Diagnosis and Management of Diabetic Macular Edema Based On Optical Cohrence Tomography (Oct) Finding

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Abstract

Purpose: To assess the effect of central macular thickness (CMT) measured by optical coherence tomography (OCT) on the response to treatment in diabetic macular edema. Methods: This was Prospective Interventional Study conducted in patients visiting the outdoor unit of Department of Ophthalmology, Maharani Laxmi Bai Medical College Jhansi, during the period of 13 months from March 2017 to March 2018. This study included patients with age above 21 years, with diabetes, which is defined as a fasting plasma glucose of more than or equal to 126 mg/dl or a 2-hour post glucose load plasma glucose of more than or equal to 200 mg/dl or a random plasma glucose of more than or equal to 200 mg/dl in the presence of symptoms of hyperglycemia, and duration > 5 years, and those who were physically fit to undergo a dilated fundus examination and OCT evaluation. **Results:** In this study the mean baseline Best corrected visual acuity right eye affected with DME is 0.822+0.249 and in left eye is 0.863+0.210. The follow up mean visual acuity BCVA as per log MAR right eye affected with DME at 1 month is 0.574+0.230 and, at 6 months is 0.379+0.166. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in Best corrected visual acuity. The follow up mean visual acuity BCVA as per log MAR left eye affected with DME at 1 month is 0.544+0.185 and, at 6 months is 0.347+0.188. The p value was < 0.05, as calculated with the help of paired ttest, indicating that there was a significant difference in Best corrected visual acuity. In our study baseline mean central macular thickness in right eye affected with DME are $664.6+31.63 (\mu m)$ and, At 1 month the follow up mean central macular thickness (CMT) thickness right eve affected with DME are 467.4+51.41(um). After 3 months, CMT is 376.4 ± 55.63 (µm), After 6 months of follow up the CMT is 299.3 ± 47.20 (µm). We also measured the baseline mean central macular thickness in left eve affected with DME is 653.6+36.78 um. At 1 month the follow up mean central macular thickness (CMT) thickness left eve affected with DME was 464.3+61.44 um. After 3 months, CMT reduced to $370.4\pm73.25 \,\mu$ m, After 6 months of follow up the CMT reduced to 302.4 ± 47.03 μm . The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean central macular thickness. conclusion: Macular edema after treatment was assessed by improvement in mean CMT and mean BCVA, which correlated with each other. In this study intravitreal ranibizumab or bevacizumab treatment provides superior visual outcomes compared to conventional laser treatment. Intravitreal ranibizumab with laser has been shown to be more effective compare to focal / grid laser alone for the treatment of DME. Intravitreal triamcinolone with focal/grid photocoagulation has been shown to be more effective than laser alone. we concluded that triamcinolone intravitreal injection has not been found to be superior to focal grid photocoagulation orAnti-VEGF treatment. Keywords: Diabetic macular edema, optical coherence tomography, Anti VEGF, Laser, Steroids, NSAIDS, Vitrectomy.

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I. Introduction

The World Health Organization (WHO) estimates that more than 180 million people worldwide have diabetes, and this number is expected to increase and to rise to epidemic proportions within the next 20 years⁻ Diabetic retinopathy^[1], one of the most common complications of diabetes, remains a most common public health problem with significant socioeconomic implications, affecting approximately 50% of diabetic patients.Diabetic retinopathy is the leading cause of blindness in working-age populations of industrialized countries.

Diabetic macular edema (DME) is common cause of loss of vision in diabetes.DME mainly affects central vision from the early stages of retinopathy^[2], and it is the most frequent sight-threatening adverse outcome of diabetic retinopathy, particularly in diabetes mellitus type-2 patients.DME leads to distortion of visual images and may cause a marked decrease in vision even in the absence of severe retinopathy. Although

macular edema is a common and characteristic complication of diabetic retinopathy and shows apparent association with the systemic metabolic alterations of diabetes, it does not necessarily fit the regular course of diabetic retinopathy progression. DME may occur at moderate or severe stage of nonproliferative diabetic retinopathy, any stage of proliferative diabetic retinopathy and more advanced stages of the retinopathy^[3]. when fluid and protein deposits collect within the macula it causes swelling and thickening of macula, leading to diabetic macular edema.

Pathogenesis of macular edema:

Vascular endothelial growth factor (VEGF) play important role in the pathogenesis of DME. VEGF promotes angiogenesis and causes a breakdown in the BRB by damaging the tight junctions between retinal endothelial cells.Plasma protein such as albumin exert high onchotic pressure deposit in the neural interstitium, leading to interstitial edema. Other comorbidities such as chronic hyperglycemia, hypertension, and hyperlipidemia are also play important role in the development of DME^[4].

Incidence and Prevalence of DME:

The incidence and prevalence of diabetic macular edema have been reported in different epidemiologic studies with significant variations, depending on the type (type I or II), treatment (insulin, oral hypoglycaemic agents, or diet only), and the mean diabetes duration.

CLASSIFICATION OF MACULAR EDEMA:

Diabetic macular edema is mainly classified into two part either being clinically significant or not. Clinically significant macular edema (CSME) is seen when one of the fallowing occurs^[5].

Criteria for Diagnosis of Clinically Significant Macular Edema:

CSME was identified in the presence of any of the following three fundoscopic examination findings^[6].

- 1. Retinal thickening at or within 500 μ m or 1/3 the disc diameter of the fovea.
- 2. Hard exudates at or within 500 μ m of the fovea, with adjacent retinal thickening.
- 3. Retinal thickening greater than one disc diameter (1500μm) in size that is within one disc diameter from the fovea.

Diabetic macular edema may also be classified based on optical coherence tomography (OCT) measurements, specifically,central macular thickness, morphology of the retina, and the presence of macular traction.

OCT Classifiation of Diabetic Macular Edema:

- **Type 1:**Early diabetic macular edema.
- Type 2:Simple diabetic macular edema.
- Type 3:Cystoid diabetic macular edema: 3a, mild; 3b, intermediate; 3c, severe.
- Type 4:Serous macular detachment.

Diabetic macular edema is also classified into three part on the basis of Fluorescein angiography^[7].

Fluorescein Angiography Classifiation of DME:

Focal leakage: Localized areas of leakage from microaneurysms or dilated capillaries.

Difuse leakage: Diffuse leakage involving the entire circumference of the fovea.

Difuse cystoid leakage: Mainly diffuse leakage, but accumulation of the dye within the cystic areas of the macula during the late phase of the angiogram.

Diagnosis of macular edema:

The first steps in the diagnosis of DME is based on biomicroscopy, Indirect ophthalmoscopy and the ETDRS study group recommends that this diagnosis can be made on the basis of retinal thickening in macular area. Fundus Fluorescein angiography has been important method used for evaluating patients with DME, which assess the severity of the characteristics of macular edema, show the areas of retinal capillary leakage.^[8,9].

Optical coherence tomography (OCT):

The optical coherence tomography has been used in measuring retinal thickness for the evaluation of diabetic macular edema. In the future, OCT becomes the gold standard method of diagnosis and monitoring of diabetic macular edema. OCT in diabetic patients show different patterns of fluid accumulation in diabetic macular edema. Three patterns of structural changes like diffuse retinal thickening , cystoid macular edema and serous retinal detachment.

Management of diabetic macular edema:

To prevent the development and progression of DME, it is essential to maintain strict glycemic control, reduce blood lipid levels, and regulate systemic blood pressure. The American Diabetes Association recommends that HbA1c levels must be kept under 7%, blood pressure under 130/80 mmHg, and total lipids under 100 mg/dL Ocular treatments include retinal laser photocoagulation, intravitreal administration of various medication and vitreoretinal surgery when necessary.

Laser Therapy:

Focal and/or grid macular laser photocoagulation (MLP) used as the gold standard for treatment of DME. The mechanism of action of the grid laser destroys photoreceptors in the retina thereby decreasing the oxygen demand. The mechanism of action of focal laser targets specific leaking microaneurysms responsible for the macular edema. Laser is still the mainstay of treatment for DME^[10], Laser is the most established primary treatment for diabetic macular edema. Laser is attached to the ophthalmic slit lamp using a contact lens on the cornea and laser energy is delivered in a coaxial fashion. The laser energy is absorbed by vascular structures within the retina or by the retinal pigmented epithelium behind the sensory retina. Focal laser spots acts on microaneurysms in order to stop leakage of fluid. A grid pattern of laser spots also may acts on the retinal pigmented epithelium in areas of more diffuse edema. The retinal pigmented epithelium functions as a pump to remove fluid from the retina, and stimulation with laser spots is thought to stimulate pump function and thereby resolve retinal edema

Intravitreal Anti-VEGF Therapy:

Since VEGF contributes to the development of macular edema, attention has recently focused on intravitreal injections of anti-VEGF antibodies. Antibodies delivered by intravitreal injection bind to VEGF and thereby decrease downstream effects on vascular leakage.Three drugs are currently in use,Bevacizumab (Avastin),Ranizumab (lucentis) and Pegaptanib (Macugen). Pegaptanib sodium (Macugen) is an RNA aptamer. Pegaptanib sodium that selectively binds the VEGF-165 isoform, believed to be the main isomer responsible for DME. Aflibercept, the latest newcomer to the market, is a recombinant protein that also binds all VEGF isoforms and fragments.Side effects of intravitreal anti-VEGF injections are rare and include retinal detachments Similarly, endophthalmitis is a rare complication of intravitreal injection and is likely related to the injection itself rather than specific pharmaceutical characteristics^[11].

Carbonic anhydrase inhibitors:

These alter ionic transport systems in the retinal pigment epithelial cells, moving fluid away from the intracellular space.Carbonic anhydrase inhibitors (CAI) have direct effects both on retinal and retinal pigment epithelial cell function by inducing an acidification of the subretinal space, a decrease of the standing potential as well as an increase in retinal adhesiveness.The acidification of the subretinal space is responsible for the increase in fluid resorption from the retina through the RPE cell into the choroid.

Pharmacological vitreolysis agents:

Enzymatic vitreolysis agents such as chondroitinase, dispase, hyaluronidase, plasmin and microplasmin can promote posterior vitreous detachment to relieve traction on the retina.

Intravitreal Corticosteroids:

The effects of glucocorticoids depend on their anti-inflammatory and anti-VEGF effects. Triamcinolone acetonide (TA) has been used for various ocular inflammatory disorders, including DME; several studies have considerable improvements in DME with TA alone or combined with anti-VEGF agents^[12-15]. Elevated intraocular pressure (IOP) and cataract formation are the main complications of intravitreal steroid injection. Fluocinolone acetonide and dexamethasone implants are newer steroids options found to be effective in various studies.

Intravitreal NSAID Therapy:

Intravitreal nonsteroidal anti-inflammatory drugs (NSAIDs) have also shown effective results in treating DME. NSAIDs block prostaglandin synthesis and reduce inflammation, which may have a role in diabetic macular edema. Certain nonselective NSAIDs such as diclofenac also inhibit lipooxygenase synthesis mimicking the method of action of steroids, which may explain their similar efficacies. The NSAIDs such as diclofenac donot increase IOP while steroid such as triamcinolone increases IOP. Hence intravitreal NSAIDs possibly be as effective as steroid therapy, while avoiding the associated complications such as IOP elevation.

Vitrectomy: The removal of the vitreous is believed to reduce vascular permeability and relieve traction on the retina.vitrectomy combined with IVTA and laser for eyes with DME refractory to prior anti-VEGF therapy has good result. DME refractory to previous IVTA therapy has also shown effective response when vitrectomy was performed before IVTA was repeated, with significant improvements in BCVA and resolution of DME. **Method and material:**

This Prospective interventional study was carried out in Department of Ophthalmology, MLB Medical College over a period of 13 months. Patients were included in the study under the following inclusion and exclusion criteria:

Inclusion criteria:

- 1. All patients of age > 21 years, with a confirmed diagnosis of DM, and NPDR or PDR with DME diagnosed with OCT or clinically and/or angiographically confirmed Diabetic Macular Edema.
- 2. All patients with diabetes, which is defind as a fasting plasma glucose of more than or equal to 126 mg/dl or a 2 hour post glucose load plasma glucose of more than or equal to 200 mg/dl or a random plasma glucose of more than or equal to 200 mg/dl in the presence of symptoms of hyperglycemia, and duration > 5 year and the patients were under medical treatment by an experienced physician/ endocrinologist
- 3. Those who were physically fit to undergo a dilated fundus examination and OCT evaluation.

EXCLUSION CRITERIA:

- 1. Pregnancy
- 2. Dense media haze interfering with acquisition of good OCT image.
- 3. Any other ocular pathology which can contribute to reduced visual acuity macular edema due to associated condition other than diabetic retinopathy like central retinal vein occlusion etc, and those with OCT scans of poor quality will be excluded.
- 4. Recent ocular surgery (<6 months).
- 5. All other macular pathologies.
- 6. Patients diagnosed to have glaucoma.
- 7. Patients with very high refractive error

Consent: Patients satisfying the inclusion criteria shall sign an informed consent before participating in the study.

Pre-Treatment Work Up: After selecting the patient, a detailed clinical record was prepared including age, sex, address, occupation, family history, duration of the diabetes and history of previous treatments. Detailed ocular examination was carried out by using Snellen's chart for BCVA, S/L biomicroscopy by 90D for fundus examination, tonometry by Schiotz tonometer for IOP assessment,Ophthalmoscopic examination(direct/indirect) and fundus photography was done under full mydriasis and FFA if clinically required.OCT is likely to become the gold standard method of diagnosis and monitoring of patients with macular edema. Each eye was dilated with tropic amide 1% before the images were recorded, and scans were performed with a minimum pupillary diameter of 5 mm.

Method:

• All patients were selected under inclusion and exclusion criteria and the above mention prospective Interventional study would be carried on 100 patients having diabetes for > 5 years suffering from diabetic macular edema in 13 months duration.

Statistical analysis: Descriptive statistics included the mean and standard deviation for numerical variables, and the percentage of different categories for categorical variables.

• Group comparisons will be done by the paired t-test test for categorical variables. A logistic regression model was performed and the adjusted OR (95% CI) will be obtained for the risk factors which had been shown to be significant in the univariate analysis. A probability (P) of less than 0.05 was considered significant.

II. Results					
TABLE 1: GENDER DISTRIBUTION IN STUDY					
Sex Number of Patients Percentage (%)					
Males	64	64%			
Females	36	36%			

Table 1 show that out of 100 patients, 64 (64%) were males and 36 (36%) were females. Our study showed male predominance.

TABLE 2. AGE DISTRIBUTION IN STUDT GROUT				
Age group (years)	Number of	Percentage		
	patients	(%)		
21-30	03	3.00%		
31-40	11	11.00%		
41-50	20	20.00%		
51-60	26	26.00%		
61-70	40	40.00%		

TABLE 2: AGE DISTRIBUTION IN STUDY GROUP

Number of patients in age group 21-30 year is 3(3%), and 31-40 year is 11(11%), 41-50 year is 20(20%), 51-60 year is 26(26%), and 61-70 year is 40(40%). In our study majority (40%) of patient were found to be > 60 year.

TABLE 3: DURATION OF DIABETES					
Duration of DM	NO	%	Mean	SD	
(in year)					
5-10	13	13%			
11-15	14	14%	15.24	+3.095	
>15	60	60%			

The Mean Duration of Diabetes in years was 15.24+3.095. Number of patient in age group 5-10 year is 13(13%), 11-15 year is 14(14%), and >15 year is 60(60%). In our study majority (60%) of patient were found to be > 15 year.

TABLE 4: MEAN BASELINE BEST CORRECTED VISUAL ACUITY (BCVA) RIGHT EYE AFEECTED WITH DME

AFFECTED WI	I H DME
BCVA	Number
Mean baseline BCVA	0.822
SD	+0.249

The mean baseline visual acuity BCVA as per logMAR right eye affected with DME is 0.822+0.249.

Mean baseline BCVA right eye affected with		Follow up at 1 month		p value		
DME						
	Mean	SD	Mean	SD		
	0.822	+0.249	0.574	+0.230	< 0.05	

TABLE 5: FOLLOW UP BCVA RIGHT EYE AFFECTED WITH DME AT 1 MONTH

Mean follow up BCVA right eye at 1 month was 0.574+0.230. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean right eye BCVA comparing to baseline.

TABLE 6: FOLLOW UP BCVA RIGHT EYE AFFECTED WITH DME AT 6 MONTHS

Mean baseline B affected wi	ean baseline BCVA right eye Follow affected with DME		at 6 months	p value
Mean	SD	Mean	SD	
0.822	+0.249	0.379	+0.166	< 0.05

Mean follow up BCVA right eye at 6 months was 0.379+0.166. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean right eye BCVA comparing to baseline.

TABLE 7: MEAN BASELINE BEST CORRECTED VISUAL ACUITY (BCVA) LEFT EYE AFFECTED WITH DME

AFFECTED WITH DME			
BCVA	Number		
Mean baseline BCVA	0.863		
SD	+0.210		

The mean baseline visual acuity BCVA as per logMAR left eye affected with DME is 0.863+0.210

TABLE 8: FOLLOW UP BCVA LEFT EYE AFFECTED WITH DME AT 1 MONTH

Mean baseline BCVA left eye affected with DME		Follow up at 1 month		p value
Mean	SD	Mean	SD	
0.863	+0.210	0.544	+0.185	< 0.05

Mean follow up BCVA left eye at 1 month was 0.544+0.185. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean left eye BCVA comparing to baseline.

TABLE 9: FOLLOW UP BCVA LEFT EYE AFFECTED WITH DME AT 6 MONTHS

Mean baseline BCVA left eye affected with DME		Follow up at 6 months		p value
Mean	SD	Mean	SD	
0.863	+0.210	0.347	+0.188	< 0.05

Mean follow up BCVA left eye at 6 months was 0.347+0.188. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean left eye BCVA comparing to baseline.

TABLE 10: MEAN BASELINE CENTRAL MACULAR THICKNESS (µm) IN RIGHT EYE AFFECTED WITH DME

CMT	Number
Mean baseline CMT	664.6
SD	+31.63

The mean baseline central macular thickness (μ m) right eye affected with DME is 664.6+31.63.

TABLE 11: FOLLOW UP CENTRAL MACULAR THICKNESS (µm) RIGHT EYE AFFECTED WITH DME AT 1 MONTH

Mean baseline CMT right eye affected with DME		Follow up at 1 month		p value
Mean	SD	Mean	SD	
664.6	+31.63	467.4	+51.41	< 0.05

Mean follow up central macular thickness (μ m) right eye affected with DME at 1 month was 467.4+51.41. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean right eye central macular thickness comparing to baseline.

TABLE 12: FOLLOW UP CENTRAL MACULAR THICKNESS (µm) RIGHT EYE AFFECTED WITH DME AT 3 MONTHS

Mean baseline CMT right eye affected with DME		Follow up at 3 months		p value
Mean	SD	Mean	SD	
664.6	+31.63	376.4	+55.63	<0.05

Mean follow up central macular thickness (μ m) right eye affected with DME at 3 months was 376.4+55.63. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean right eye central macular thickness comparing to baseline.

TABLE 13: FOLLOW UP CMT (µm) RIGHT EYE AFFECTED WITH DME AT 6 MONTHS

Mean baseline CMT with D	CMT right eye affected Follow up at 6 months with DME		p value	
Mean	SD	Mean	SD	
664.6	+31.63	299.3	+47.20	<0.05

Mean follow up central macular thickness (μ m) right eye affected with DME at 6 months was 299.3+47.20. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean right eye central macular thickness comparing to baseline.

TABLE 14: MEAN BASELINE CENTRAL MACULAR THICKNESS (µm) LEFT EYE AFFECTED WITH DME

CMT	Number	
Mean baseline CMT	653.6	
SD	+36.78	

The mean baseline central macular thickness (μ m) left eye affected with DME is 653.6+36.78.

TABLE 15: FOLLOW UP CMT) (µm) LEFT EYE AFFECTED WITH DME AT 1 MONTH

Mean baseline affected wi	CMT left eye ith DME	Follow up at 1 month		p value
Mean	SD	Mean	SD	
653.6	+36.78	464.3	+61.44	< 0.05

Mean follow up central macular thickness (μ m) left eye affected with DME at 1 month was 464.3+61.44. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean left eye central macular thickness comparing to baseline.

TABLE 16: FOLLOW UP CMT (µm) LEFT EYE AFFECTED WITH DME AT 3 MONTHS

Mean baseline CMT I DM	eft eye affected with E	Follow up a	at 3 months	p value
Mean	SD	Mean	SD	
653.6	+36.78	370.4	+73.25	<0.05

Mean follow up central macular thickness (μ m) left eye affected with DME at 3 months was 370.4+73.25. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean left eye central macular thickness comparing to baseline.

TABLE 17: FOLLOW UP CMT (µm) LEFT EYE AFFECTED WITH DME AT 6 MONTHS

Mean baseline affected wi	CMT left eye ith DME	Follow up at 6 months		p value
Mean	SD	Mean	SD	
653.6	+36.78	302.4	+47.03	<0.05

Mean follow up central macular thickness (μ m) left eye affected with DME at 6 months was 302.4+47.03. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean left eye central macular thickness comparing to baseline.

Treatment	Number of patients	%
Anti -VEGF	35	35.00%
Laser	20	20.00%
NSAIDS	17	17.00%
Steroid	06	6.00%
Vitrectomy	10	10.00%
Anti-VEGF +Laser	07	7.00%
Laser+ Steroid	02	2.00%
Laser+ NSAIDS	03	3.00%

TABLE 18: TREATMENT DISTRIBUTION IN STUDY

In our study out of 100 patient, 35(35%) patient taking anti-VEGF and 20(20%) patient taking laser and 17(17%) patient taking NSAIDS and 6(6%) patient taking steroid and 10(10%) patient taking vitrectomy and 7(7%) taking Anti-VEGF+ Laser and 2(2%) taking Laser+ steroid and 3(3%) patient taking Laser+ NSAIDS.

III. Discussion

Our study was conducted at MLB Medical College in the year March 2017-March 2018 which included 100 patients who fulfilled the inclusion criteria. The present prospective interventional study was designed to compare the central macular thickness before and after treatment who have established diabetic macular edema. The study was carried out during the period of 13 months from March 2017 to March 2019 on patients coming to the out patient department of Ophthalmology in M.L.B. Medical College, Jhansi.

A total number of 100 patients with 200 eyes were enrolled for the study which were followed up and assessed over 13 months. The results of this study are summarized as -

- 1. The study included 64 males (64%) and 36 females (36%) that is there was male preponderance.
- **2.** Majority of the patients were > 60 years of age.
- 3. The total mean duration of diabetes was 15.24+3.095 years
- **4.** In this study the mean baseline Best corrected visual acuity right eye affected with DME is 0.822+0.249 and in left eye is 0.863+0.210
- 5. The follow up mean visual acuity BCVA as per log MAR right eye affected with DME at 1 month is 0.574+0.230 and, at 6 months is 0.379+0.166. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in Best corrected visual acuity.
- 6. The follow up mean visual acuity BCVA as per log MAR left eye affected with DME at 1 month is 0.544+0.185 and, at 6 months is 0.347+0.188. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in Best corrected visual acuity
- 7. The CMT assessed by SD-OCT for the comparison of mean central macular thickness and analysed the CMT thickness after treatment in 1 month,3 months and 6 months follow up, the difference of mean CMT comes out to be statistically significant in mean central macular thickness (μm).

In our study baseline mean central macular thickness in right eye affected with DME are 664.6+31.63 (µm) and, At 1 month the follow up mean central macular thickness (CMT) thickness right eye affected with DME are 467.4+51.41(µm). After 3 months, CMT is 376.4 ± 55.63 (µm), After 6 months of follow up the CMT is 299.3 ± 47.20 (µm).

We also measured the baseline mean central macular thickness in left eye affected with DME is $653.6+36.78 \mu m$, At 1 month the follow up mean central macular thickness (CMT) thickness left eye affected with DME was $464.3+61.44 \mu m$. After 3 months, CMT reduced to $370.4\pm73.25 \mu m$, After 6 months of follow up the CMT reduced to $302.4\pm47.03 \mu m$. The p value was <0.05, as calculated with the help of paired t-test, indicating that there was a significant difference in mean central macular thickness.

IV. Conclusion

Macular edema after treatment was assessed by improvement in mean CMT and mean BCVA, which correlated with each other. In this study intravitreal ranibizumab or bevacizumab treatment provides superior visual outcomes compared to conventional laser treatment. Intravitreal ranibizumab with laser has been shown to be more effective compare to focal / grid laser alone for the treatment of DME. Intravitreal triamcinolone with focal/grid photocoagulation has been shown to be more effective than laser alone. we also concluded that triamcinolone intravitreal injection has not been found to be superior to focal / grid photocoagulation or Anti-VEGF treatment.

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