily in the inner retinal layers as well as capillary endothelial cell degeneration and regeneration, the unusual capillary endothelial abnormalities observed could, in fact, be secondary to Müller cell dysfunction. Müller cells in the retina, like astrocytes in the brain, are critical to proper function of the retinal capillary endothelium and the health of the surrounding neurons.[16–19] A primary degeneration or dysfunction of the parafoveolar Müller cells, could lead in this scenario to (1) retinal endothelial cell degeneration as suggested by Gass and Yannuzzi,[14,20] or accelerated rate of cell death and replacement as suggested by Green[12] as a possible triggering mechanism for proliferation of retinal capillaries; and (2) retinal thinning, accompanied later by a breakdown of the blood–retinal barrier at the parafoveolar retinal capillaries,

causing a superimposed limited edema. The precise mechanism for the retinal capillary proliferation, however, remains unknown.

Superficial crystalline deposits are a common finding in IJFT IIA and are thought to represent the remnants of degenerated Müller cells because of their location near the internal limiting membrane (ILM).[15] The process of subretinal proliferation is similar to the neovascular form of age-related macular degeneration known as retinal angiomatous proliferation (RAP).[21] Both IJFT IIA and RAP can be associated with retino-retinal anastomoses that may extend beneath the retina when SRNV occurs [22,23] and plaques of subretinal pigmentation.[24] The latter are hypothesized to represent reactive hyperplasia of the RPE induced by extension of the retinal vascular proliferation posteriorly toward it.[20]

Initially, telangiectasis is not visible on biomicroscopy, and fluorescein angiography shows only mild staining within one disk diameter of the foveola, especially temporally. With progression, these eyes may develop dilated right-angled retinal venules and arterioles, and stellate plaques of retinal pigment hyperplasia, as well as intra-and subretinal anastomosis and subretinal neovascularisation occur.[25,26,27] Loss of central visual acuity may be rapid, and 81% of untreated eyes have a final acuity of 20/200 or worse.

The grid laser is not indicated for treatment of type 2 parafoveal telangiectasis because it does not improve or stabilize long-term visual acuity[28]. In addition, use of laser may lead to the development of subretinal hemorrhage [29] and choroidal neovascularization.[28] A variety of therapeutic modalities have been employed to treat subretinal neovascularization complicating this disorder. Photo-dynamic therapy[30,31]

and transpupillary thermotherapy[32,33] have demonstrated efficacy; however, the subretinal neovascularization in idiopathic juxtafoveal retinal telangiectasis arises from the retina, as opposed to the choroid, and activation of photosensitizing dye in the retinal layers may cause iatrogenic damage.[30,33] Laser photocoagulation may help stabilize central visual acuity but induces an iatrogenic scotoma,[34] submacular surgery has demonstrated poor outcomes. [35] Anti-VEGF drugs hold promise for treating neovascularization associated with CNVM. The role of bevacizumab in the treatment of parafoveal telengectasia without CNVM is less clear.

# AIM OF THE STUDY

a) To document the clinical features, fundus fluorescein angiographic (FFA) and optical coherence tomographic (OCT) findings in patients presenting with type 2 parafoveal telangiectasia at a tertiary eye care facility.

b) To evaluate the effect of intravitreal bevacizumab in treatment of type 2 parafoveal telangiectasia.

# REVIEW OF LITERATURE

Matsumoto et al. [36] investigated the efficacy of intravitreal bevacizumab for the treatment of idiopathic macular telangiectasia (IMT). Ten eyes of eight consecutive patients with IMT were studied. All patients were treated with intravitreal bevacizumab (1.25 mg) injections at baseline. There were no changes in the mean visual acuity in either eye in any of the patients. These authors concluded that Type 2 IMT improved anatomically with bevacizumab treatment, despite a lack of improvement in vision, and bevacizumab effectively decreased vascular permeability and retinal edema in the short term.

Gamulescu et al.[37] described functional and morphological long-term follow-up results in patients with IMT treated with intravitreal bevacizumab. This was a retrospective case series of three consecutive male patients with IMT who were treated with intravitreal bevacizumab injections. A single intravitreal bevacizumab injection resulted in a marked improvement in best corrected visual acuity (BCVA) from 20/50 to 20/20 than the patient with type 1 (aneurysmal) IMT during the first 4 weeks. These authors concluded that patients with type 1 IMT with pronounced macular oedema on OCT may benefit from intravitreal bevacizumab injections, showing functional as well as morphological improvement, while patients with type 2 IMT with minimal cystic changes on OCT do not show functional improvement despite repeated injections.

Issa et al.[38] reported on the short-term effects of intravitreal bevacizumab in patients with type 2 IMT in a non-comparative, interventional, retrospective case series. Seven eyes of six patients with type 2 IMT were evaluated. Patients received 2 doses of intravitreal bevacizumab (1.5 mg) at a 4-week interval. A significant increase in mean

VA of 8 ETDRS letters at 8 weeks was found. Visual acuity improved by more than 15 letters in one patient and by 10 to 15 letters in two patients and remained stable (to +10 letters) in another four patients, compared with baseline. All patients showed a reduction in extension and intensity of late-stage parafoveal hyperfluorescence on fluorescein angiography. In OCT imaging, mean retinal thickness showed a significant reduction in the foveal and in the parafoveal zones. The most pronounced effect (mean decrease, 22Î¼m) was in the parafoveal temporal zone. No significant ocular or systemic side-effects were observed. These authors concluded, based on their short-term results, that inhibition of vascular endothelial growth factor (VEGF) by intravitreal bevacizumab is associated with a decrease in retinal thickness and a reduction in angiographic leakage in type 2 IMT; they also opined that intravitreal bevacizumab may improve VA in affected patients.

Kovach et al.[39] sought to determine whether inhibition of VEGF-A affects visual acuity, fluorescein angiographic, and OCT outcomes in patients with perifoveal telangiectasia (PT)/ (synonyms are macular telangiectasia, Type 2 juxtafoveolar retinal telangiectasis group 2A). In this retrospective review of patients with PT treated with intravitreal bevacizumab at the Bascom Palmer Eye Institute, in Florida (USA), best-corrected visual acuity, fluorescein angiography, and OCT measurements were performed on nine eyes of eight patients. Five eyes had proliferative PT, characterized by subretinal neovascularization involving the macula. After treatment (follow-up ranged from 4 to 27 months) the mean BCVA remained stable for the four eyes with nonproliferative PT, whereas in the five eyes with proliferative PT, BCVA was unchanged or improved after treatment. All eyes demonstrated decreased intraretinal leakage on fluorescein angiography after an injection of bevacizumab, and eyes with proliferative PT showed decreased growth and leakage of the subretinal

neovascularization. The mean decrease in OCT central retinal thickness was less than 30 μ. These investigators were of the opinion that in nonproliferative PT, intravitreal bevacizumab decreases fluorescein angiographic leakage without any short-term effect on visual acuity or OCT appearance whereas in proliferative PT, intravitreal bevacizumab arrests the leakage and growth of subretinal neovascularization with the possibility of visual acuity improvement.

Moon et al.[40] assessed the potential visual benefit of intravitreal bevacizumab in a patient with idiopathic juxtafoveal retinal telangiectasis refractory to focal laser treatment; an intravitreal injection of bevacizumab (1.25 mg) was given. Within 1 week, visual acuity improved from 20/50 to 20/25 and OCT demonstrated complete resolution of macular edema without any adverse effect due to the injection. The macular edema recurred after 3 months, requiring a repeat injection of bevacizumab, with subsequent resolution of macular edema. These authors believed that an intravitreal injection of bevacizumab may provide potential short-term visual benefit in patients with macular edema from idiopathic juxtafoveal retinal telangiectasis.

Idiopathic perifoveal telangiectasia (IPT) is a disorder characterized by focal parafoveal capillary dilatation of unknown cause. Photodynamic therapy (PDT) is reported to be efficacious in the treatment of subretinal neovascularization (SRN) associated with idiopathic juxtafoveal retinal telangiectasis; however, visual improvement is limited. Cakir et al.[41] described the clinical, angiographic, and OCT findings for a patient with IPT complicated by SRN who underwent treatment with PDT and bevacizumab. The patient underwent two sessions of PDT; however, metamorphopsia persisted, and visual acuity further decreased to 20/100 after the second PDT. Hence, after PDT, two injections of intravitreal bevacizumab (1.25 mg)

were administered, following which visual acuity increased dramatically from 20/100 to 20/25. Four months after the second injection, there was no recurrence, and the patient was asymptomatic. These authors concluded that intravitreal bevacizumab treatment should be considered for patients with SRN associated with perifoveal telangiectasis, especially after insufficient therapeutic response to PDT.

Mandal et al.[42], in a nonrandomised interventional case series, evaluated the efficacy and safety of intravitreal bevacizumab in the treatment of SRN secondary to type 2A idiopathic juxtafoveal telangiectasia (IJT). Intravitreal bevacizumab (1.25 mg/0.05 ml) was injected as primary treatment into six eyes of six patients with SRN due to IJT. OCT and fundus fluorescein angiography (FFA) findings were examined before and after treatment (the patients were followed up for 3-6 months). Pre-injection BCVA measured from 20/400 to 20/120 (mean 20/200). After a mean follow-up of 4.2 months, post-injection BCVA measured from 20/200 to 20/50 (mean 20/100). At the last visit, BCVA was found to have improved by two or more lines in five eyes (83%) and remained the same in one eye (17%). The mean central foveal thickness improved from 263 μ (range, 165 to 393 μ) to 201 μ (range, 126 to 351 μ), representing an average reduction of 62. Only one eye received more than one two bevacizumab injections and no significant complications were observed. These authors concluded that in their small series, intravitreal bevacizumab appeared to be a safe and effective treatment for SRN secondary to type 2A IJT.

Charbel et al.[43] evaluated retrospectively the effects of intravitreal bevacizumab for non-proliferative type 2 idiopathic macular telangiectasia (type 2 IMT) for a mean follow-up of 18 months. Six eyes of five patients with type 2 IMT received two doses of intravitreal bevacizumab (1.5mg) at a 4 week interval. All patients underwent visual

acuity testing, slit lamp biomicroscopy, fluorescein angiography, and retinal thickness analysis by optical coherence tomography and fundus-controlled micro-perimetry. The mean VA increased significantly, Parafoveal leakage in FFA and mean central retinal thickness was decreased in all eyes following treatment. A rebound effect was observed after 3-4 months, and at the last visit, retinal thickness was increased in selected retinal sectors including the fellow eye. Inhibition of vascular endothelial growth factor (VEGF) by intravitreal bevacizumab may lead to functional improvement as well as transient decrease in leakage and retinal thickness in patients with type 2 IMT.

Initially, telangiectasis is not visible on biomicroscopy, and FFA shows only mild staining within one disk diameter of the foveola, especially temporally. With progression, these eyes may develop dilated right-angled retinal venules and arterioles and stellate plaques of retinal pigment epithelial hyperplasia, as well as intra- and subretinal anastomosis and subretinal neovascularization. The typical FFA features include dilated, ectatic parafoveal capillaries; these incompetent vessels display hyperfluorescence, hyperplasia of the retinal pigment epithelium (RPE) routinely block fluorescence.

Optical coherence tomography (OCT) is a new diagnostic imaging technology. The basic principle of OCT is that low coherence light is directed onto a beam splitter, resulting in 2 beams, one directed at the tissue of interest ( signal beam ) and the other at a reference mirror ( reference beam ). The amplitude and delay of tissue reflection is determined by an interferometer that adds the electromagnetic waves in the 2 reflected light beams. Because of the low coherence of the light source, interference of light reflected from the signal and reference beams only occurs when the delay of the reflections is almost

matched, resulting in high resolution. Axial resolution of the OCT system is determined by the coherence length of the source. The super luminescent diode source has a bandwidth of 30nm, with a full-width half maximum ranging resolution of 14 microns in air. After adjusting for the index of refraction of the eye, the full-width half-maximum ranging resolution is 10 microns. The lateral image resolution is a function of the beam waist diameter in the sample could be made as small as a few tens of micrometers. This high resolution permits tomographic imaging of tissue microstructure

The OCT software provides a variety of retinal scanning protocols including linear, circular, radial and parallel line scans. The duration can be shortened by performing three fast scans, and then averaging them to give the final interpretation. The scan can be adjusted manually or automatically with the position of the signal. The signal strength can be optimised automatically by pressing z-axis offset and polarisation 'buttons'. The images are continuously displayed on the monitor, and can be freezed whenever necessary. All the frozen scan images (upto 8) can be reviewed and image of the best quality can be saved. If none of the scans is good, the scanning process can be repeated.

Following the scanning phase, the appropriate scan analysis protocol can be used. There are different scan analysis protocols which allow quantitative analysis of the scan. The scan image can be qualitatively analysed. For qualitative analysis, individual scan images can be studied and interpreted in detail. The alignment algorithm reduces artefacts caused by axial movement of the eye during scan. The normal algorithm allows comparison of scans with varying signals. The OCT machine has Gaussian smoothing, proportional elevation and profile algorithm scan for

refinement of the scan image. Applying of grey scale image allows detection of various minute differences in the contrast sensitivity. A database is also available in the latest software for comparison of peripapillary retinal nerve fibre layer and macular thickness. Each eye of each subject was dilated with 1% tropicamide and 2-5% phenylephrine hydrochloride before recording the image. The patient was seated comfortably in front of the OCT machine with chin positioned on the chin rest, and is asked to fixate on the fixation target. The internal fixation (green colour light) target was the commonly used fixation target. After fixation, the operator selected the desired scan and aligned the instrument so that the fundus image and scan beam were displayed on the screen. In patients with significant refractive errors, rotation of the dioptre compensation device was found to improve image quality. Ranging measurements were performed using a fibro-optically integrated Michelson interferometer with a short-coherence length super luminescent diode source. The system used for this study used new data analysis software to quantitate nerve fibre layer (NFL) and retinal thickness. Retinal thickness was quantitated by the computer for each scan in the image as the distance between the first reflection at the vitreoretinal interface and the anterior boundary of the red, reflective layer corresponding to the retinal pigment epithelium and choriocapillaries.

Specific scanning protocols in OCT include retinal scanning and macular scanning protocols. Retinal scanning protocols include line scans, radial lines, raster lines, cross hair, X-line and circle.

| SCAN | TECHNICAL SPECIFICATIONS | UTILITY |
|---|---|---|
| Line | Default length:5mm<br>Default angle:0◦<br>Nasal position is zero position.<br>Defaults can be altered as needed. | Multiple line scans can be obtained by changing default angle/length without returning to main window. |
| Radial lines | Default number:6 lines. (30◦ separation)<br>Default length of each line:6mm.(this can be altered)<br>Defaults can be altered as needed (± 24lines) | For determining entire macular thickness/volume. |
| Raster lines | Multiple equidistant parallel lines are used to scan a square area.<br>Default setting scans a 3mm square area using six lines.<br>Lines scan from superior to inferior and nasal to temporal.<br>Defaults can be altered as needed (± 24lines) | By altering defaults an entire area of pathology can be scanned. |
| Repeat scan | Fixes defaults (size, angle, fixation LED and landmark) automatically to that used in an earlier selected scan. | Monitoring change during follow up visits. |
| Macular thickness | Default number:6 lines.(30◦ separation)<br>Default length of each line:6mm.(this cannot be altered)<br>Defaults can be altered as needed (± 24lines) | For determining entire macular thickness/volume. |
| Fast macular thickness | Obtains six scans of 6mm length rapidly (within ± 2seconds)<br>Defaults cannot be altered. | Allows comparative thickness/volume analysis. |

**Macular scanning protocols** include macular thickness scan, fast macular thickness, raster lines, and single line scan. Macular thickness map scan and fast macular thickness map are the two commonly used protocols. These protocols consist of radial scan length of 6mm at equally spaced angular (30◦) orientation.

Measurement of retinal thickness at selected points on the tomographs is obtained automatically by means of a computer algorithm, which assumes that the first highly reflective band corresponds to the vitreoretinal interface and the second corresponds to the retinal pigment epithelium. Thus, evaluating the displacement between anterior surfaces of these two interfaces determines retinal thickness.

Optical coherence tomography can demonstrate hyporeflective intraretinal cavitation without foveolar thickening and middle or inner layer hyperreflectivity corresponding to areas of retinal pigment epithelial migration.

# PATIENTS AND METHODS

This prospective interventional study was done at Retina Clinic, Institute of Ophthalmology, Joseph Eye Hospital, Tiruchirapalli from April 2010 to August 2011.

## INCLUSION CRITERIA

Inclusion criteria included the following:

- Patients with clinical diagnosis of PFT Type 2
- Media clarity adequate to perform FFA and OCT
- Follow up of at least 3 months

## EXCLUSION CRITERIA

Exclusion criteria included the following:

- Patients with other retinal pathology affecting visual acuity
- Patients with hazy media where FFA and OCT could not be performed
- Patients who did not complete follow up for three months

## PROCEDURE

All patients underwent a complete ophthalmic examination which included best corrected visual acuity (BCVA), intraocular pressure (IOP), slit lamp examination, fundus examination, fundus photography, fundus fluorescein angiography(FFA), optical coherence tomography (OCT) (fast macular scan using Zeiss Stratus 3 OCT).

Eighty eyes of forty patients (14 males and 26 females), ranging in age from 40 to 70 were included in the study. All patients were first informed of the procedure, and

the possible complications and informed written consent was obtained. The Institutional Ethics Committee (Institutional Review Board) approved the study.

Patients included in the study were divided into three groups for follow-up and treatment.

- Group 1: eyes with PFT treated conservatively
- Group 2: eyes with PFT and CME receiving intravitreal bevacizumab
- Group 3: eyes with PFT and CNVM receiving intravitreal bevacizumab

All patients were followed up at 1 week, 1 month and 3 months after initial examination or intervention.

The parameters checked after intervention included:

1. Measurement of BCVA
2. Slit-lamp examination
3. Fundus examination and photography
4. Measurement of IOP
5. OCT.

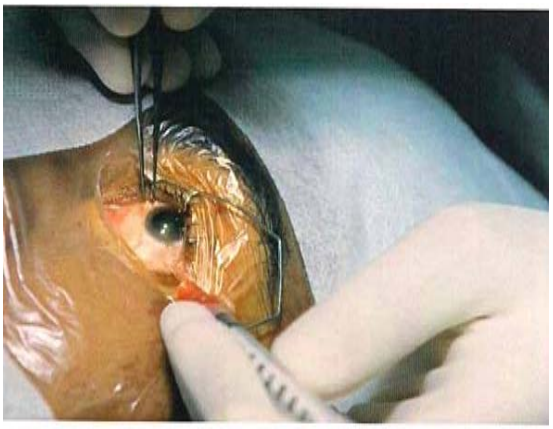
Main outcome measures studied were changes in clinical picture, OCT picture and visual acuity.

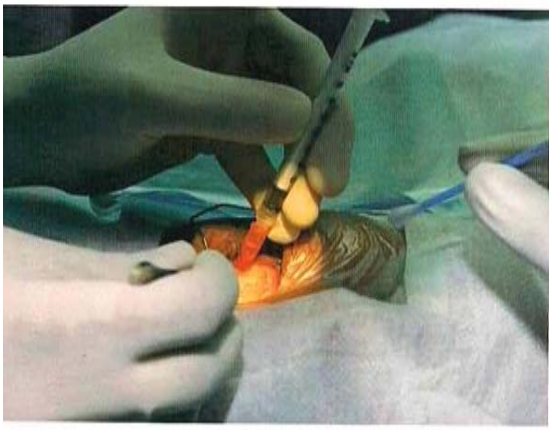
**INTRA VITREAL BEVACIZUMAB**

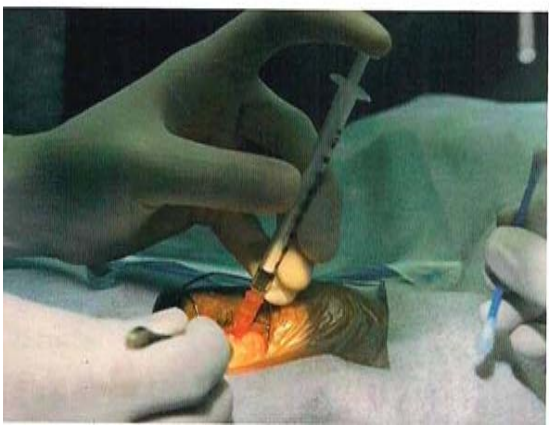
Under aseptic precautions, after instillation of xylocaine (4%) drops, intravitreal bevacizumab (2.5 mg in 0.1 ml) was given. The site of injection was 4mm from the limbus in the inferotemporal quadrant. After the procedure, subconjunctival antibiotics were given and the patient was advised to take acetazolamide tablets and topical antibiotic drops.

# FIGURE 1

## BEVACIZUMAB INJECTED INTO THE EYE

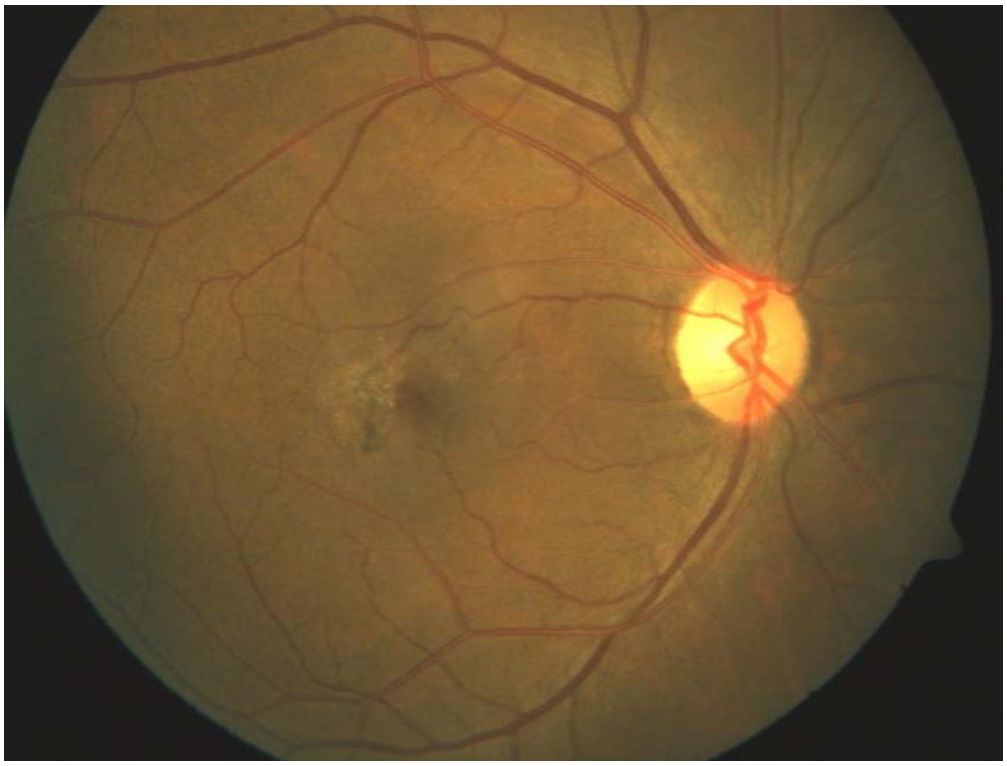


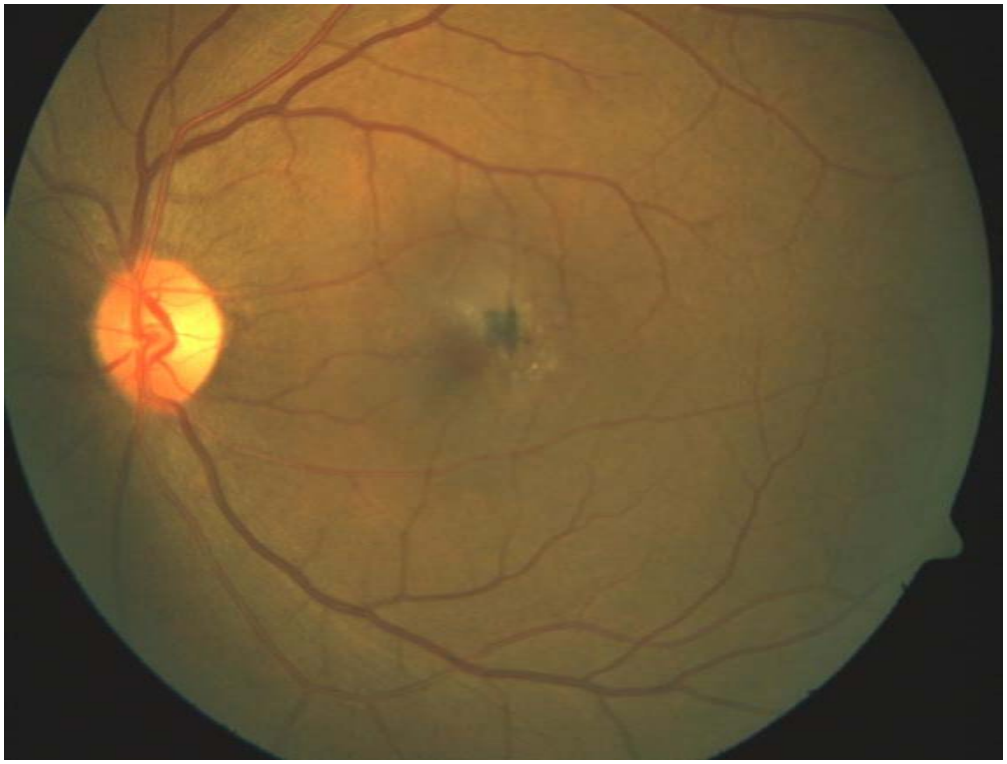




# FIGURE 2

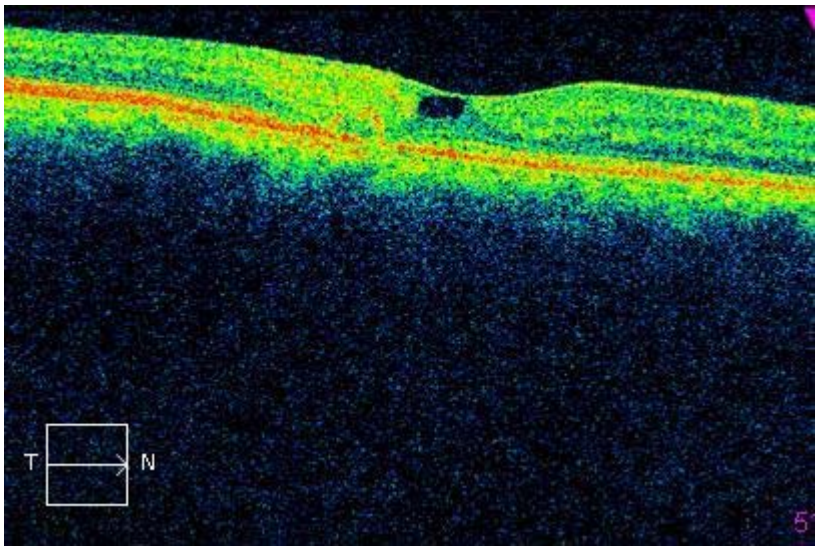
# FUNDUS APPEARANCE OF PARAFOVEAL TELANGIECTASIA





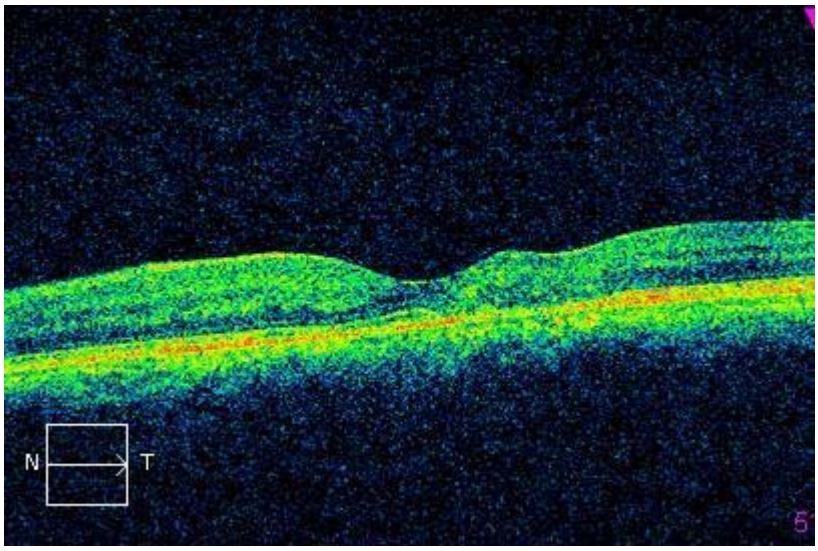
## FIGURE 3

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CYSTIOD MACULAR EDEMA




## FIGURE 4

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE



# RESULTS

Eighty eyes of forty patients were enrolled in the study. Of these 40 patients, fourteen were males (35%) and twenty-six were females (65%) (Figure 5). The patients ranged in age from 40 to 70 years. (Mean age: 54.16 ± [standard deviation {SD}] 8.25 years). Fifteen patients were between 40 to 50 years of age, 14 patients were between 51 to 60 years of age and 11 patients were between 61 to 70 years of age (Figure 6), (Table 1).

All the 40 patients enrolled in the study suffered from bilateral parafoveal telangiectasis. They were divided into three groups:

**Group 1:** This group included patients with PFT who were under observation and treated conservatively. Out of 15 patients in this group, three were males and 12 were females. The age distribution ranged between 40 and 69 years (mean age 52.73 ± 9.05 years). Five patients had both diabetes mellitus (DM) and hypertension (HTN) as associated systemic illnesses whereas one patient had both DM and ischemic heart disease (IHD) and five patients had only DM.

Details about the pre-treatment and post-treatment best corrected visual acuity (BCVA) and macular thickness in each individual eye in this group are shown in Fig. 8a and Fig. 8b, respectively. The mean BCVA at presentation was 0.264 ± 0.14 decimals, which was identical to the mean vision (0.264 ± 0.14 decimals) after the three

month follow-up period. The mean macular thickness at presentation was 205.8 ± 56.32 µ which also showed no change after the three month follow-up period.

**Group 2:** This group included 14 patients with PFT and cystoid macular oedema who were treated with an intravitreal injection bevacizumab (0.1) ml under aseptic conditions. Out of 14 patients, five were males and nine were females. The age distribution ranged from 40 to 65 years (mean age 54.0 ± 7.18 years). Four patients had DM and HTN as associated systemic illnesses, whereas two patients had only HTN and two patients had only DM and one patient had bronchial asthma.

Details about the pre-treatment and post-treatment BCVA and macular thickness values in each individual eye in this group are shown in Figs. 9 a and 11 a and Figs. 9b and 11b, respectively. The mean BCVA at presentation was 0.19 (approximately 6/18) ± 0.1 decimals which, after injection of bevacizumab, showed a significant improvement to 0.26 (6/12) ± 0.096 decimals (`t' test [degree of freedom {d.f.}= 42] = 2.13; P= 0.04) (Figure 13a). The mean macular thickness at presentation was 360.73 ± 43.2 µ which, when compared to the post-bevacizumab macular thickness, showed a significant decrease to 253.45 ± 42.6 µ ('t' test [d.f. = 42] = 8.3; P<0.0001) (Figure 13b). None of the patients exhibited decreased BCVA after intravitreal injection of bevacizumab. Six eyes did not show any change in visual acuity, but showed a significant reduction in macular thickness.

**Group 3:** This group included 11 patients with PFT with choroidal neovascular membrane (CNVM) who were treated with an intravitreal injection of bevacizumab (0.1 ml) under aseptic conditions. Of these 11 patients six were males and five were

females. The age distribution ranged between 45 to 70 years (mean age 56.27 ± 8.5 years). Two patients had both DM and HTN, whereas one patient had both DM and IHD.

Details about the pre-treatment and post-treatment BCVA and macular thickness values in each individual eye in this group are shown in Figs 10 a and 12a and Figs. 10 b and 12 b, respectively. The mean BCVA at presentation was 0.127(approximately 6/60) ± 0.08 decimals, which was not significantly different from the post-bevacizumab injection BCVA value of 0.146 (approximately 6/36) ± 0.07 decimals (`t test [d.f.= 34] = 0.95; P= 0.34). However, there was a significant difference (`t' test [d.f. = 34] = 6.76; P= 0.001) between the mean macular thickness at presentation (469.17 ± 89.67 μ) and the mean macular thickness (310.78 ± 85.61) μ after the intravitreal injection of bevacizumab. One eye showed a decreased BCVA (from 6/60 to 3/60) following intravitreal bevacizumab; however, the same eye exhibited a reduction in macular thickness (from 417μ to 313μ) following the injection. Eight eyes did not exhibit any change in visual acuity, but showed a significant reduction in macular thickness

Comparisons were made between the three groups at several levels, namely:

**a) Age:** Differences between the three groups in the mean ages of the patients in the groups were analysed by one-way analysis of variance (ANOVA); the differences were not statistically significant (Fisher `f' value= 0.614; P= 0.546).

**b) Gender:** Males accounted for 20 % of patients in Group 1, 36 % of patients in Group 2 and 55 % of patients in group 3; these differences were not statistically significant (chi-square (d.f.=2)=2.7; P> 0.05).

**c) Pre-intervention mean visual acuity:** Differences between the groups in the pre-intervention mean visual acuity were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 11.018; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

**d) Post-intervention mean visual acuity:** Differences between the groups in the post-intervention mean visual acuity were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 10.329; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 3 and Group 2 vs. Group 3; however, the difference between the mean values in Group 1 and Group 2 were not statistically significant.

**e) Pre-intervention mean macular thickness:** Differences between the groups in the pre-intervention mean macular thickness were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 112.4; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

**e) Post-intervention mean macular thickness:** Differences between the groups in the post-intervention mean macular thickness were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 18.753; P= 0.000). Post-hoc

testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

Although Group 1 did not differ significantly from Groups 2 and 3 in the mean age of the patients, there was a definite preponderance of female patients in group 1, which was similar to the pattern in Group 2 but different from the pattern in Group 3 which showed a preponderance of male patients. However, in the 30 eyes of 15 patients who suffered from PFT alone, the pre-intervention and post-intervention mean visual acuities and the pre-intervention and post-intervention macular thickness values were all significantly better than the values noted in the 22 eyes of 14 patients with PFT and CME which received intravitreal bevacizumab. Similarly, in the 30 eyes of 15 patients who suffered from PFT alone, the pre-intervention mean visual acuity and the pre-intervention and post-intervention macular thickness values were all significantly better than the values noted in the 18 eyes of 11 patients with PFT and CNVM which received intravitreal bevacizumab; however, there was no significant difference between the post-intervention mean visual acuity in the eyes of these 2 groups of patients.

**FIGURE 5**

**GENDER OF THE 40 PATIENTS WHO PRESENTED WITH PARAFOVEAL TELANGIECTASIA**

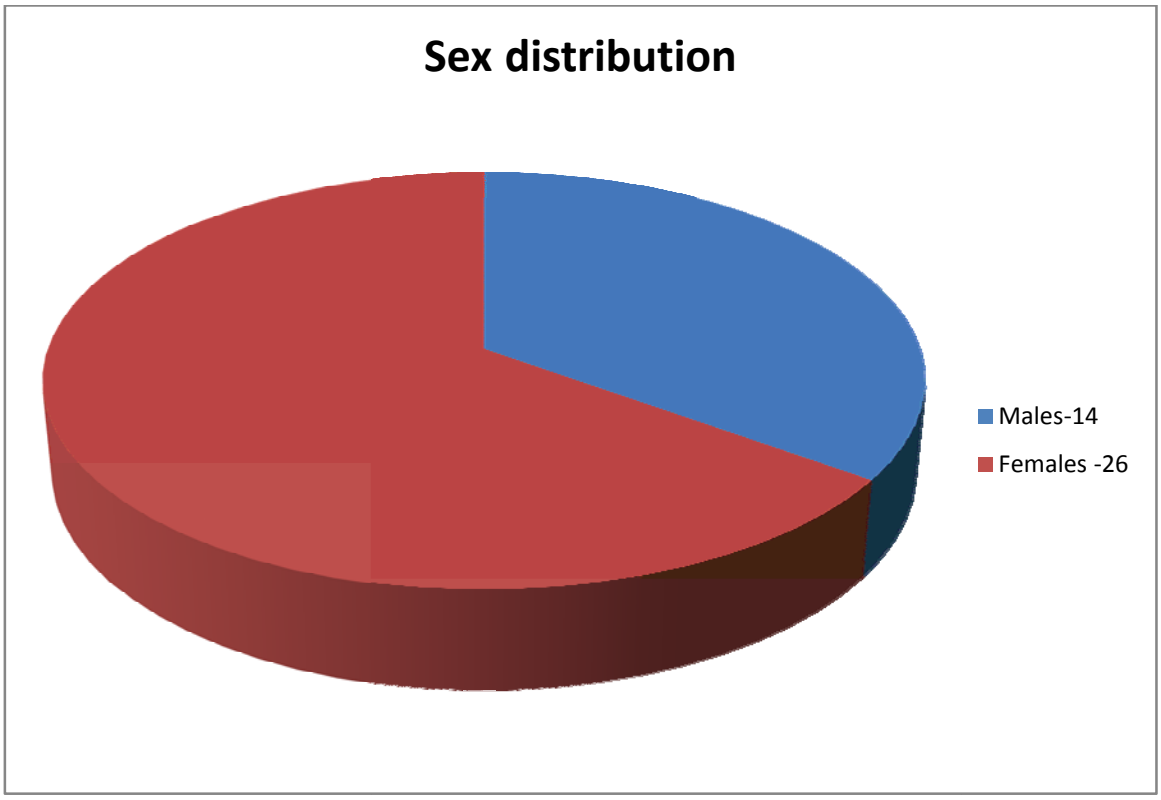


**TABLE 1**

**AGE CATEGEORIES OF THE 40 PATIENTS WHO PRESENTED WITH PARAFOVEAL TELANGIECTASIA**

| Age group (Years) | No. of patients | Percentage (%) |
|---|---|---|
| 40 – 50 | 15 | 37.5 % |
| 51 – 60 | 14 | 35 % |
| 61 – 70 | 11 | 27.5 % |
| Total | 40 | 100 |

**MEAN AGE: 54.16 ± 15 YEARS (RANGE =40 YEARS TO 70 YEARS) MEDIAN: 55 YEARS**

**FIGURE 6**

**AGE CATEGEORIES OF THE 40 PATIENTS WHO PRESENTED WITH PARAFOVEAL TELANGIECTASIA**

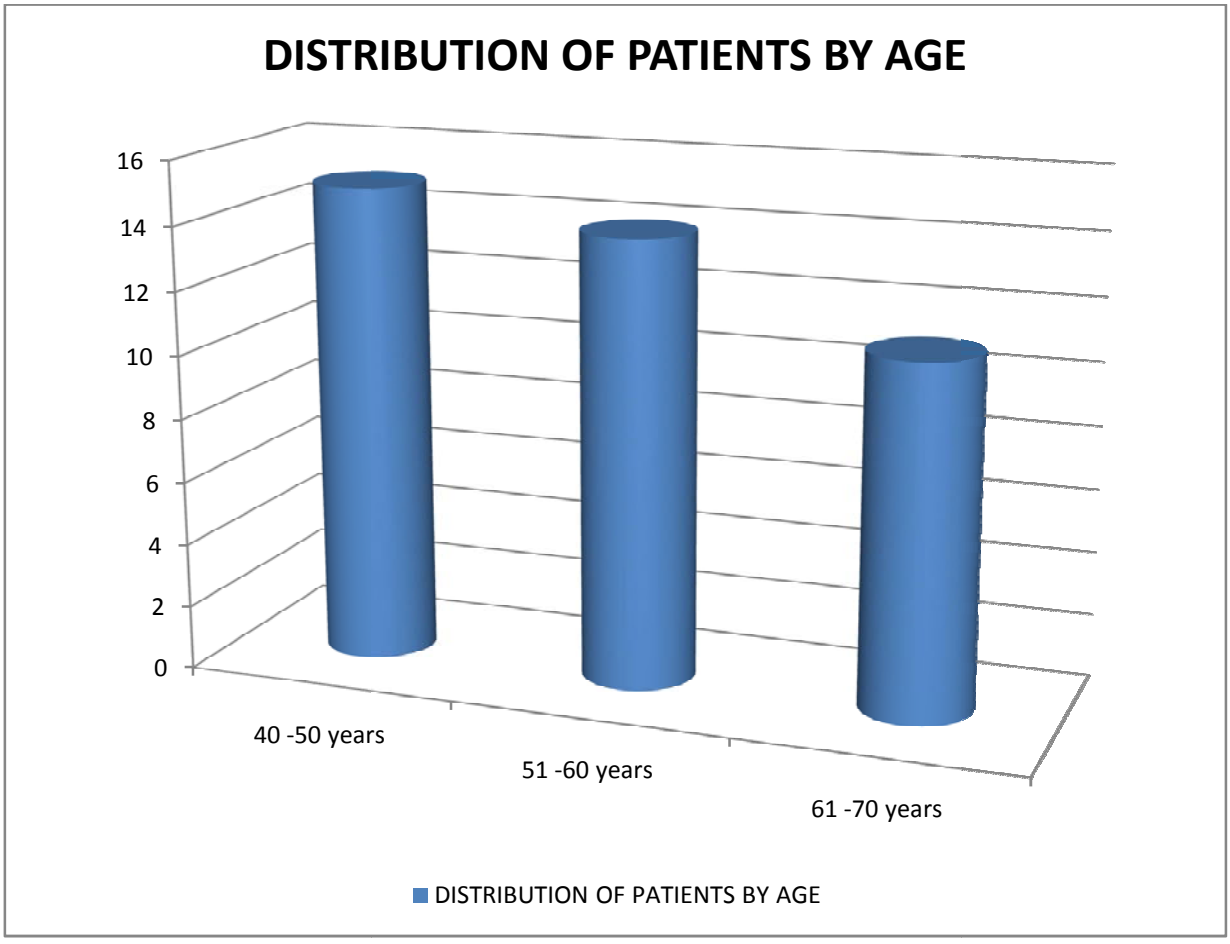


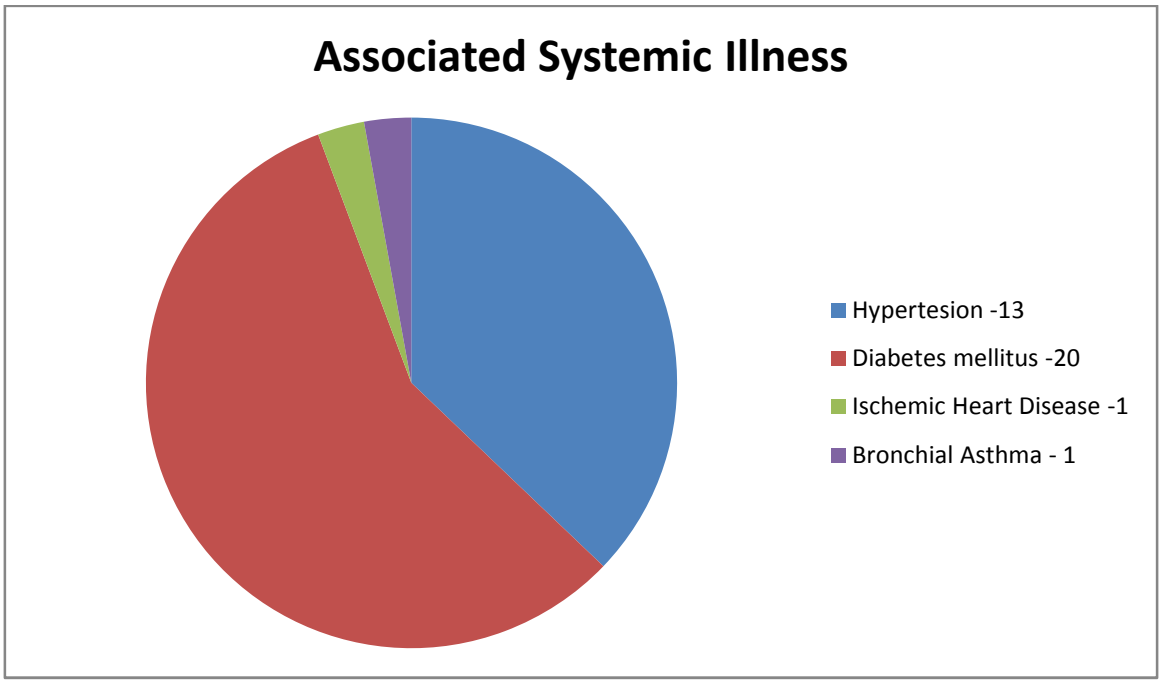
**TABLE 2**

**ASSOCIATED SYSTIMIC DISEASES IN THE 40 PATIENTS WHO PRESENTED WITH PARAFOVEAL TELANGIECTASIA**

| ASSOCIATED DISEASES | NUMBER OF PATIENTS AFFECTED |
|---|---|
| HYPERTENSION | 13 |
| DIABETES MELLITUS | 20 |
| ISCHEMIC HEART DISEASES | 1 |
| BRONCHIAL ASTHMA | 1 |

**FIGURE 7**

**ASSOCIATED SYSTIMIC DISEASES IN THE 40 PATIENTS WHO PRESENTED WITH PARAFOVEAL TELANGIECTASIA**



# FIGURE 8

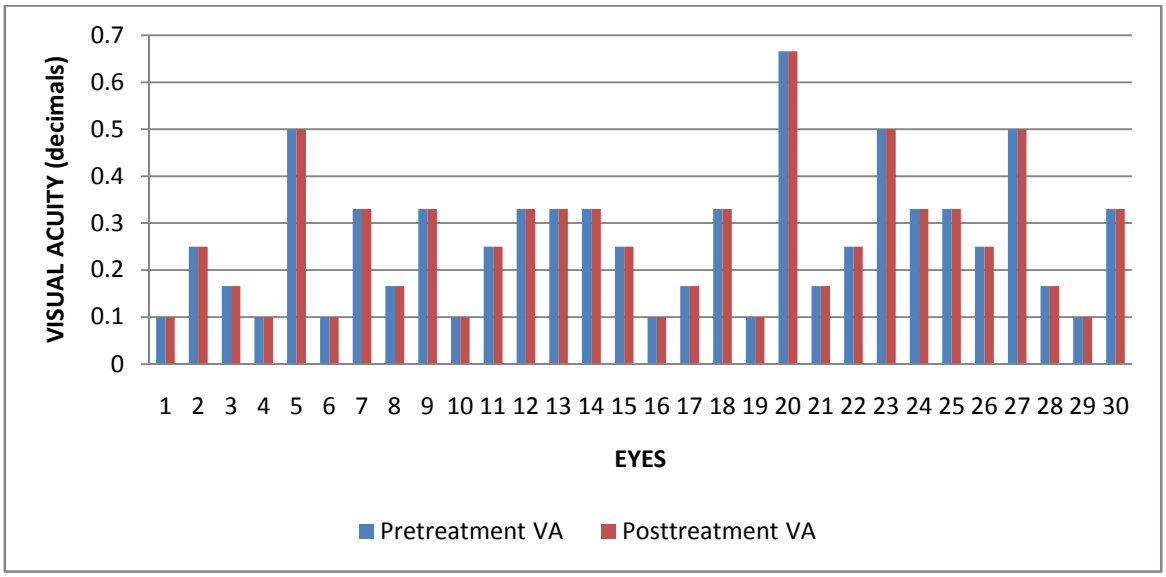
## COMPARISON OF PRE-TREATMENT AND POST-TREATMENT

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 30 EYES OF PARAFOVEAL TELANGIECTASIA ONLY

## a) BEST CORRECTED VISUAL ACUITY (decimals)



# FIGURE 8

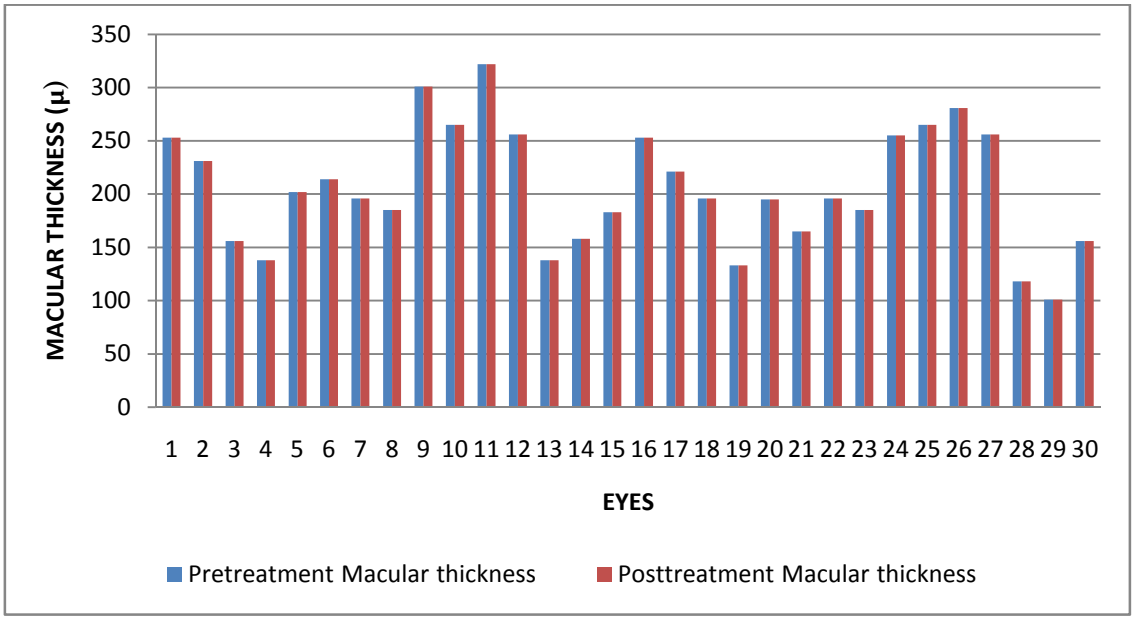
## COMPARISON OF PRE-TREATMENT AND POST-TREATMENT

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 30 EYES OF PARAFOVEAL TELANGIECTASIA ONLY

### b) MACULAR THICKNESS (µ)



# FIGURE 9

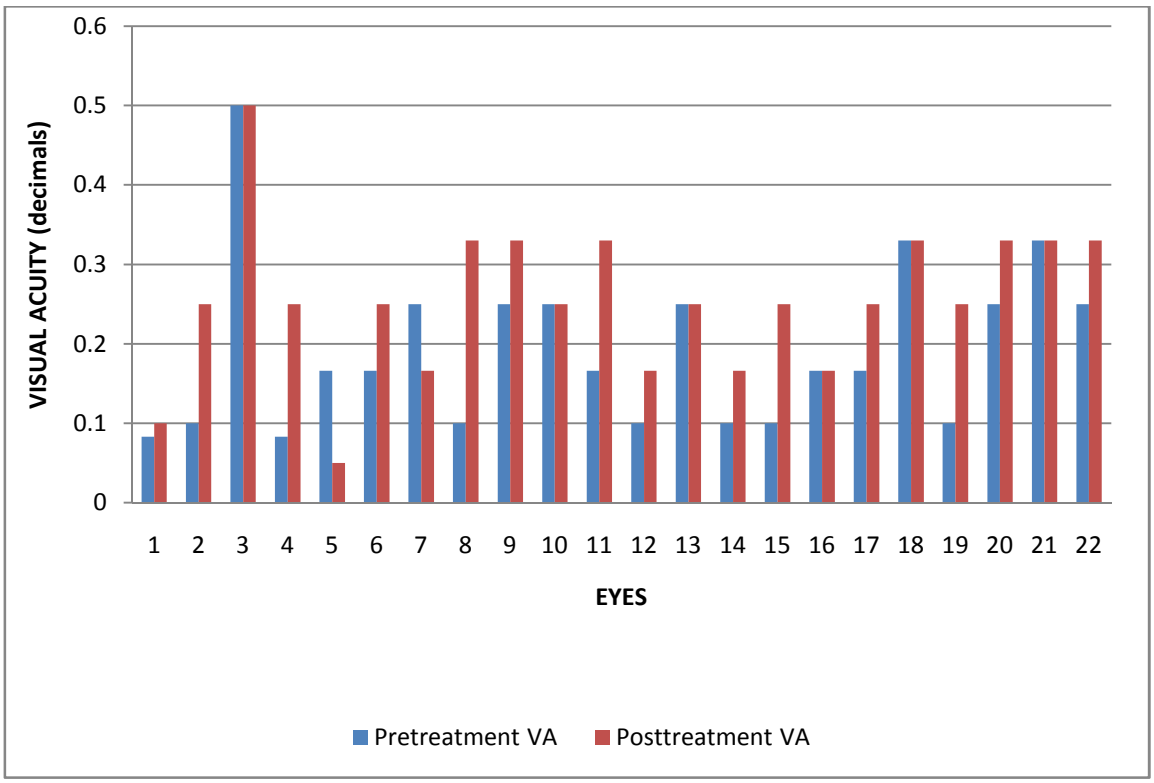
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 22 EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

## a) BEST CORRECTED VISUAL ACUITY (decimals)



# FIGURE 9

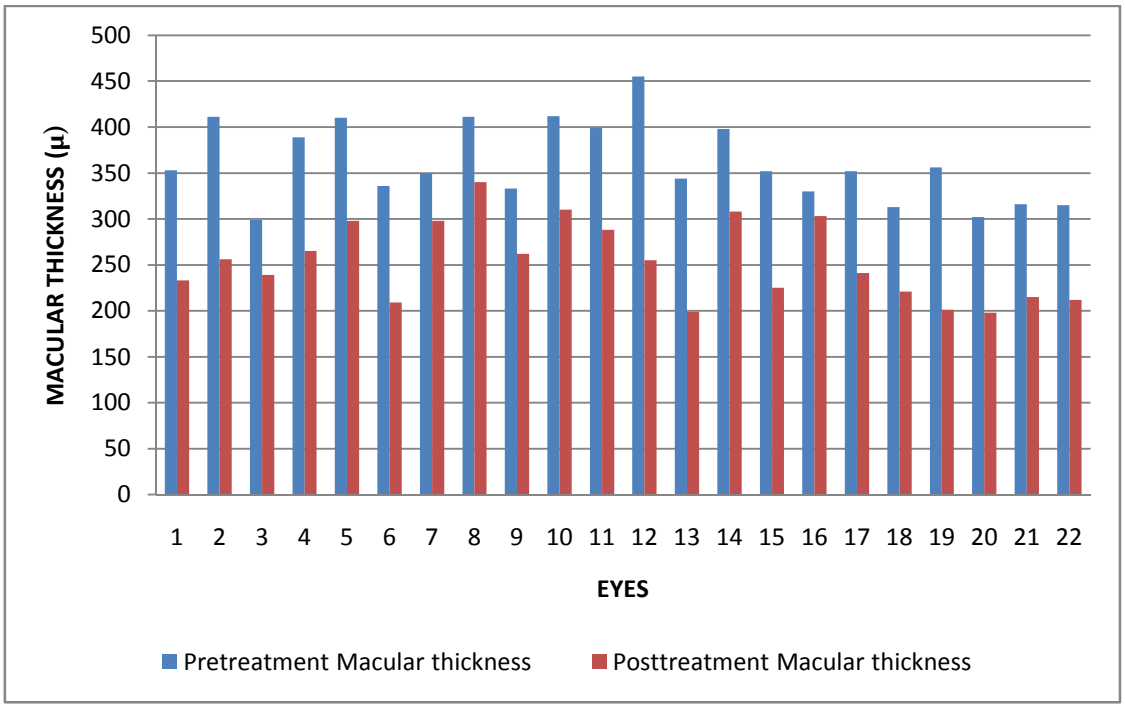
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 22 EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

### b) MACULAR THICKNESS (µ)



# FIGURE 10

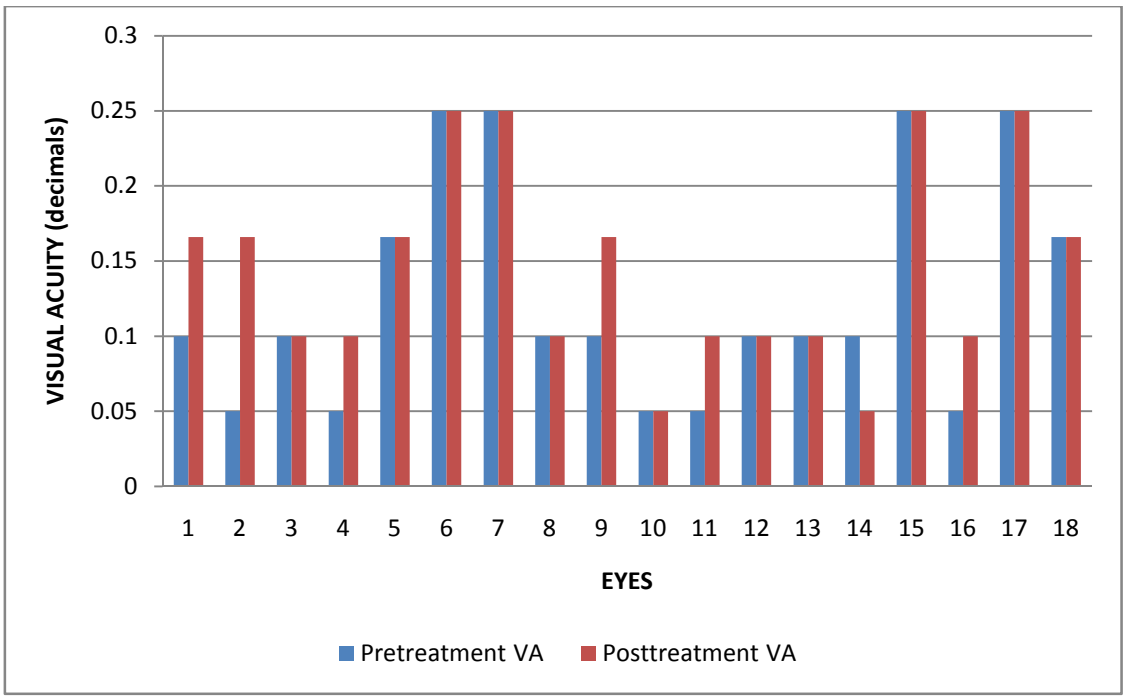
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 18 EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE

## a) BEST CORRECTED VISUAL ACUITY (decimals)



## FIGURE 10

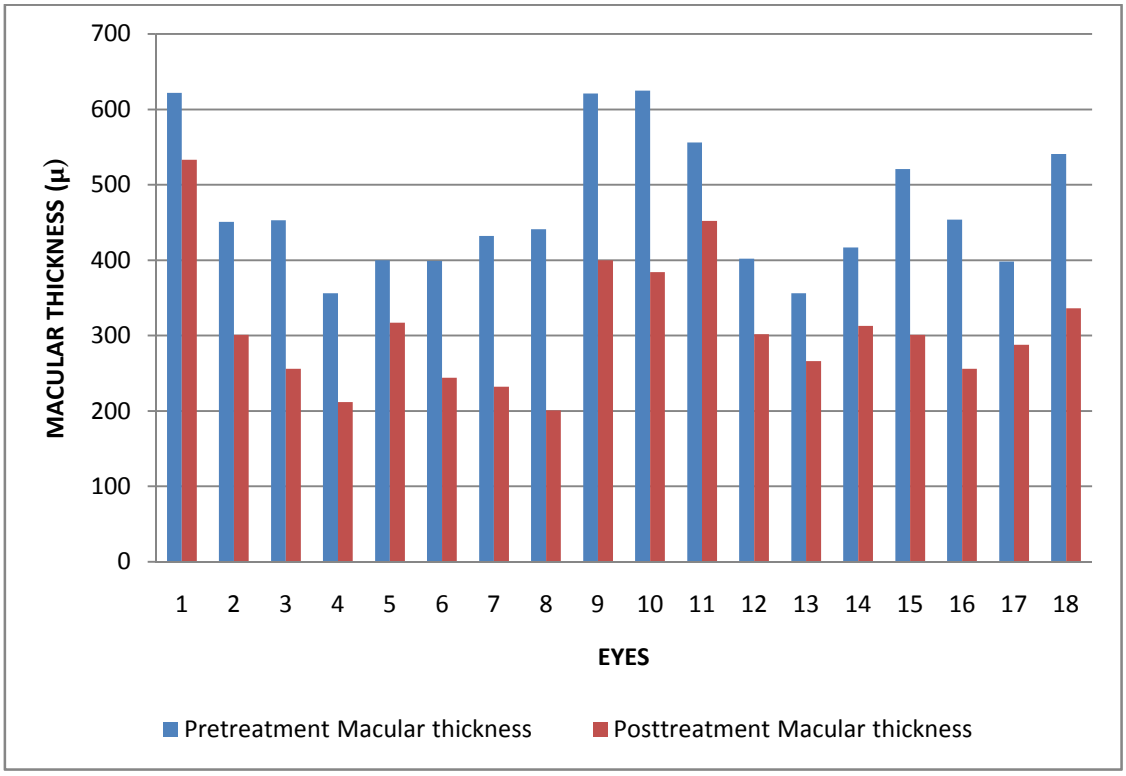
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 18 EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE

### b) MACULAR THICKNESS (µ)



# FIGURE 11

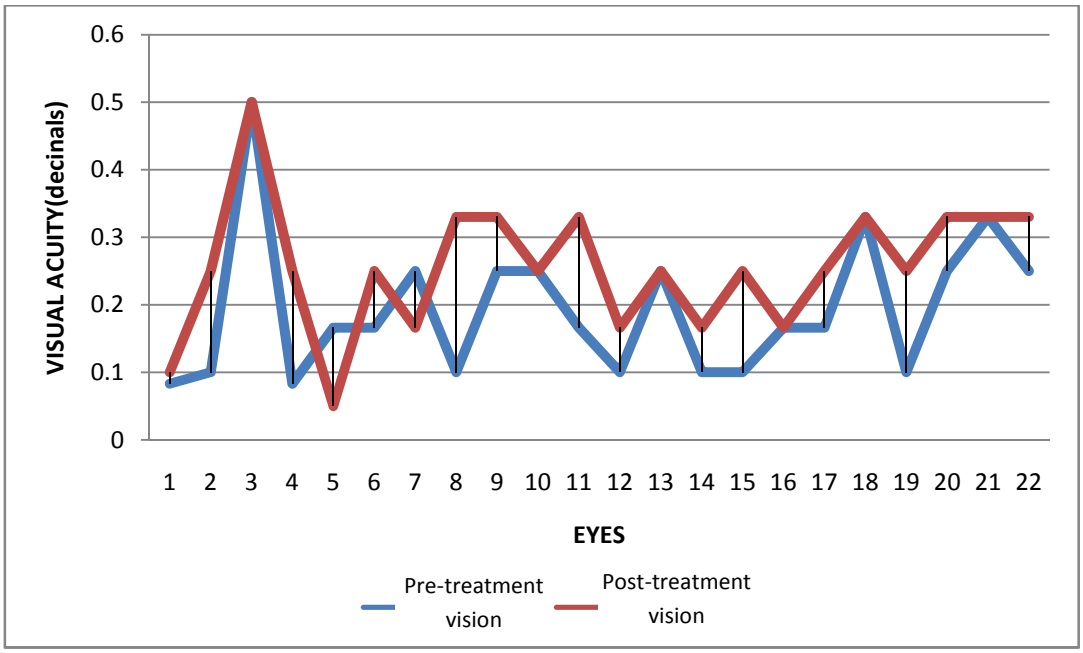
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 22 EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

## a) BEST CORRECTED VISUAL ACUITY (decimals)



## FIGURE 11

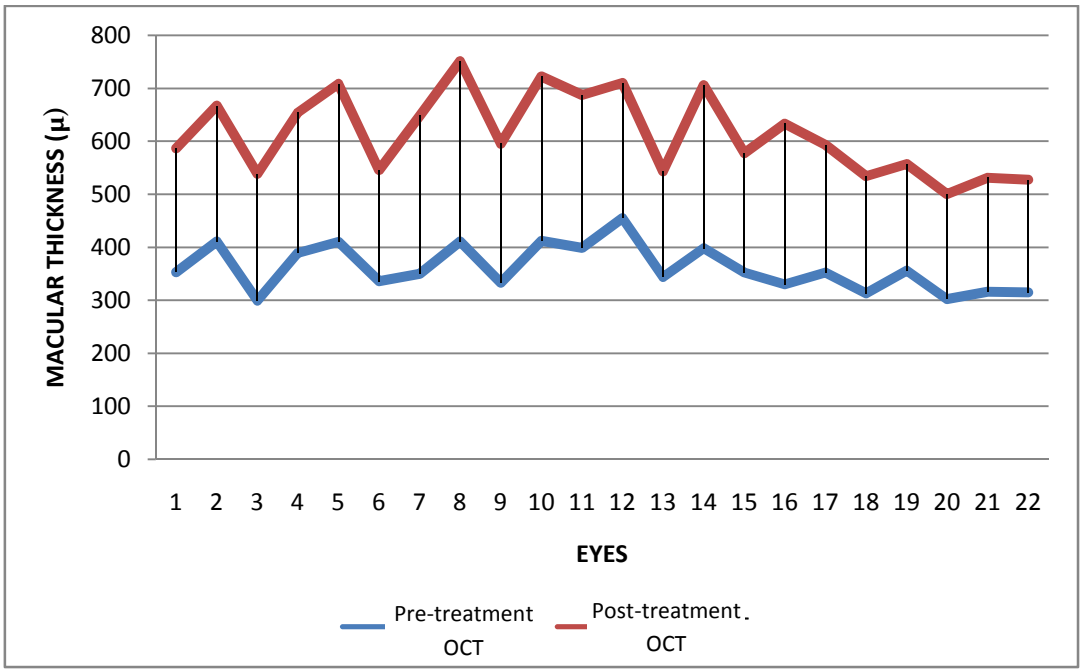
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 22 EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

### b) MACULAR THICKNESS (µ)



## FIGURE 12

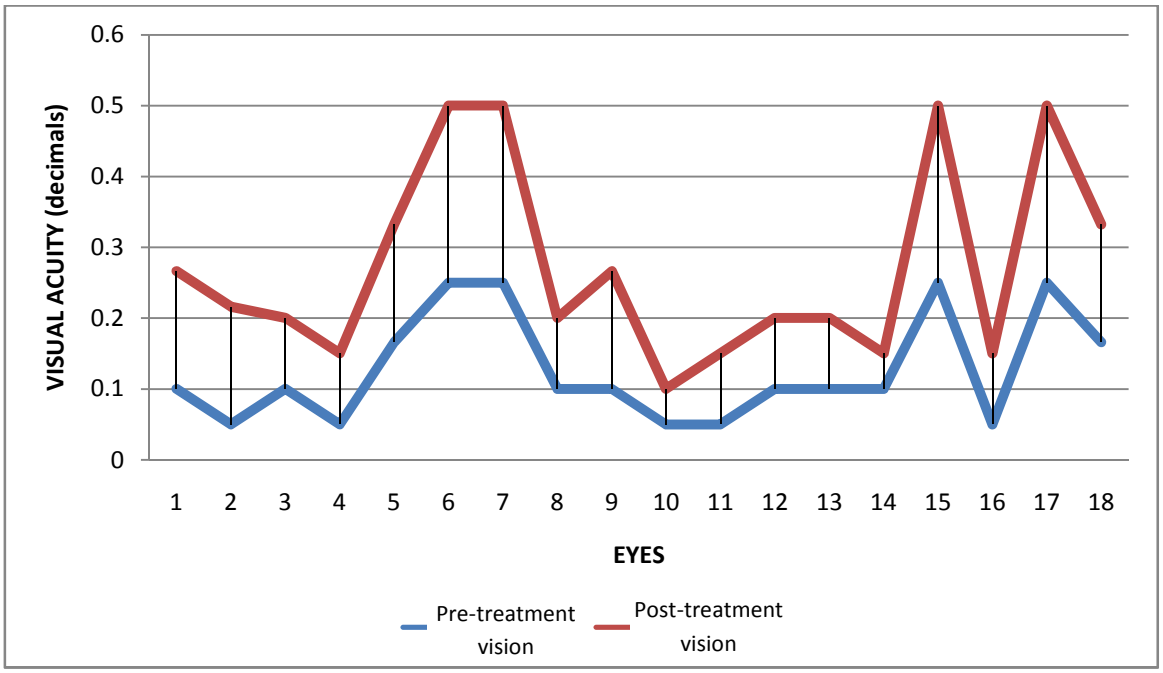
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 18 EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE

## a) BEST CORRECTED VISUAL ACUITY (decimals)



## FIGURE 12

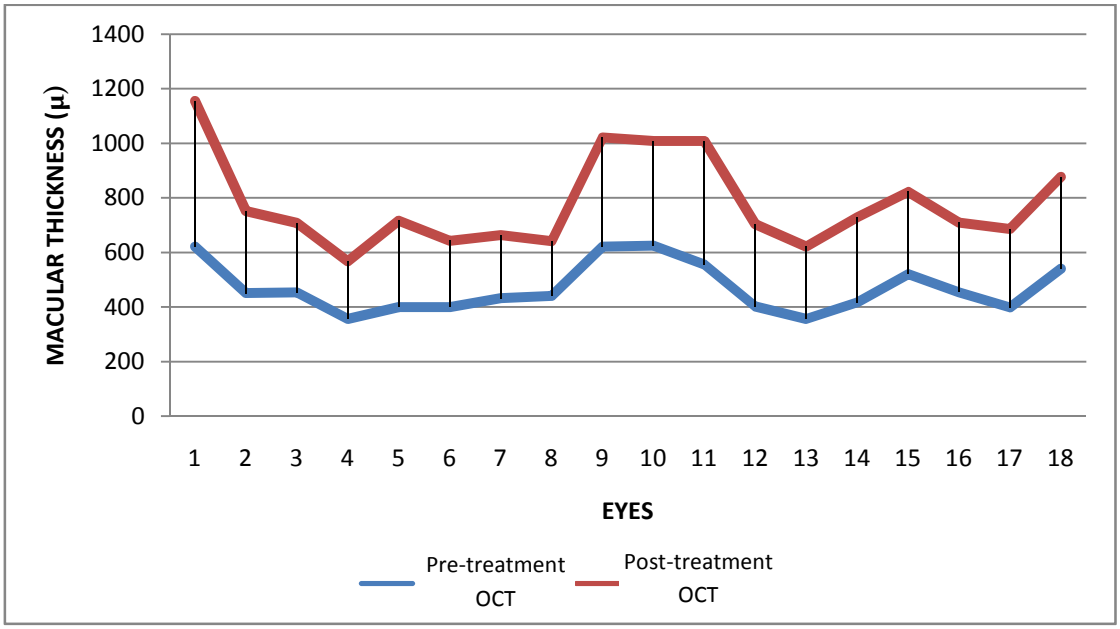
## COMPARISON OF THE PRE-INJECTION AND POST-INJECTION

## a) BEST CORRECTED VISUAL ACUITY AND

## b) MACULAR THICKNESS

## IN 18 EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE

### b) MACULAR THICKNESS (µ)



# FIGURE 13

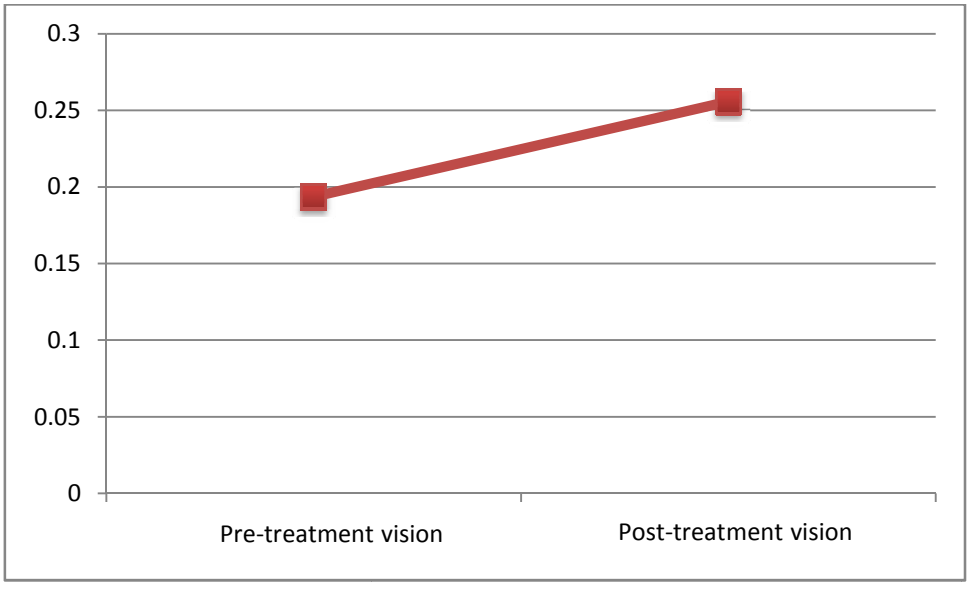
## COMPARISON OF PRE-INJECTION AND POST-INJECTION

## a) MEAN BEST CORRECTED VISUAL ACUITY AND

## b) MEAN MACULAR THICKNESS

## IN 22EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

## a) BEST CORRECTED VISUAL ACUITY (decimals)



# FIGURE 13

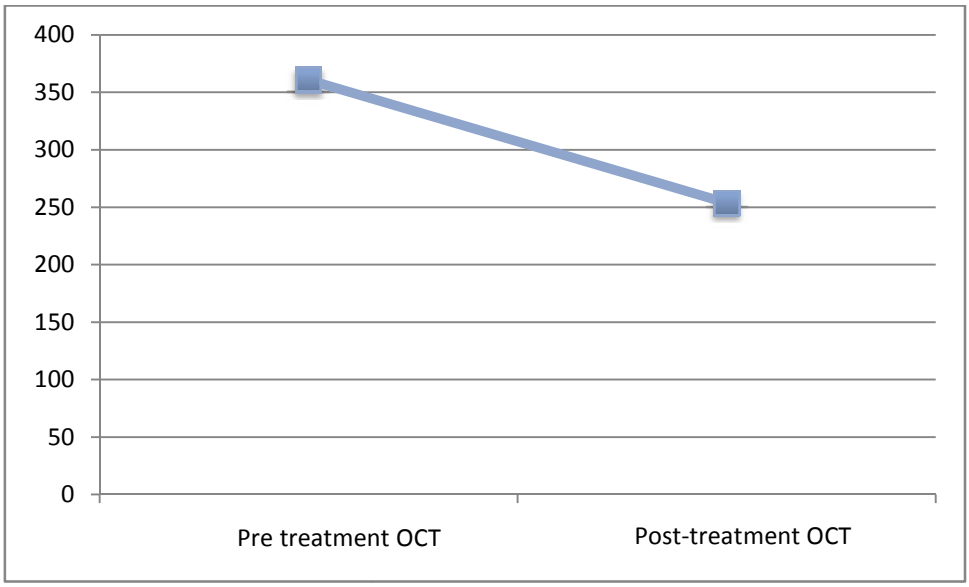
## COMPARISON OF PRE-INJECTION AND POST-INJECTION

## a) MEAN BEST CORRECTED VISUAL ACUITY AND

## b) MEAN MACULAR THICKNESS

## IN 22EYES OF PARAFOVEAL TELANGIECTASIA WITH CYSTOID MACULAR EDEMA

## b) MACULAR THICKNESS (µ)



# FIGURE 14

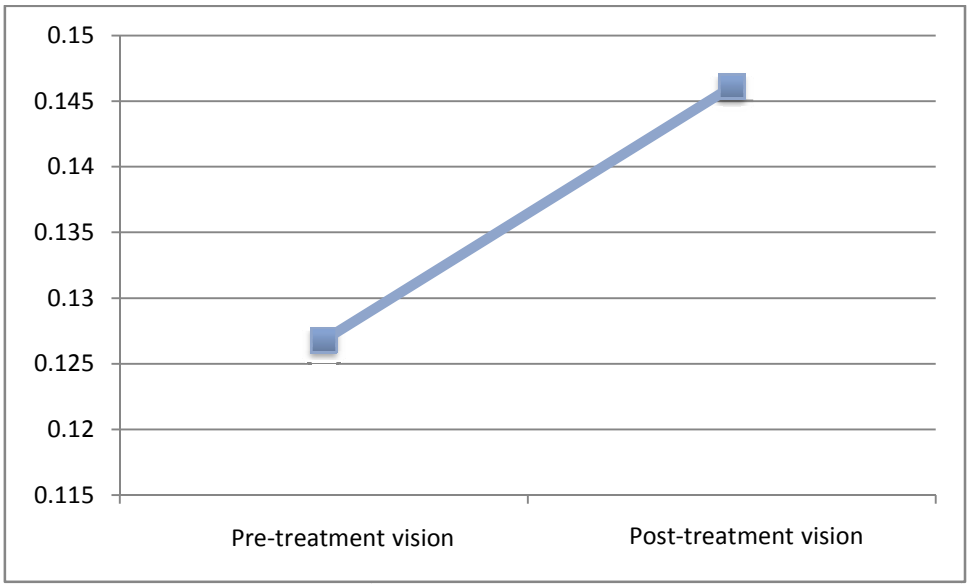
## COMPARISON OF PRE-INJECTION AND POST-INJECTION

## a) MEAN BEST CORRECTED VISUAL ACUITY AND

## b) MEAN MACULAR THICKNESS

## IN 22EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBRANE

## a) BEST CORRECTED VISUAL ACUITY (decimals)



# FIGURE 14

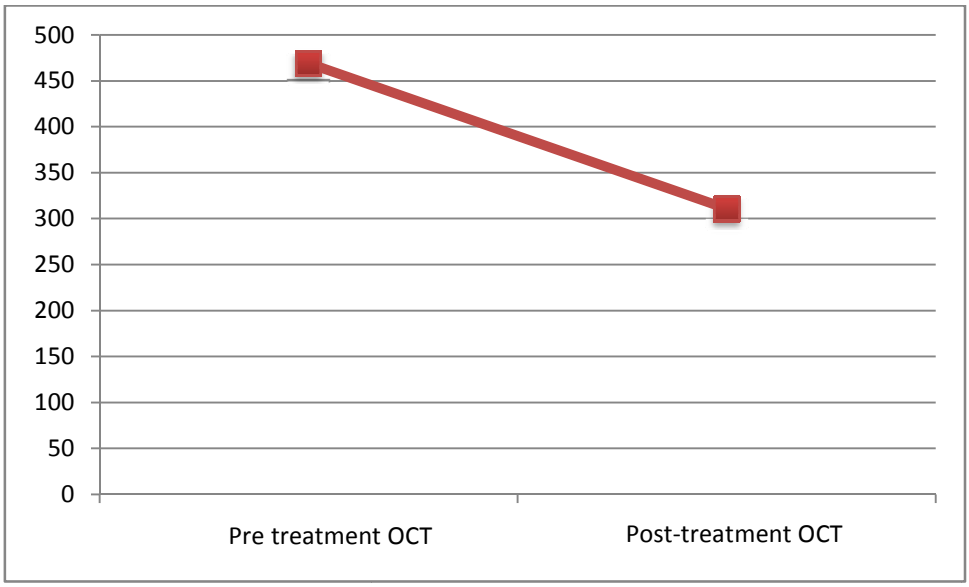
## COMPARISON OF PRE-INJECTION AND POST-INJECTION

## a) MEAN BEST CORRECTED VISUAL ACUITY AND

## b) MEAN MACULAR THICKNESS

## IN 22EYES OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBRANE

**b) MACULAR THICKNESS (µ)**



## TABLE 3

**Comparison of mean age, pre-intervention and post-intervention best corrected visual acuity and pre-intervention and post-intervention macular thickness in patients and eyes with parafoveal telangiectasis**

| Parameter | Group 1 | Group 2 | Group 3 |
|---|---|---|---|
| Age | **52.73±9.05 (15 patients)** | **53.5±7.18 (14 patients)** | **56.27±8.50 (11 patients)** |
| Pre-intervention Visual acuity | **0.264±0.14 (30 eyes)** | **0.193±0.1 (22 eyes)** | **0.127±0.08 (18 eyes)** |
| Post-intervention Visual acuity (decimals) | **0.264±0.14 (30 eyes)** | **0.256±0.096 (22 eyes)** | **0.146±0.07 (18 eyes)** |
| Pre-intervention Macular thickness (μ) | **205.8±56.32 (30 eyes)** | **360.73±43.32 (22 eyes)** | **469.17±89.67 (18 eyes)** |
| Post- intervention Macular thickness (μ) | **205.8±56.32 (30 eyes)** | **253.45±42.6 (22 eyes)** | **310.78±85.61 (18 eyes)** |

**Group 1: 30 eyes with parafoveal telangiectasis only**

**Group 2: 22 eyes with parafoveal telangiectasis and cystoid macular edema**

**Group 3: 18 eyes with parafoveal telangiectasis and choroidal neovascular membrane**

**Statistical analysis of differences between the 3 groups of patients/eyes**

**1. Age.** One way analysis of variance (ANOVA); Fisher `f' value= 0.614; P= 0.546 (not significant).

**2. Pre intervention best corrected visual acuity (BCVA).**

a) One way ANOVA; Fischer 'f' value=11.018; P=0.000 (significant)

b) Post hoc testing (Tukey's). Group I vs II;q=11.186;P<0.001(significant)

Group I vs III;q=5.8;P<0.01(significant)

Group II vs III;q=-5.38;P<0.01(significant)

**3. Post intervention BCVA :**

a) One way ANOVA; Fischer 'f' value=10.329; P=0.000(significant)

b) Post- hoc testing (Tukey's). Group I vs II;q=0.653;P>0.05(not significant)

Group I vs III; q=9.363;P<0.001(significant)

Group II vs III;q=8.98;P<0.001(significant)

**4. Pre intervention macular thickness.**

a) One way ANOVA; Fischer 'f' value=112.4; P=0.000

b) Post hoc testing (Tukey's). Group I vs II; q=20.6; P<0.0001

Group I vs III; q=35.0;P<0.0001

Group II vs III;q=14.42;P<0.001

**5. Post intervention macular thickness.**

a) One way ANOVA; Fischer ‘f’ value=18.753; P=0.000

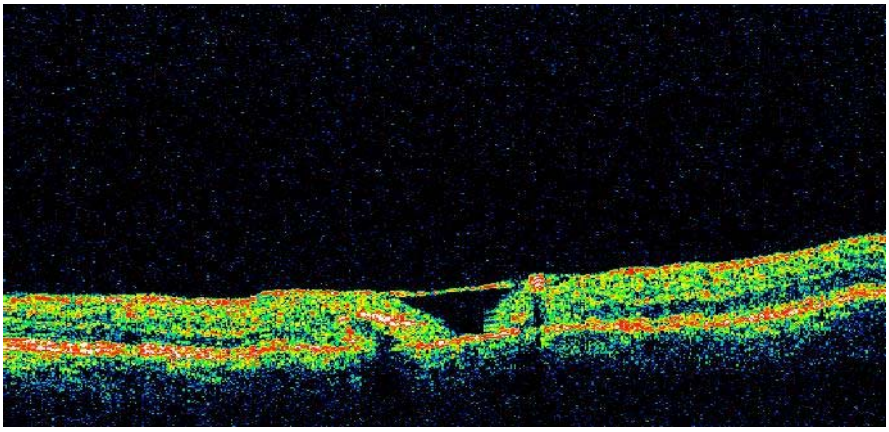
b) Post hoc testing (Tukey’s). Group I vs II;q=6.53;P<0.01

Group I vs III;q=14.38;P<0.001

Group II vs III;q=7.85;P<0.01

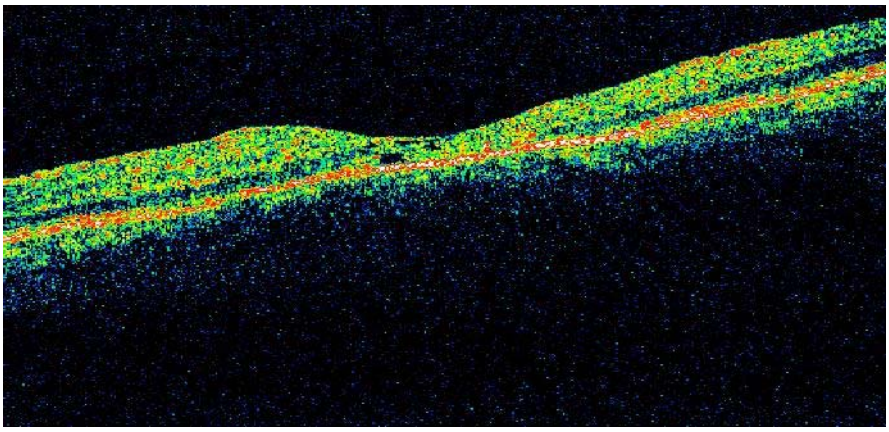
## FIGURE 15

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CYSTIOD MACULAR EDEMA BEFORE TREATMENT



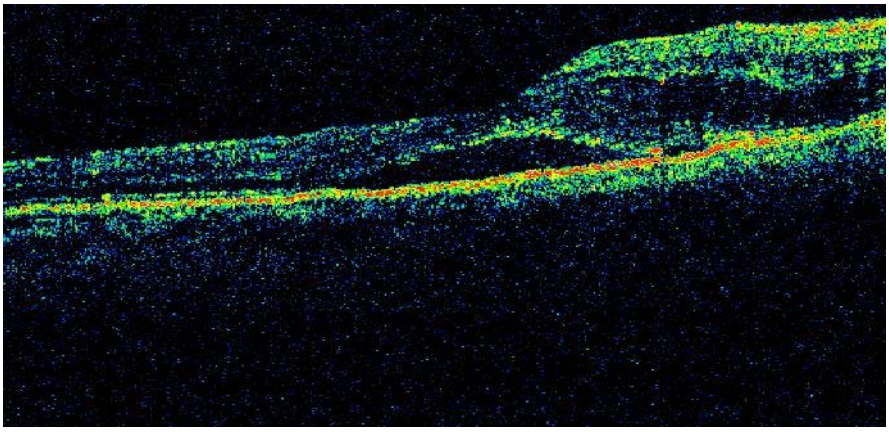
## FIGURE 16

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CYSTIOD MACULAR EDEMA AFTER TREATMENT



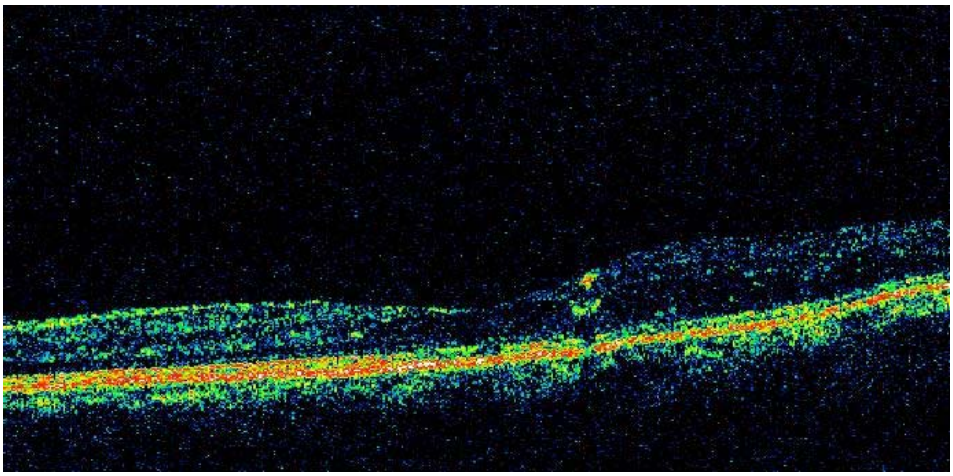
## FIGURE 17

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE BEFORE TREATMENT



## FIGURE 18

## OCT APPEARANCE OF PARAFOVEAL TELANGIECTASIA WITH CHOROIDAL NEOVASCULAR MEMBERANE AFTER TREATMENT



# DISCUSSION

Type 2 parafoveal telangiectasia is the most common type of parafoveal telangiectasia. It is acquired, not congenital. Affected patients are middle-aged or older (mean 55 years). Males and females are affected equally. This disorder is bilateral, but may be asymmetric appearing as unilateral in its early stages. Similarly, patients may have visual loss in only one eye.

The natural course of Idiopathic juxta foveal telangiectasia (IJFT) II has been subdivided by Gass and Blodi into five stages.

Three key and distinguishing features of IJFT II are:

1. The absence of prominent aneurysms or haemorrhage;
2. The absence of cystic macular edema (CME) or lipid exudation (unless Subretinal neovascularisation (SRNV) has developed). The loss of retinal transparency and fluorescein staining are primarily caused by intracellular edema (contrary to the extracellular fluid causing CME and lipid exudation in IJFT I); and
3. The presence of foveolar atrophy, best seen with OCT, which can simulate a lamellar macular hole. Foveal atrophy is the primary cause of the slow progressive visual loss occurring over years in these patients (to 20/200 or worse), and is distinguishable from the rapid and severe visual loss that may occur with the advent of SRNV and fibrosis.

The following OCT features have been described in Type 2 Parafoveal telangiectasia

1. Retinal thickness is variable and does not correlate with the degree of leakage seen on FA.[46,47,49–52] Central foveolar thickening is consistently absent,[47] and intraretinal edema is either absent[6] or minimal,[3,7,47,49–52] superimposed on a fovea of normal or reduced thickness.[47,53–54]
2. Thinning and disruption of the photoreceptor layer, more readily visualized by ultra-high-resolution OCT, is common,[7,47,48] confirming that foveal atrophy is the primary cause for reduced vision. This disruption was found to correlate with VA in 63% of eyes[7] and increased with advancing disease.[47,48] This finding may be of importance in guiding treatment decisions.
3. Cyst-like structures in the foveola and inner retinal layers are very common (50–100% of eyes with Stage 3 or higher. [3,6,7,47–49,51,52] They are referred to as "cystoids," have variable size and are not seen clinically or on FA.[48] Because of the consistent absence of associated cystoid macular edema[6,47] or petaloid pooling on FA, these cysts are unlikely a result of exudation, but rather a progressive retinal tissue loss.[6,47,49] At the foveola, the inner lamellar cyst appears as a loss of tissue[3,7] with the internal limiting membrane (ILM) spanning across it and draping over it.[7] This unique feature, the ILM drape, may be specific to IJFT IIA.[7] This feature is what was previously described as a lamellar retinal hole by Gass who did not have the advantage of OCT imaging.[2,5] Full-thickness macular holes are uncommon in IJFT IIA but have been reported.[55,56] In most eyes with cysts VA is decreased (20/40 to 20/70),[6]

and cyst enlargement has been reported with disease progression and more pronounced visual loss.[3,6]

4. Blunting of the foveal pit is common to all stages.[48] Foveal flattening or thinning is encountered with more advanced disease.[6]
5. Intraretinal neovascularization near the foveola, seen as highly reflective dots in the inner and outer nuclear layers[48] is found in 21% of eyes.[7]
6. Central intraretinal hyper-reflective lesions that cause nonspecific posterior shadowing and correspond to hyperpigmented RPE plaques are observed.[3,5,57] The crystalline deposits are generally too small to induce photoreflectance.[3]

When considering treatment, the therapeutic attempts for nonproliferative IJFT II, and treatment modalities for the SRNV of the proliferative stage must be distinguished.

The angiographic late intraretinal staining pattern in IJFT IIA has prompted many ophthalmologists to interpret it as macular edema secondary to retinal vascular leakage. Several treatment modalities have been tried to treat this “macular edema.”

To start, argon laser photocoagulation (ALPC) is not effective in the treatment of nonproliferative IJFT IIA.[3,-11]. Gass and Blodi noted that the long-term prognosis for patients with IJFT IIA is poor and laser treatment resulted in either worsened or no change in VA. Gass did not recommend ALPC in nonproliferative IJFT. Park *et al.* [3,] also found no improvement or stabilization of vision with grid ALPC.

Verteporfin photodynamic therapy (PDT) has also been tried in patients with nonproliferative IJFT II in an attempt to reduce the permeability of the telangiectatic vessels.[58] However, PDT improved neither vision nor the macular edema and appeared therefore not effective. No adverse effects were reported.

Preliminary case reports (five cases) of Intravitreal triamcinolone acetate( IVTA) in nonproliferative IJFT II suggested an effect, with mild improvement of visual acuity, but the follow-up was very short[59–61]. The retrospective study of 19 eyes by Wu *et al*., however, showed that IVTA, at a dose of 4 mg, does not improve VA in most eyes with nonproliferative IJFT I1[62]. IVTA is likely to have a minor, or no, therapeutic effect in nonproliferative IJFT IIA, which, coupled with a high incidence of cataract and increased intraocular pressure, suggest that it should be avoided as treatment of this condition.

Recent publications on intravitreal anti-VEGF injections, namely bevacizumab, report on possible short-term VA increase in some cases of IJFT II [63, 64]. In one case, with initial VA 20/50, treated with an intravitreal injection of bevacizumab (1.25 mg), VA improved but macular edema recurred after 3 months, requiring a repeat injection with no significant improvement of VA thereafter.[64]. Another retrospective series of seven eyes treated with two doses of intravitreal bevacizumab (1.5 mg) at 4-week intervals demonstrated a short-term decrease in retinal thickness and a reduction in angiographic leakage[63]. When these same patients received further injections, depending on disease activity, and were followed for approximately 18 months, it was noted that mean VA had increased, central retinal thickness had decreased following each treatment, but treatment effect appeared less pronounced with subsequent

injections, and rebound effect recurred earlier than it had after the initial two injections. At the last visit, retinal thickness had increased in selected retinal sectors including the fellow eye. The authors concluded that intravitreal bevacizumab for nonproliferative IJFT II has only a transient effect. They believed that VEGF plays a pathophysiological role in IJFT II, because, in their opinion, the structural capillary changes described histopathologically lead to a disturbed exchange of oxygen and substrates between the vascular lumen and neurosensory retina, which in turn may lead to a hypoxia-induced increased VEGF release by retinal cells.

However, in another two patients with nonproliferative IJFT IIA treated with intravitreal bevacizumab (1.25 mg) and followed for 12 months, leakage on FA decreased likewise, underlining the effect of bevacizumab on vessel stability and permeability, but this was not accompanied by an increase in VA despite triple injections.[66] Furthermore, small cystic changes seen on OCT remained unchanged, emphasizing that visual deterioration is caused by microcystic degeneration and progressive retinal atrophy and not by intraretinal edema, and therefore cannot be halted with intravitreal anti-VEGF injections. A more recent and larger retrospective review of nine eyes treated with intravitreal bevacizumab and followed from 4 to 27 months, corroborated that intravitreal bevacizumab decreased FA leakage but had no short-term effect on VA or OCT appearance.[67] Similar results to these two reports were observed with intravitreal injections of pegaptanib.[68] These three studies suggest that eyes with minimal cystic changes on OCT do not show functional improvement despite repeated intravitreal anti-VEGF injections,[66,68] and that there seems to be no apparent visual acuity or OCT benefit to using intravitreal anti-VEGF in the absence of SRNV.[66,67] Moreover, it has been suggested that VEGF plays a role in photoreceptor

differentiation, may contribute to photoreceptor survival, and may serve a role in maintaining retinal vascular homeostasis.[69]Therefore, it cannot be ruled out that blocking VEGF may cause an acceleration of apoptosis among ganglion cells and photoreceptors in IJFT IIA.[65] This remains to be tested.

The natural history of untreated SRNV in IJFT II is generally poor with 80% of eyes in a series of 26 eyes having a final VA of 20/200 or worse. Before the advent of VEGF antagonists, therapeutic options for SRNV associated with IJFT II included laser photocoagulation, PDT with or without IVTA, transpupillary thermotherapy (TTT), and surgical removal of the SRNV.

Matsumoto et al. [36] investigated the efficacy of intravitreal bevacizumab for the treatment of idiopathic macular telangiectasia (IMT). All the ten eyes of eight patients were treated with intravitreal bevacizumab (1.25 mg) injections at baseline. There were no changes in the mean visual acuity in either eye in any of the patients. These authors concluded that Type 2 IMT improved anatomically with bevacizumab treatment, despite a lack of improvement in vision, and bevacizumab effectively decreased vascular permeability and retinal edema in the short term. In the present investigation (dissertation), it was found that best corrected visual acuity improved for at least one line and showed significant reduction in macular thickness.

Gamulescu et al.[37] described functional and morphological long-term follow-up results in patients with IMT treated with intravitreal bevacizumab in three consecutive male patients with IMT who were treated with intravitreal bevacizumab injections. A single intravitreal bevacizumab injection resulted in a marked improvement in best

corrected visual acuity (BCVA) from 20/50 to 20/20 than the patient with type 1 (aneurysmal) IMT during the first 4 weeks. The patients with type 1 IMT with pronounced macular oedema on OCT may benefit from intravitreal bevacizumab injections, showing functional as well as morphological improvement, while patients with type 2 IMT with minimal cystic changes on OCT do not show functional improvement despite repeated injections. The present investigation also showed the same outcome after intravitreal bevacizumab injection.

Issa et al.[38] reported on the short-term effects of intravitreal bevacizumab in patients with type 2 IMT in a noncomparative, interventional, retrospective case series in seven eyes of six patients with type 2 IMT. Patients received 2 doses of intravitreal bevacizumab (1.5 mg) at a 4-week interval. A significant increase in mean VA of 8 ETDRS letters at 8 weeks was found. Visual acuity improved by more than 15 letters in one patient and by 10 to 15 letters in two patients and remained stable (to +10 letters) in another four patients, compared with baseline. In OCT imaging, mean retinal thickness showed a significant reduction in the foveal and in the parafoveal zones. The most pronounced effect (mean decrease, 22I¼m) was in the parafoveal temporal zone. No significant ocular or systemic side effects, were observed. These authors concluded, based on their short-term results, that inhibition of vascular endothelial growth factor (VEGF) by intravitreal bevacizumab is associated with a decrease in retinal thickness and a reduction in angiographic leakage in type 2 IMT; they also opined that intravitreal bevacizumab may improve VA in affected patients.

Kovach et al.[39] sought to determine whether inhibition of VEGF-A affects visual acuity, fluorescein angiographic, and OCT outcomes in patients with perifoveal

telangiectasia (PT) (also synonyms are macular telangiectasia, Type 2 juxtafoveolar retinal telangiectasis group 2A). In this retrospective review of patients with PT treated with intravitreal bevacizumab at the Bascom Palmer Eye Institute, in Florida (USA), best-corrected visual acuity, fluorescein angiography, and OCT measurements were performed on nine eyes of eight patients. Five eyes had proliferative PT, characterized by subretinal neovascularization involving the macula. After treatment (follow-up ranged from 4 to 27 months) the mean BCVA remained stable for the four eyes with nonproliferative PT, whereas in the five eyes with proliferative PT, BCVA was unchanged or improved after treatment. All eyes demonstrated decreased intraretinal leakage on fluorescein angiography after an injection of bevacizumab, and eyes with proliferative PT showed decreased growth and leakage of the subretinal neovascularization. The mean decrease in OCT central retinal thickness was less than 30 μ. These investigators were of the opinion that in nonproliferative PT, intravitreal bevacizumab decreases fluorescein angiographic leakage without any short-term effect on visual acuity or OCT appearance whereas in proliferative PT, intravitreal bevacizumab arrests the leakage and growth of subretinal neovascularization with the possibility of visual acuity improvement.

Moon et al.[40] assessed the potential visual benefit of intravitreal bevacizumab in a patient with idiopathic juxtafoveal retinal telangiectasis refractory to focal laser treatment; an intravitreal injection of bevacizumab (1.25 mg) was given. Within 1 week, visual acuity improved from 20/50 to 20/25 and OCT demonstrated complete resolution of macular edema without any adverse effect due to the injection. The macular edema recurred after 3 months, requiring a repeat injection of bevacizumab, with subsequent resolution of macular edema. These authors believed that an

intravitreal injection of bevacizumab may provide potential short-term visual benefit in patients with macular edema from idiopathic juxtafoveal retinal telangiectasis.

Idiopathic perifoveal telangiectasia (IPT) is a disorder characterized by focal parafoveal capillary dilatation of unknown cause. Photodynamic therapy (PDT) is reported to be efficacious in the treatment of subretinal neovascularization (SRN) associated with idiopathic juxtafoveal retinal telangiectasis; however, visual improvement is limited. Cakir et al.[41] described the clinical, angiographic, and OCT findings for a patient with IPT complicated by SRN who underwent treatment with PDT and bevacizumab. The patient underwent two sessions of PDT; however, metamorphopsia persisted, and visual acuity further decreased to 20/100 after the second PDT. Hence, after PDT, two injections of intravitreal bevacizumab (1.25 mg) were administered, following which visual acuity increased dramatically from 20/100 to 20/25. Four months after the second injection, there was no recurrence, and the patient was asymptomatic. These authors concluded that intravitreal bevacizumab treatment should be considered for patients with SRN associated with perifoveal telangiectasis, especially after insufficient therapeutic response to PDT.

Mandal et al.[42], in a nonrandomised interventional case series, evaluated the efficacy and safety of intravitreal bevacizumab in the treatment of SRN secondary to type 2A idiopathic juxtafoveal telangiectasia (IJT). Intravitreal bevacizumab (1.25 mg/0.05 ml) was injected as primary treatment into six eyes of six patients with SRN due to IJT. OCT and fundus fluorescein angiography (FFA) findings were examined before and after treatment (the patients were followed up for 3-6 months). Pre-injection BCVA measured from 20/400 to 20/120 (mean 20/200). After a mean follow-up of 4.2

months, post-injection BCVA measured from 20/200 to 20/50 (mean 20/100). At the last visit, BCVA was found to have improved by two or more lines in five eyes (83%) and remained the same in one eye (17%). The mean central foveal thickness improved from 263 mm (range, 165 to 393 mm) to 201 mm (range, 126 to 351 mm), representing an average reduction of 62 . Only one eye received more than one two bevacizumab injections and no significant complications were observed. These authors concluded that in their small series, intravitreal bevacizumab appeared to be a safe and effective treatment for SRN secondary to type 2A IJT.

Charbel et al.[43] evaluated, retrospectively, the effects of intravitreal bevacizumab for non-proliferative type 2 idiopathic macular telangiectasia (type 2 IMT) for a mean follow-up for 18 months. Six eyes of five patients with type 2 IMT received two doses of intravitreal bevacizumab (1.5mg) at a 4 week interval. The mean VA increased significantly, Parafoveal leakage in FFA and mean central retinal thickness was decreased in all eyes following treatment. A rebound effect was observed after 3-4 months, and at the last visit, retinal thickness was increased in selected retinal sectors including the fellow eye. Inhibition of vascular endothelial growth factor (VEGF) by intravitreal bevacizumab may lead to functional improvement as well as transient decrease in leakage and retinal thickness in patients with type 2 IMT.

# SUMMARY

The term “retinal telangiectasis” has been used to describe congenital vascular anomalies in retinal blood vessels, resulting in localised, abnormal vessel segments that exhibit leakage. Parafoveal telangiectasis (PFT) refers to telangiectasis that is localised to the capillaries of the parafoveal region, and is characterized by microaneurysmal and saccular dilation and capillary nonperfusion of the parafoveal capillaries. While many previous studies have documented clinical presentation and angiographic features, there have been fewer studies on imaging and management of the condition. The aims of the present investigation were to document the clinical features, fundus fluorescein angiographic (FFA) and optical coherence tomographic (OCT) findings, and to evaluate the efficacy of intravitreal bevacizumab in treatment, in patients presenting with type 2 parafoveal telangiectasis at a tertiary eye care facility in southern India.

Forty patients (80 eyes) were enrolled in this investigation, fourteen of whom were males and twenty-six of whom were females. The patients ranged in age from 40 to 70 years (mean [± standard deviation] age: 54.16 [±15] years). Fifteen patients were between 40 to 50 years of age, 14 patients were between 51 to 60 years of age and eleven patients were between 61 to 70 years of age.

All forty patients enrolled in the study suffered from bilateral parafoveal telangiectasis. These were divided into three groups:

a) Group 1, which included 15 patients with PFT who were under observation and treated conservatively; three were males and 12 were females, ranging in age

from 40 to 69 years (mean age 52.73 ± 9.05 years). Five patients had diabetes mellitus and hypertension as associated systemic illnesses, one patient had both DM and ischemic heart disease (IHD) and five patients had only DM. In the 30 eyes in this group of 15 patients, the mean vision at presentation was 0.264 ± 0.14 decimals, which was exactly the same as the mean vision (0.264 ± 0.14 decimals) after the three month follow -up period. In the 30 eyes in this group of 15 patients, the mean macular thickness at presentation (as measured by OCT) was 205.8 ± 56.32 μ, which was exactly the same as the mean macular thickness (205.8 ± 56.32 μ) after the three month follow- up period.

b) Group 2 included 14 patients with PFT complicated by cystoid macular oedema (CME), who were treated with intravitreal injections of bevacizumab (each 0.1ml) under aseptic conditions. Five of the fourteen patients were males and nine were females; the patients ranged in age from 40 to 65 years (mean age 53.5 ± 7.18 years). Four patients had DM and HTN as associated systemic illnesses, two patients had only HTN, two patients had only DM and one patient had bronchial asthma. In the 22 eyes which received an intravitreal injection of bevacizumab in this group of 14 patients, the mean vision at presentation was 0.19 (approximately 6/18) ± 0.1 decimals, which significantly improved to 0.256 (6/12) ± 0.096 following the injection of bevacizumab (`t' test (degree of freedom [d.f. =42] = 2.13; P= 0.04). In the 22 eyes which received an intravitreal injection of bevacizumab in this group of 14 patients, the mean macular thickness at presentation was 360.73 ± 43.22 μ, which showed a significant decrease to 253.45 ± 42.6 μ following the injection of bevacizumab (`t' test [d.f.=42]= 8.29; P < 0.0001). None of the eyes showed decreased visual

acuity after the intravitreal injection. Six eyes did not exhibit any change in visual acuity, but exhibited a significant reduction in macular thickness.

c) Group 3 included 11 patients with PFT and choroidal neovascular membrane (CNVM) who were treated with intravitreal injections of bevacizumab (each dose being 0.1 ml) under aseptic conditions; six of the 11 patients were males and five were females. The patients ranged in age from 45 to 70 years, with a mean age of 56.27 ± 8.50 years. Two patients had both DM and HTN, whereas one patient suffered from both DM and IHD. In the 18 eyes which received an intravitreal injection of bevacizumab in this group of 11 patients, the mean vision at presentation was 0.13 (approximately 6/60) ± 0.08 decimals, while the mean vision after the bevacizumab injection was 0.15 (6/36) ± 0.07; this difference was not statistically significant (`t' test [d.f. =54]= 0.99; P= 0.35). In the 18 eyes which received an intravitreal injection of bevacizumab in this group of 11 patients, the mean macular thickness at presentation was 469.17 ± 89.67 μ , which showed a significant decrease to 310.78 ± 85.61 μ following the injection of bevacizumab (`t' test [d.f.=54]= 6.76; P < 0.0001). One of the patients showed a decreased visual acuity from 6/60 to 3/60; however, the macular thickness in this eye showed a reduction from 417 μ to 313 μ. Eight eyes did not exhibit any changes in visual acuity, but exhibited significant reductions in macular thickness.

Comparisons were made between the three groups at several levels, namely:

**a) Age:** Differences between the three groups in the mean ages of the patients in the groups were analysed by one-way analysis of variance (ANOVA); the differences were not statistically significant (Fisher `f' value= 0.614; P= 0.546).

**b) Gender:** Males accounted for 20 % of patients in Group 1, 36 % of patients in Group 2 and 55 % of patients in group 3; these differences were not statistically significant (chi-square (d.f.=2)=2.7; P> 0.05).

**c) Pre-intervention mean visual acuity:** Differences between the groups in the pre-intervention mean visual acuity were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 11.018; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

**d) Post-intervention mean visual acuity:** Differences between the groups in the post-intervention mean visual acuity were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 10.329; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 3 and Group 2 vs. Group 3; however, the difference between the mean values in Group 1 and Group 2 were not statistically significant.

**e) Pre-intervention mean macular thickness:** Differences between the groups in the pre-intervention mean macular thickness were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 112.4; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

**f) Post-intervention mean macular thickness:** Differences between the groups in the post-intervention mean macular thickness were analysed by one-way ANOVA; the differences were statistically significant (Fisher `f' value= 18.753; P= 0.000). Post-hoc testing (Tukey's method) revealed statistically significant differences between the mean values in Group 1 vs. Group 2, Group 1 vs. Group 3 and Group 2 vs. Group 3.

Although Group 1 did not differ significantly from Groups 2 and 3 in the mean age of the patients, there was a definite preponderance of female patients in group 1, which was similar to the pattern in Group 2 but different from the pattern in Group 3 which showed a preponderance of male patients. However, in the 30 eyes of 15 patients who suffered from PFT alone, the pre-intervention and post-intervention mean visual acuities and the pre-intervention and post-intervention macular thickness values were all significantly better than the values noted in the 28 eyes of 14 patients with PFT and CME. Similarly, in the 30 eyes of 15 patients who suffered from PFT alone, the pre-intervention mean visual acuity and the pre-intervention and post-intervention macular thickness values were all significantly better than the values noted in the 22 eyes of 11 patients with PFT and CNVM; however, there was no significant difference between the post-intervention mean visual acuity in the eyes of these 2 groups of patients.

The results suggest that in patients with parafoveal telangiectasis alone, the mean visual acuity and mean macular thickness values are better than the same values in patients with parafoveal telangiectasis complicated by cystoid macular oedema or by choroidal neovascular membrane; there is neither short-term improvement nor deterioration in these parameters when the patients with parafoveal telangiectasis alone are managed conservatively. In patients with parafoveal telangiectasis complicated by

cystoid macular oedema there is definite short-term improvement in the visual acuity and in the macular thickness following intravitreal injection of bevacizumab. In patients suffering from parafoveal telangiectasis complicated by choroidal neovascular membrane, there is definite short-term improvement in the macular thickness, but not in visual acuity, following intravitreal injection of bevacizumab. Further studies of a longer duration and on a larger sample size of patients are required to confirm these initial results.

# CONCLUSION

The results suggest that in patients with parafoveal telangiectasis alone, the mean visual acuity and mean macular thickness values are better than the same values in patients with parafoveal telangiectasis complicated by cystoid macular oedema or by choroidal neovascular membrane; there is neither short-term improvement nor deterioration in these parameters when the patients with parafoveal telangiectasis alone are managed conservatively. In patients with parafoveal telangiectasis complicated by cystoid macular oedema there is definite short-term improvement in the visual acuity and in the macular thickness following intravitreal injection of bevacizumab. In patients suffering from parafoveal telangiectasis complicated by choroidal neovascular membrane, there is definite short-term improvement in the macular thickness , but not in visual acuity, following intravitreal injection of bevacizumab. Further studies of a longer duration and on a larger sample size of patients are required to confirm these initial results.

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# PROFORMA

Group: 1/2/3

Name:- Age: Sex:

MR No. :-

Address:-

Mobile No.:-

Systemic Diseases: -

Duration

Diabetes Mellitus-
Hypertension-
IHD-
Cerebrovascular accident-
Others -

SCREENING:-

BEST CORRECTED VISUAL ACUITY :

SLIT LAMP EXAMINATION:

90 D LENS EXAMINATION:

INDIRECT OPHTHALMOSCOPY:

OCT EXAMINATION:

F F A EXAMINATION:

INTERVENTION:

FOLLOW UP:

| Date | Visual acuity | OCT | Fundus | Remarks |
|---|---|---|---|---|
| | | | | |
| | | | | |

# Only PFT

| Name | Age | Sex | No | Sys illness | RE | LE | RE OCT- pre | LE OCT- pre | RE post | LE post | RE OCT post | LE OCT post |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Saghayamary.D | 45 | F | P747271 | DM,IHD | 6/60 | 6/60 | 253 | 253 | 6/60 | 6/60 | 253 | 253 |
| Pushpam.N | 59 | F | P743515 | None | 6/24P | 6/36P | 231 | 221 | 6/24P | 6/36P | 231 | 221 |
| Masilamani | 55 | F | P715262 | None | 6/36 | 6/18 | 156 | 196 | 6/36 | 6/18 | 156 | 196 |
| Maheswari.V | 47 | F | P697895 | DM,HTN | 6/60 | 6/60 | 138 | 133 | 6/60 | 6/60 | 138 | 133 |
| Kulandai Theresa.S | 42 | F | P695305 | DM | 6/12 | 6/9 | 202 | 195 | 6/12 | 6/9 | 202 | 195 |
| Hamdunisa.S | 62 | F | P609727 | DM,HTN | 6/60 | 6/36 | 214 | 165 | 6/60 | 6/36 | 214 | 165 |
| Chitra.J | 43 | F | P541652 | DM | 6/18 | 6/24 | 196 | 196 | 6/18 | 6/24 | 196 | 196 |
| Rajeswari.B | 50 | F | P730418 | DM,HTN | 6/36 | 6/12 | 185 | 185 | 6/36 | 6/12 | 185 | 185 |
| Malliha.V | 52 | F | P761085 | DM,HTN | 6/18 | 6/18 | 301 | 255 | 6/18 | 6/18 | 301 | 255 |
| Saravanan.S | 48 | M | P761091 | DM | 6/60 | 6/18 | 265 | 265 | 6/60 | 6/18 | 265 | 265 |
| Pappa.S | 60 | F | P768045 | DM | 6/24 | 6/24 | 322 | 281 | 6/24 | 6/24 | 322 | 281 |
| Padmavathy | 40 | F | P602027 | None | 6/18 | 6/12P | 256 | 256 | 6/18 | 6/12P | 256 | 256 |
| Sivaramakrishnan.R | 69 | M | P591981 | DM,HTN | 6/18 | 6/36 | 138 | 118 | 6/18 | 6/36 | 138 | 118 |
| Indrani.K | 67 | F | P608899 | DM | 6/18 | 6/60 | 158 | 101 | 6/18 | 6/60 | 158 | 101 |
| Velayutham.P | 52 | M | P542027 | None | 6/24 | 6/18 | 183 | 156 | 6/24 | 6/18 | 183 | 156 |

# PFT with CME

| Name | Age | Sex | No | Sys illness | RE | LE | Laterality | RE OCT- pre | LE OCT- pre | RE Avastin doses | LE Avastin doses | RE post | LE post | RE OCT post | LE OCT post |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Rajmohamad.M | 52 | M | P654296 | None | 5/60 | 6/60 | B/L | 353 | 398 | 1 | 1 | 6/60 | 6/36 | 233 | 308 |
| Selvarangam.V | 65 | M | P660465 | DM,HTN | 6/60 | 6/60 | B/L | 411 | 352 | 1 | 1 | 6/24 | 6/24 | 256 | 225 |
| Rajammal.G | 50 | F | P748932 | Asthma | 6/12 | 6/6 | U/L | 299 | 158 | 1 | 0 | 6/12 | 6/6 | 239 | 155 |
| Valarmathi.R | 47 | F | P663156 | None | 5/60 | 6/36 | B/L | 389 | 330 | 1 | 1 | 6/24 | 6/36 | 265 | 303 |
| Sundari.G | 40 | F | P645825 | None | 6/36 | 6/9 | U/L | 410 | 212 | 1 | 0 | 3/60 | 6/9 | 298 | 212 |
| Vijayalakshmi S.S | 61 | F | P708140 | HTN | 6/36 | 6/36 | B/L | 336 | 352 | 1 | 1 | 6/24 | 6/24 | 209 | 241 |
| Vasantha.A | 53 | F | P699494 | DM,HTN | 6/24 | 6/18p | U/L | 350 | 255 | 1 | 0 | 6/36 | 6/18 | 298 | 255 |
| Maruthambal.G | 56 | F | P746873 | DM,HTN | 6/60 | 6/18 | U/L | 411 | 209 | 1 | 0 | 6/18 | 6/18 | 340 | 209 |
| Haridoss.S | 54 | M | P792192 | HTN | 6/24 | 6/18 | B/L | 333 | 313 | 1 | 1 | 6/18 | 6/18 | 262 | 221 |
| Abdul malik.A | 60 | M | P795477 | DM | 6/24 | 6/60 | B/L | 412 | 356 | 1 | 1 | 6/24 | 6/24 | 310 | 201 |
| Manjula.S | 54 | F | P816527 | DM,HTN | 6/9 | 6/24 | U/L | 131 | 302 | 0 | 1 | 6/9 | 6/18 | 131 | 198 |
| Hassan.S | 50 | M | P819258 | None | 6/36 | 6/18 | B/L | 399 | 316 | 1 | 1 | 6/18 | 6/18 | 288 | 215 |
| Indra.K | 44 | F | P827018 | DM | 6/60 | 6/12 | U/L | 455 | 212 | 1 | 0 | 6/36 | 6/12 | 255 | 212 |
| Pushpavalli.A | 63 | F | P815556 | None | 6/24 | 6/24 | B/L | 344 | 315 | 1 | 1 | 6/24 | 6/18 | 199 | 212 |

# PFT with CNVM

| Name | Age | Sex | No | Sys illness | RE | LE | Laterality | RE OCT- pre | LE OCT- pre | RE Avastin doses | LE Avastin doses | RE post | LE post | RE OCT post | LE OCT post |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sathia kumari.A | 54 | F | P831789 | DM,HTN | 6/60 | 3/60 | B/L | 622 | 556 | 3 | 3 | 6/36 | 6/60 | 533 | 452 |
| Vadivel.A | 46 | M | P832732 | None | 6/36 | 6/60 | B/L | 451 | 402 | 3 | 3 | 6/36 | 6/60 | 301 | 302 |
| Kandhasamy.K.R | 65 | M | P840708 | DM,HTN | 6/60 | 6/60 | B/L | 453 | 356 | 3 | 3 | 6/60 | 6/60 | 256 | 266 |
| Noor mohamed.S | 63 | M | P775061 | None | 6/12 | 6/60 | U/L | 212 | 417 | 0 | 4 | 6/12 | 3/60 | 212 | 313 |
| Raghavendra rao.C.V | 61 | F | P825963 | DM,IHD | 3/60 | 6/18 | U/L | 356 | 229 | 3 | 0 | 6/60 | 6/18 | 212 | 240 |
| Selvaraj.N | 63 | M | P780216 | None | 6/36 | 6/24 | B/L | 400 | 521 | 3 | 4 | 6/36 | 6/24 | 317 | 301 |
| Bhaskar.R | 45 | M | P775756 | None | 6/24 | 3/60 | B/L | 399 | 454 | 3 | 3 | 6/24 | 6/60 | 244 | 256 |
| Elangovan.S | 53 | M | P761330 | None | 3/60 | 6/12 | U/L | 432 | 122 | 5 | 0 | 6/24 | 6/12 | 232 | 256 |
| Fathima rojali | 51 | F | P763922 | None | 6/60 | 6/18 | U/L | 441 | 130 | 3 | 0 | 6/60 | 6/18 | 201 | 131 |
| Rajalakshmi.B | 70 | F | P767084 | None | 6/60 | 6/24 | B/L | 621 | 398 | 3 | 5 | 6/36 | 6/24 | 400 | 288 |
| Nadiyammal.G | 48 | F | P841243 | None | 3/60 | 6/36 | B/L | 625 | 541 | 3 | 3 | 6/60 | 6/36 | 384 | 336 |