Evaluation Of Choroidal Thickness Before and After Intravitreal Injection Of Anti Vascular endothelial growth factors for Treatment Of Diabetic Macular Edema using optical coherence tomography

Thesis

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List of Abbreviations

AMD: Age-related Macular Degeneration

CI: Confidence Interval.

CME: Cystoid Macular Edema.

DD: Disc Diameter.

DME: Diabetic Macular Edema.

DORL: Disruption of Outer Retinal Layers.

DR: Diabetic Retinopathy.

DRIL: Disorganization of Retinal Inner Layers.

GABA: γ-aminobutyric acid.

GCC: Ganglion Cell Layer.

IRMAs: Intraretinal Microvascular Abnormalities.

NIR: Near-infrared Reflectance

NPDR: Non-Proliferative Diabetic Retinopathy.

NVD: Neovascularization of the Disc.

NVE: Neovascularization Elsewhere.

OCT: Optical Coherence Tomography.

PDR: Proliferative Diabetic Retinopathy.

PRN: Pro Re Nata

PRP: Panretianl Photocoagulation.

RNFL: Retinal Nerve Fiber Layer.

RPE: Retinal Pigment Epithelium

SD: Standard Deviation.

SD-OCT: Spectral Domain – Optical Coherence Tomography.

SD-OCT: Swept Domain-Optical Coherence Tomography

SPSS: Statistical Package for Social Sciences.

SS-OCT: Swept Source-Optical Coherence Tomography

TD-OCT: Time Domain-Optical Coherence Tomography

VEGF: Vascular Endothelial Growth Factor.

INTRODUCTION

Diabetic macular edema (DME) is a critical eye condition that is considered a common cause of loss of vision in diabetics affected by diabetic retinopathy (DR) [1,2], with macular edema and proliferative retinopathy being the leading causes of visual impairment [2,3]. Anti-vascular endothelial growth factor (anti-VEGF) drugs are widely used in the treatment of diabetic macular edema (DME); this is supported by an extensive body of literature, demonstrating substantial improvements in visual and anatomic outcomes [4-10].

The choroid is a vascularized tissue that plays a vital role in providing metabolic support to the outer retina, including the photoreceptors and prelaminar portion of the optic nerve head [11,12]. Abnormal choroidal blood volume and compromised flow may result in the dysfunction and necrosis of the photoreceptor cells [13]. Choroidal vasculopathy, such as obstruction of the choriocapillaris, vascular degeneration, choroidal aneurysms, and neovascularization, may be involved in the pathogenesis of DR [13-15].

Enhanced depth imaging using spectral domain optical coherence tomography (SD-OCT) enables improved visualization of the choroid and assessment of choroidal thickness [16,17]. Results of clinical studies have indicated that choroidal thickness may be related to DR severity, and that the presence of DME is associated with a significant decrease in choroidal thickness [16]. In patients with neovascular age related macular degeneration (AMD), anti-VEGF drugs have been shown to induce significant thinning in choroidal thickness, which may lead to unknown long-term consequences or complications [18].

Currently, intravitreal anti-VEGF injection is the most common treatment for DME. Anti-VEGF injections improve visual acuity and several studies have demonstrated an association between anti-VEGF with decreased central retinal thickness [18,19]. However, there is currently a lack of studies investigating the effect of anti-VEGF injections on the choroid in patients with diabetes. The purpose of the present study is to evaluate the effect of intravitreal anti-VEGF injections on choroidal thickness using SD-OCT in patients treated for DME.

Aim of The Work

The purpose of the present study is to evaluate the effect of intravitreal anti-VEGF injections on choroidal thickness using SD-OCT in patients treated for DME.

Review of Literature

1 – Anatomy of the Retina

The eye is composed of essentially three layers (Figure 1):

- An outer protective coat; the cornea & the sclera.
- A middle vascular part; the choroid, ciliary body, and uveal tract.
- An inner neural part; the retina.

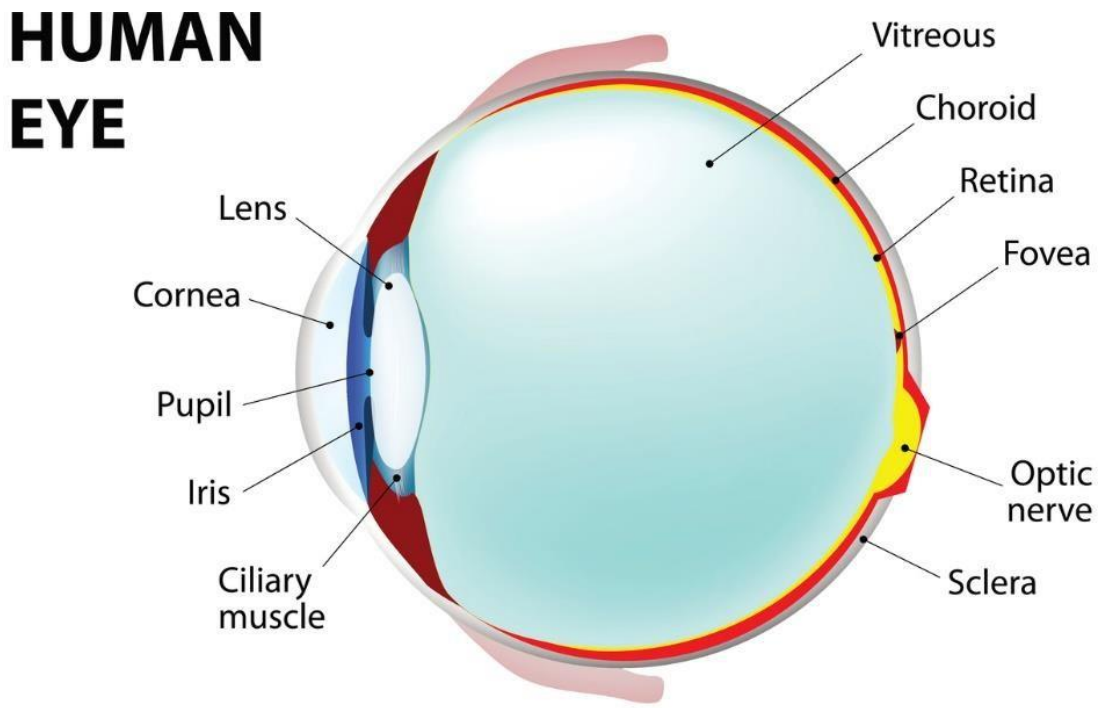

Figure 1. Layers of the human eye [2, 3].

There are ten layers from the outside to the inside of the eyeball (Figure 2):

1 - Cells of the retinal pigment epithelium (RPE);

2 - The outer and inner segments of the photoreceptors;

3 - The outer limiting membrane, where the Müller glial cells connect with the inner segments of the photoreceptors and where the photoreceptors have connections between them through adherent and tight junctions;

4 - The outer nuclear layer which houses the nuclei of the photoreceptors;

5 - The external plexiform layer which is formed by the synapses between the bipolar cells and the photoreceptors and with the horizontal cells;

6 - The inner nuclear layer which is the layer of the nuclei of the horizontal, bipolar, amacrine cells and the glial cells of Müller;

7 - The internal plexiform layer which is made up of the dendrites

of the ganglion cells and the axons of the bipolar cells;

8 - The ganglion cell layer (GCC);

9 - The layer of nerve fibers, which is made up of the axons of ganglion cells, surrounded by glial extensions that form the optic nerve which exits through the optic disc to be connected to the brain;

10 - The internal limiting membrane which is a membranous expansion composed of the internal feet of the Müller glial cells and their basal membrane [20, 21].

Light travels through the retinal layers to be blocked by the RPE where it stimulates the photoreceptors initiating the phototransduction process which in turn stimulates the bipolar cells. The bipolar cells undergo various signal modification procedures through the amacrine, horizontal, and glial cells. They eventually stimulate the ganglion cells whose axons go on to form the optic nerve [20].

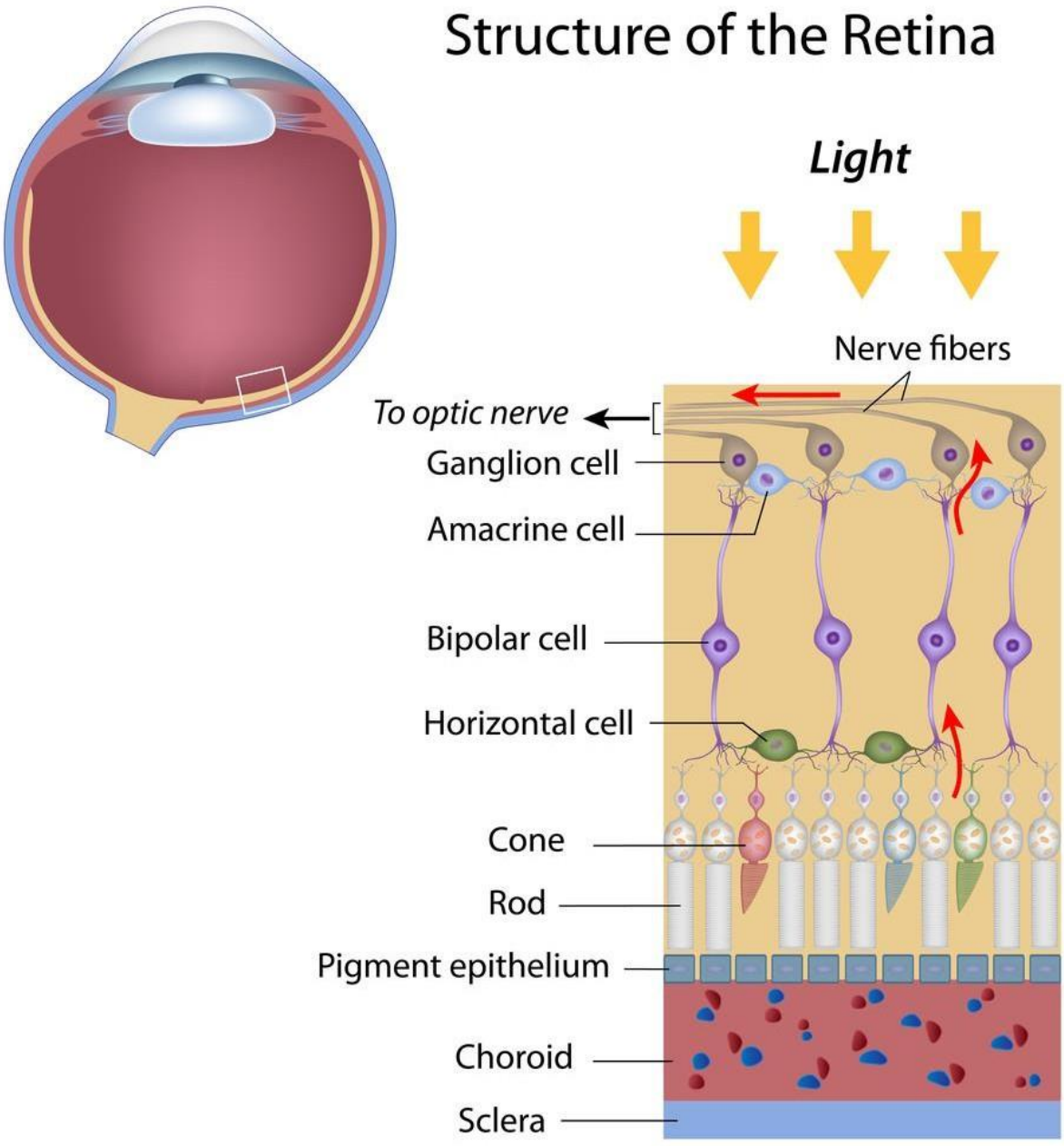

Figure 2. The microscopic structure of the retina [16].

The retina lines the back of the eye. It is a thin transparent tunic, less than 500 µm in thickness. The transparency reveals the vascularization of the pigments of the RPE layer that underlies it, as well as the vascularization of the choroid that further underlies the RPE layer.

The head of the optic nerve is visible in the form of a pinkish disc, lighter than that of the retina, around which emerge retinal arteries and veins, called the optic papilla. The macula is an area of the posterior retina whose center is avascular. It houses the fovea, which is the area of the highest visual acuity, being the area with the highest concentration for cones. The macula is discernible on fundus examination by its orange coloration due to the presence of xanthophyll pigments. It is located temporally to the optic disc [22] (Figure 3).

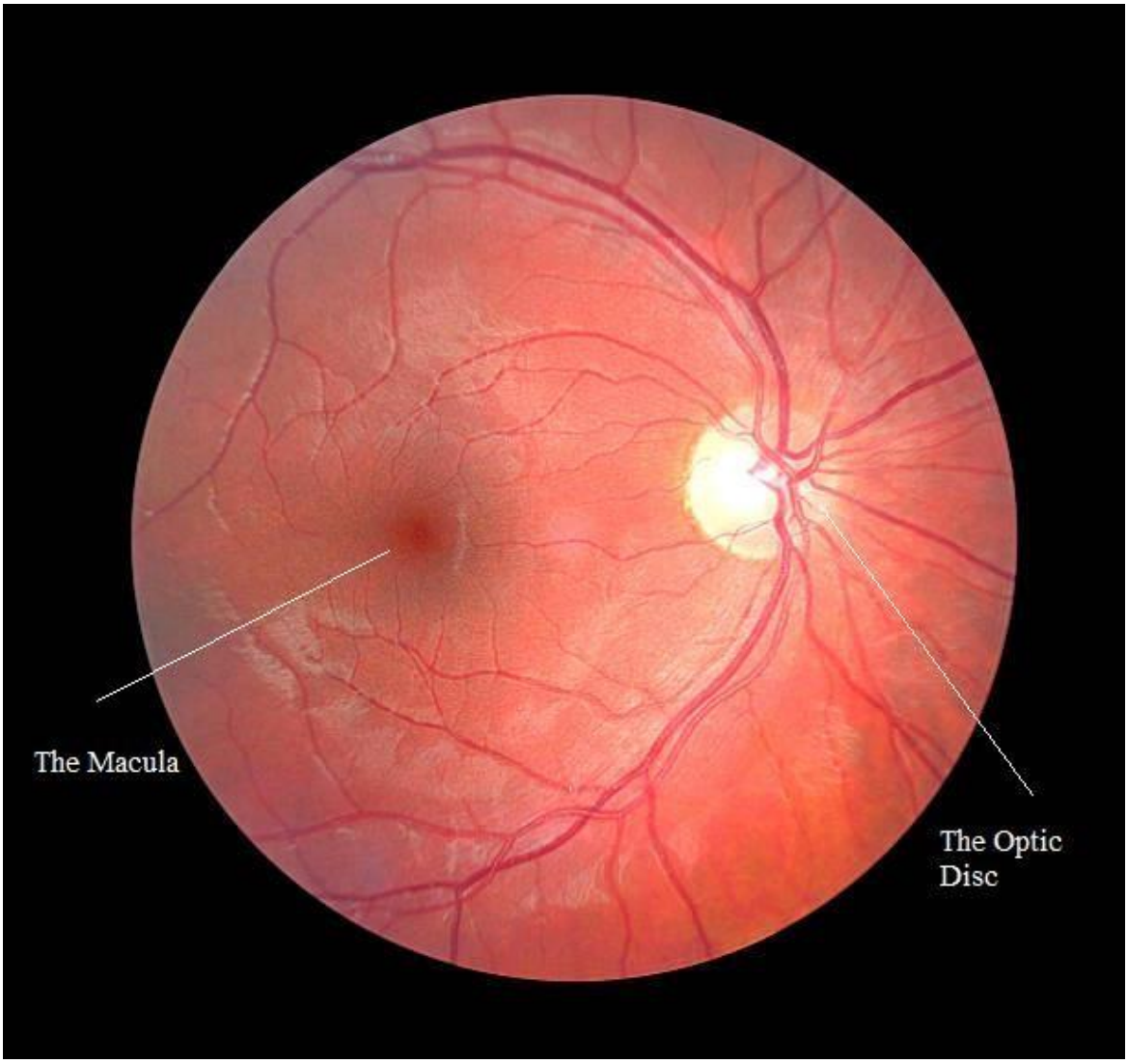

Figure 3. The macroscopic structure of the retina [22].

The retina has two different systems of vascularization, which are normally not connected with each other:

1 - The retinal capillary network, which provides the direct vascularization of the inner retinal layers.

2 - The choroidal network, which provides the vascularization of the outer part of the retina indirectly through diffusion, since there are no capillaries in the outer retina.

The central retinal artery derives primarily from the internal carotid, and more specifically the ophthalmic artery. It follows an intraneural course, to emerge at the optic disc where it divides into its four terminal branches: temporal and nasal, superior and inferior (Figure 3). The terminal arteries divide into collateral arteries, which divide into terminal arteries which also include collaterals, and so on dichotomously until they form a mesh network covering, for each of the arteries, a quadrant of the inner retina. The retinal capillaries come from these collateral vessels and are organized into superficial, intermediate and deep plexuses (Figure 4).

2 – Anatomy of the Choroid

The choroidal vascular system brings nutrients and oxygen to the outer retina, especially the photoreceptors, since the outer retina does not have a nourishing capillary network. The choroidal vasculature comes from branches of the ophthalmic artery which is a branch of the internal carotid artery. The choroid is a tissue 300 to 500 µm thick in humans, bounded by Bruch's membrane in front, and adherent to the sclera behind.

The choroid is made up of pigmented cells (melanocytes), mast cells, microglial cells and vessels. The vessels of the choriocapillaris are formed of a layer of endothelial cells with tight junctions comprising large diaphragmatic fenestrations (60 to 90 nm), with the size of the opening being dependent on the vascular endothelial growth factor (VEGF) [23], thus regulating the passage of proteins and macromolecules.

This protein gradient between the retina and the choroid is essential for keeping the retina attached, and for a state of transparency necessary for the transmission of photons [24]. The choroid is richly innervated by parasympathetic, sympathetic, and

trigeminal sensory nerve fibers that regulate choroidal blood flow (Figure 5) [25].

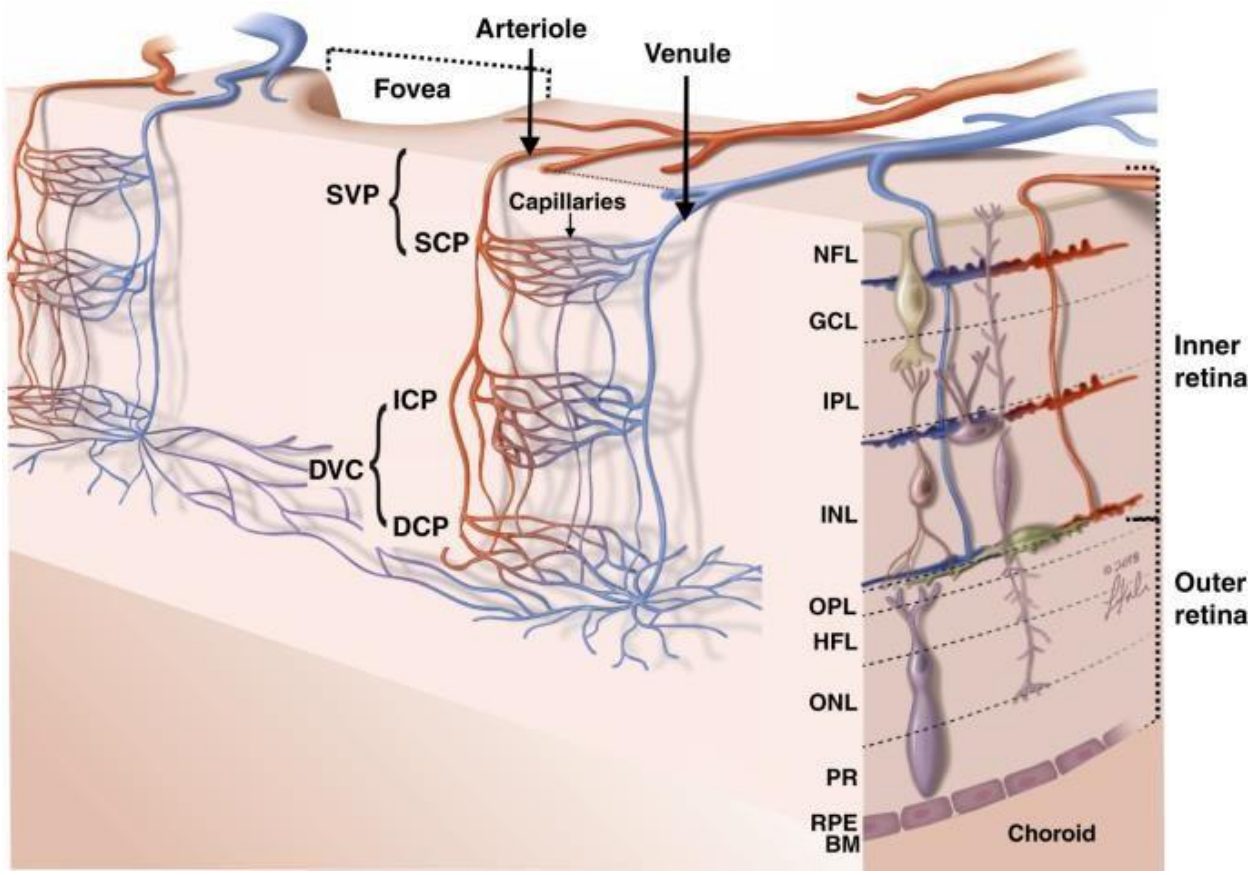

Figure 4. The vascular system of the retina.

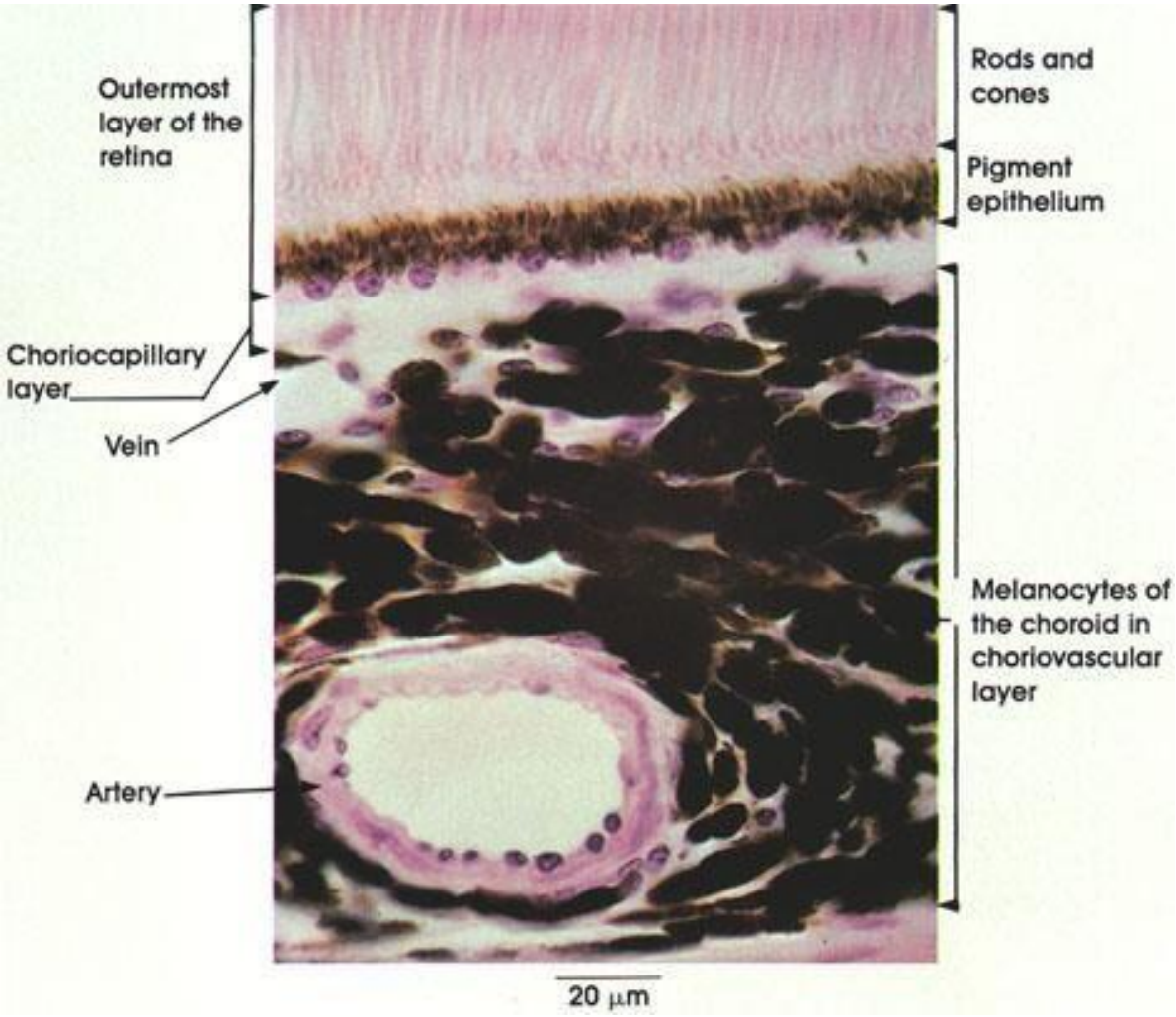

Figure 5. The anatomy of the choroid.

Being one of the most highly vascularized tissues of the body, the choroid's main function is to supply oxygen and nutrients to the outer retina. Other likely functions include thermoregulation via heat dissipation and modulation of intraocular pressure (IOP) via vasomotor control of blood flow. This last function is mediated by the fact that the choroid plays an important role in the drainage of the aqueous humor from the anterior chamber, via the uveoscleral pathway. This pathway is responsible for approximately 35% of the aqueous drainage [26].

Histologically, the choroid has been divided into 5 layers:

1 – The bruch's membrane.

2 - The choroiocapillaris.

3, 4 - The two vascular layers (Haller's and Sattler's).

5 - The suprachoroidea [27].

The choroid is approximately 200 μm thick at birth and decreases to about 80 μm by age 90 [28]. The choriocapillaris, the highly anastomosed network of capillaries, is about 10 μm thick at the fovea, where there is the greatest density of capillaries, thinning to about 7 μm in the periphery [29].

The innermost choroidal layer is Bruch's membrane which is a 5-layered structure consisting of (from outer to inner):

1 - Basement membrane of the choroiocapillaris.

2 - Outer collagenous zone.

3 - Elastic layer.

4 - Inner collagenous zone.

5 - Basement membrane of the retinal pigment epithelium [30].

The vascular region of the choroid consists of:

1 - The outer Haller's layer: Large blood vessels.

2 - The inner Sattler's layer: Medium and small arteries and arterioles that feed the capillary network, and veins.

3 – Diabetic Retinopathy

Diabetic retinopathy (DR) is a microvascular disorder occurring due to the long-term effects of diabetes mellitus. Diabetic retinopathy may lead to vision-threatening damage to the retina, eventually causing blindness. It affects people with diagnosed or undiagnosed diabetes mellitus. The propensity to develop diabetic retinopathy is directly proportional to the patient's age and duration of diabetes, as well as poor glycemic control and fluctuating blood pressure levels [31].

Histologically diabetic retinopathy presents with microangiopathy associated with vascular changes like focal capillary closure, dilatation of venules, hyalinization of arterioles, and capillary changes like basement membrane thickening, pericyte degeneration, and focal outpouchings (microaneurysms). Capillary closure leads to cotton wool spots and ischemic areas, leading to the development of IRMA (intraretinal microvascular abnormalities) and neovascularization, followed by fibrosis and contracture of the retina [32, 33].

Optical Coherence Tomography (OCT) biomarkers of prognosis

of diabetic retinopathy include refractile bodies, disorganization of inner layers of the retina (DRIL), disruption of outer layers of the retina (DORL), choroidal thickness, epiretinal membrane, vitreomacular adhesions, subretinal fluid, macular thickness, and integrity of ellipsoid zone [34, 35].

It is essential to consider the choroidal thickness status in diabetic retinopathy patients given that a structurally and functionally normal choroidal vasculature is essential for function of the retina. Meaning that abnormal choroidal blood volume and/or compromised choroidal blood flow can result in photoreceptor dysfunction and death [36].

Literature showed a significant decrease in choroidal thickness in patients with diabetic macular edema (DME) as well as patients with treated proliferative diabetic retinopathy (PDR), compared with normal subjects [37]. The thinner choroid may indicate an overall reduction of choroidal blood flow in patients with DME, therefore, it is likely that the decreased choroidal thickness may be related to retinal tissue hypoxia, as the choroid is the major source of nutrition for the RPE and outer retinal layers [37-39].

The management of diabetic edema depends on whether the center of the retina; the macula, is involved or not. For Center-involving diabetic macular edema-, Anti-VEGF agents have become the first line of treatment. Bevacizumab, ranibizumab, and aflibercept have beneficial effects in patients with baseline better visual acuity. Aflibercept has better visual outcomes in patients with worse baseline visual acuity on presentation [40]. Recently, aflibercept and ranibizumab have received FDA approval for use in both DME and diabetic retinopathy [41]. Intravitreal steroid implants also are used in resistant cases [42].

Non-center involving macular edema - Focal or Grid laser.

After the emergence of Anti-VEGF medications, laser treatment became avoided in the center involving macular edema. It may be added as adjuvant therapy in patients not responding to anti-VEGF therapy alone. Laser helped in halting the progression of moderate visual loss, but it did not improve the visual acuity [40, 43].

4 – Optical Coherence Tomography

The Optical Coherence Tomography (OCT) has become an essential part of the clinical ophthalmic practice ever since its inception in 1991 in Massachusetts Institute of Technology [44]. OCT uses an interferometric imaging technique that maps depth-wise reflections of near-infrared (NIR) light from tissue to form cross sectional images of morphological features at the micrometer scale. It can be used to image the retina, and particularly the macula [45-48] (Figure 6).

Various techniques for OCT imaging has been devised as well, including the Time-Domain (TD-OCT), Spectral-Domain (SD-OCT), and Swept-Source (SS-OCT).

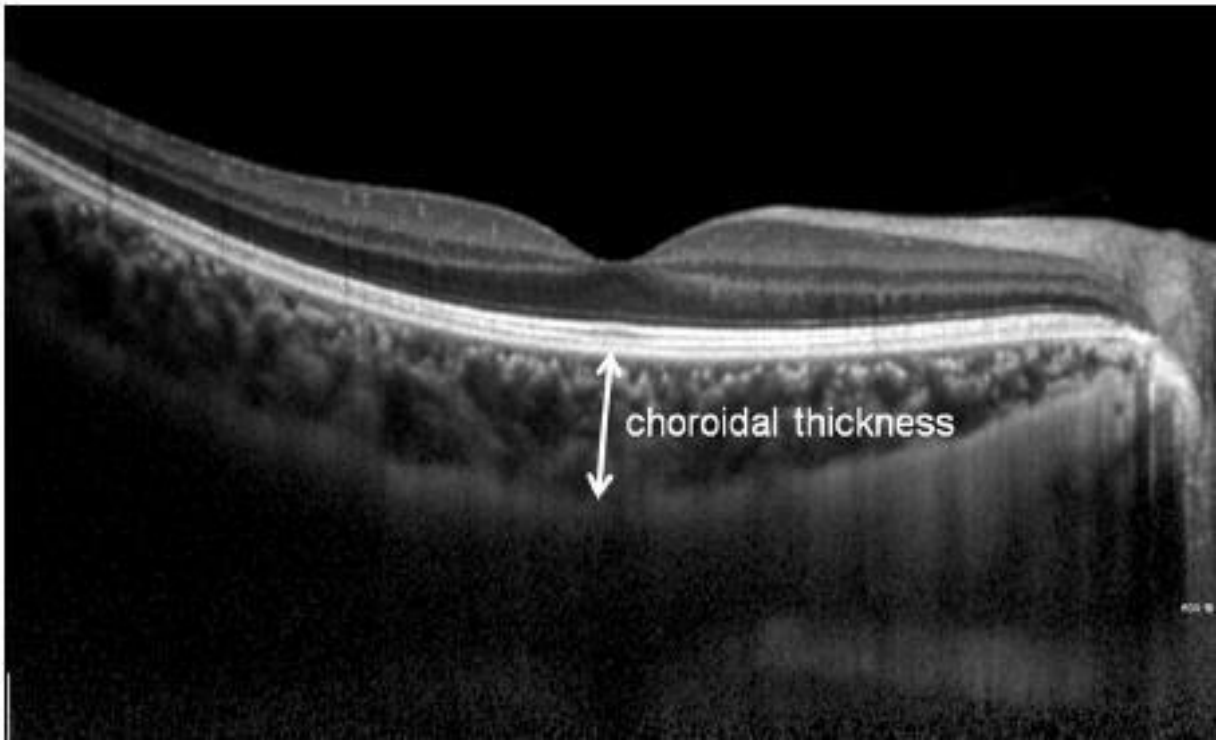

Figure 6. Optical Coherence Tomography (OCT) image of the macula, demonstrating the thickness.

While fluorescein angiography remains the only method for the identification of leaking vessels in DR, it cannot identify cases of nonvascular macular edema. OCT, however, has been extensively employed for the identification of cystoid macular edema and the monitoring of macular thickness, which is associated with a decrease in central vision [49-51].

The uses of OCT in DR are vast and priceless. It can guide the treatment of Anti-VEGF, monitor the progression of the disease, detect areas of atrophy, detect accompanying lesions, and also, in our present study, it can be used to image the choroid, detect its thickness, and view any associated lesions within it [52, 53] (Figure 7).

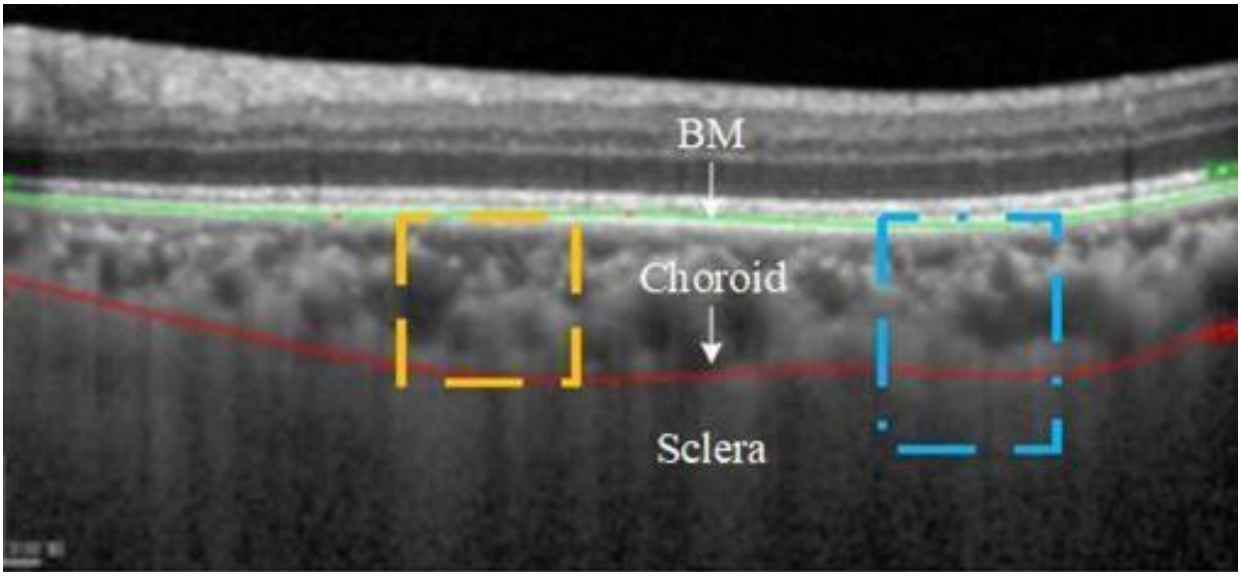

Figure 7. Optical Coherence Tomography (OCT) image of the macula, showing the underlying basement membrane (BM), choroid, and sclera.

Patients & Methods

Type of study: Prospective cohort interventional study.

Setting: Ophthalmology department, South Valley University Hospitals.

Time: patients who were examined and imaged in the outpatient clinic and were injected in the operating room between May and November of 2022.

Patients: 40 eyes, unilaterally, from 40 patients with diabetic macular edema.

Inclusion criteria: Adult phakic patients with diabetic macular edema receiving anti vascular endothelial growth factors injections without prior anti-VEGF therapy about to receive anti-VEGF intravitreal injections for the first time.

Exclusion criteria: pseudophakic patients, children and uncooperative patients with disturbed conscious level and patients with other causes of visual loss.

The study was performed according to the Declaration of Helsinki. Local ethics committee approval was obtained. Patient

consent was obtained from all patients and then they underwent full history taking and clinical examination. All the patients underwent Spectral Domain Optical Coherence Tomography (SD-OCT), visual acuity assessment, pupil reaction assessment, and fundus photography. OCT Measurements were performed before and after anti-VEGF treatment.

Methods: The procedure was performed using topical anesthesia and under complete sterile conditions. 0.5 mg\0.05 ml ranibizumab is injected 4 mm from the limbus intravitreally (in the lower temporal quadrant) by a needle (27 gauge) once monthly for 3 months. For images to be included in the present study, they were taken as close to the fovea as possible (thinnest macular point), with the understanding that slight differences in positioning affect the measured thicknesses. Using the Spectralis linear measurement tool, CT was measured perpendicularly from the outer edge of the hyper-reflective RPE to the inner sclera at 500-mm intervals temporal and nasal to the fovea up to 1,000 mm, as well as subfoveal. Measurements were performed prior to treatment and at a 6-month follow-up.

To achieve this study purpose, we evaluated and compared choroidal layer thickness changes using SD-OCT using OCT-Spectralis (Heidelberg Engineering GmbH 69121 Heidelberg / Germany, SN: TR-KT-2069, Manufactured 02/13) with the software Heidelberg Eye Explorer version 1.9.10.0 [54] (Figure 8, 9), before starting intra-vitreal Ranibizumab injections and again after finishing six months.

The primary (main) outcome was the choroidal thickness pre- and postoperatively. The secondary outcomes included visual acuity using the logMAR scale (uncorrected visual acuity UCVA and best corrected visual acuity BCVA) and central foveal thickness.

Figure 8. OCT Imaging Device.

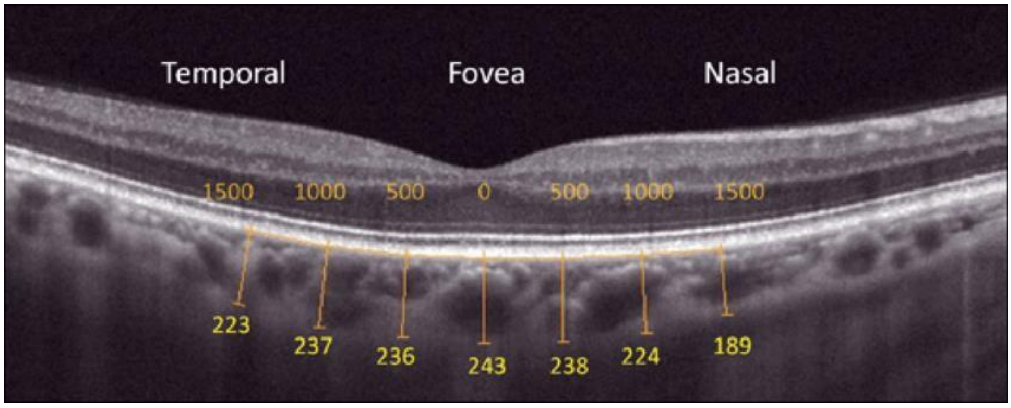

Figure 9. Choroidal Thickness Measurements from the Nasal, Temporal, & Subfoveal areas of an OCT Image.

Statistical Analysis

- Sample size was 40 eyes.
- Data was analyzed using Statistical Package for Social Sciences (SPSS) software program (version 26) [55].
- Qualitative variables were recorded as frequencies and percentages and were compared by Chi-square test.
- Quantitative measures were presented as means ± standard deviation (SD) and were compared by Student t-test.
- Regression correlation and analysis between different variables were performed as indicated. A P-value of $< 0{\cdot}05$ was considered to be significant.

Results

Our study included 40 eyes of 40 patients with diabetic macular edema. 20 eyes (50%) were right & 20 eyes (50%) were left. 17 patients (42.5%) were males, and 23 patients (57.5%) were females. The mean age of the patients was 57.68 years (SD = 7.7, range = 47:73) (Table 1) (Figure 10, 11).

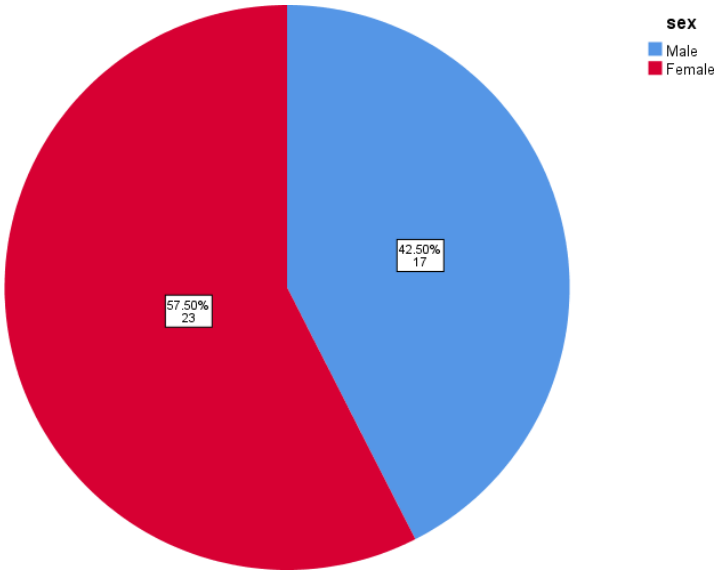

Figure 10. Pie Chart showing the sex distribution of patients.

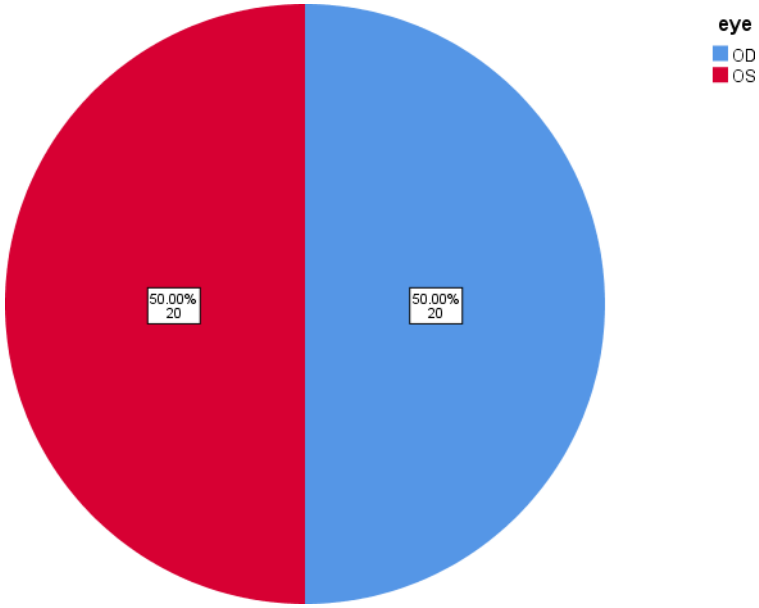

Figure 11. Pie Chart showing the eye of the patients.

Right	Left
20 (50%)	20 (50%)

Table 1. Eyes of the Patients.

Male	Female
17 (50%)	23 (50%)

Table 2. Sex of the Patients.

	Preoperative	Postoperative
Visual Acuity	1.09 (0.21)	1.01 (0.32)

Table 3. Preoperative & Postoperative Visual Acuity on the LogMAR Scale. Mean (SD)

The mean preoperative visual acuity on LogMar was 1.09 (SD = 0.21, range = 0.78:1.48) which improved to 1.01 postoperatively (SD = 0.32, range = 0.48:1.48) (Table 2). The mean preoperative nasal choroidal thickness was 241 microns (SD = 9.7, range = 216:253), and postoperatively it decreased to 226 microns (SD = 12.4, range = 195:244). The mean preoperative subfoveal choroidal thickness was 250 microns (SD = 9.4, range = 226:263) and postoperatively, it decreased to 238 microns (SD = 11.5, range = 209:253). The preoperative temporal choroidal thickness was 246 microns (SD = 9.6, range = 221:257) and postoperatively it decreased to 233 microns (SD = 12.4, range
= 205:250) (Table 3)
(Figure 12-14).

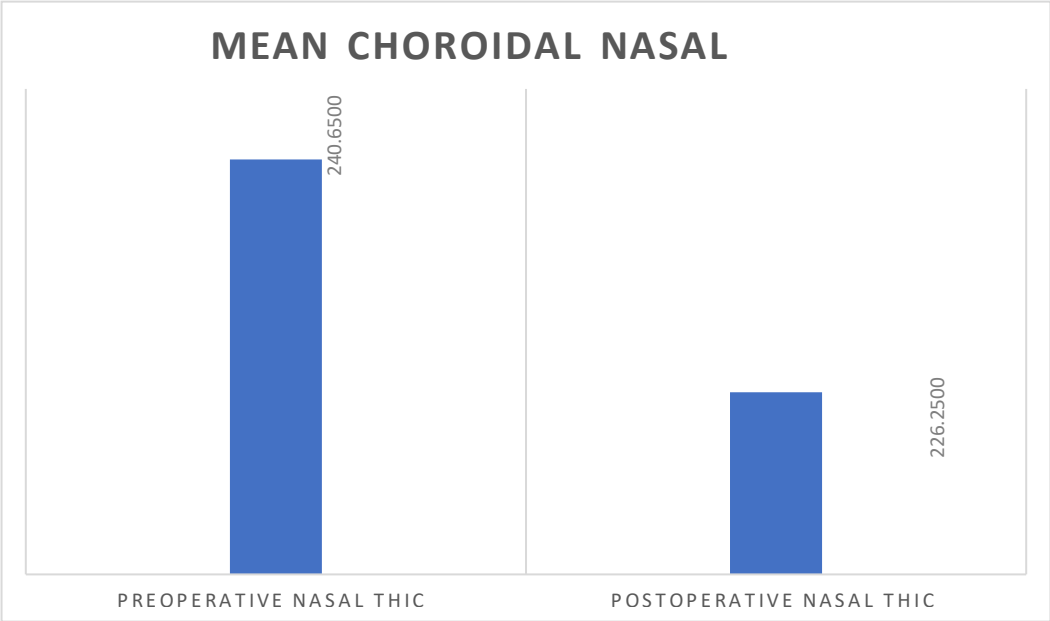

Figure 12. Bar chart of Preoperative vs Postoperative Nasal Choroidal Thickness.

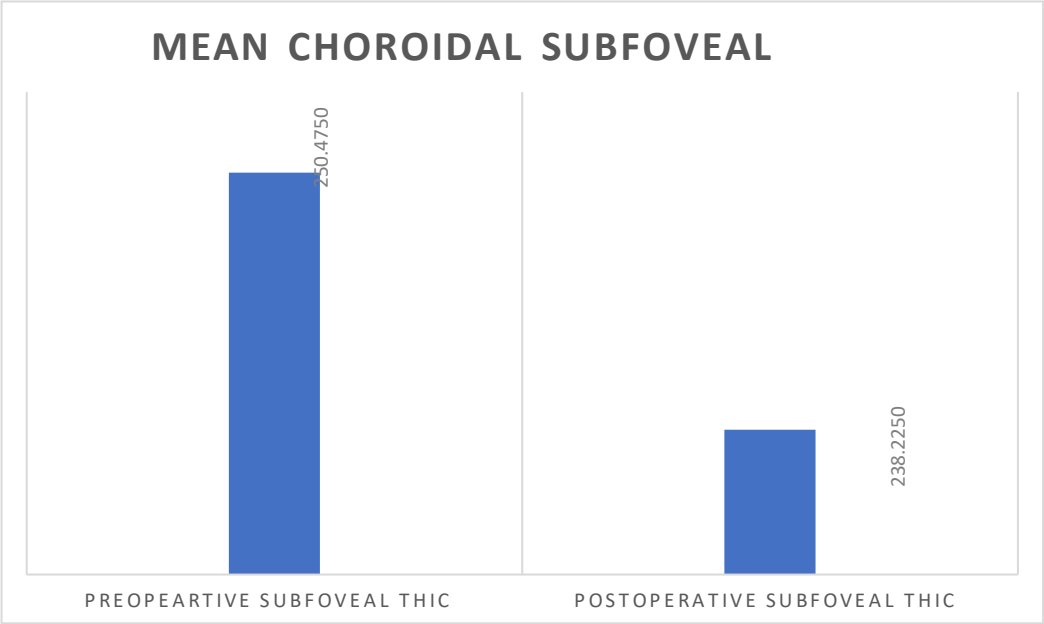

Figure 13. Bar chart of Preoperative vs Postoperative Subfoveal Choroidal Thickness.

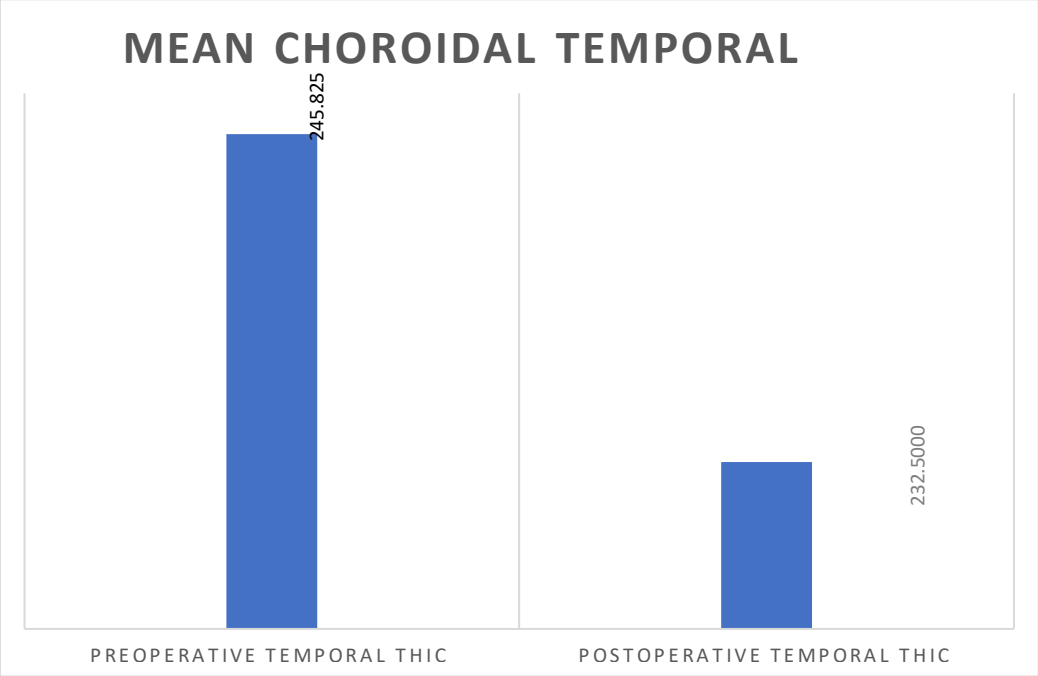

Figure 14. Bar chart of Preoperative vs Postoperative Temporal Choroidal Thickness.

Using Paired samples t-test, the mean improvement in visual acuity was 0.08 (SD = 0.13, SEM = 0.02, 95% CI = 0.04:0.12, t = 3.8, df = 39, p = 0). The mean decrease in nasal choroidal thickness was 14.4 (SD = 4.3, SEM = 0.68, 95% CI = 13.02:15.78, t = 21.1, df = 39, p value = .000). The mean decrease in subfoveal choroidal thickness was12.25 (SD = 3.59, SEM = 0.57, 95% CI = 11.1:13.4, t = 21.6, df =39, p value = .000).

The mean decrease in temporal choroidal thickness was13.33 (SD = 4.2, SEM = 0.67, 95% CI = 11.98:14.67, t = 19.97, df = 39, pvalue = .000).

	Preoperative	**Postoperative**	**p**
Nasal	241 (9.7)	226 (12.6)	.000
Subfoveal	250 (9.4)	238 (11.5)	.000
Temporal	246 (9.6)	233 (12.4)	.000

Table 4. Preoperative & Postoperative Choroid Thickness Measurements, Mean (SD).

Discussion

The results of our study show that anti-VEGF injection affect the thickness of the choroid in eyes affected with diabetic retinopathy. The temporal, nasal, and subfoveal choroid showed a decrease in thickness post anti-VEGF injections and the results were statistically significant. The mean decrease in nasal choroidal thickness was 14.4 (SD = 4.3, 95% CI = 13.02:15.78, p = 0). The mean decrease in subfoveal choroidal thickness was 12.25 (SD = 3.59, 95% CI = 11.1:13.4, p = 0). The mean decrease in temporal choroidal thickness was 13.33 (SD = 4.2, 95% CI = 11.98:14.67, p = 0).

Wei Wang et al. 2020, who examined 1347 patients, found that in early diabetic retinopathy, the choroid tends to increase in thickness in the early stages of diabetic retinopathy, but decrease as diabetic retinopathy progressed along [56]. This goes along with a meta-analysis performed by Endo et al. 2020 which included 17 studies comparing 3016 non-diabetic eyes to 1197 diabetic eyes and found that subfoveal choroidal thickness is lower in diabetic eyes [57]. Lains et al. 2018 also found that patients with proliferative diabetic retinopathy had thinner central

choroidal thickness than controls [58].

Wang et al. 2018 found that not only the choroid, but also the retinal pigment epithelium and outer retinal layers were significantly decreased in volume in diabetic retinopathy compared to controls in different regions of the retina [59]. Regatieri et al. 2012 found that the average choroidal thickness in the temporal, nasal, and subfoveal areas was lower in NPDR patients than in normal patients, and lower in DME patients than in NPDR patients, and lower in PDR patients than in DME patients [16]. Unsal et al. 2014 found a similar finding [60].

Rayess et al. 2015 had a similar sample size to ours, with 53 eyes, and found that diabetic eyes with a thicker subfoveal choroid thickness responded better to anti-VEGF treatment in terms of BCVA [61]. The exact mechanism for the changes in choroid is interesting, especially given that even in patients with diabetes without diabetic retinopathy, Ferreira et al. 2018 found that the choroidal thickness is significantly more than non-diabetic patients by 6.16-24.27 μm [62]. Xu et al. 2013 demonstrated a similar finding [63].

However, a few studies found that choroidal thickness increases as diabetic retinopathy increases in severity [64]. Endo et al. 2018 resorted to dividing the choroid into its layers and found that the total and outer choroid thicknesses in mild to moderate NPDR patients were significantly thinner than normal controls. They also found that the choroidal outer layer thickness of the severe NPDR patients was significantly thicker than normal controls [65].

Treatment of diabetic retinopathy and diabetic macular edema can also affect choroidal thickness. Ohara et al. 2018 found that PRP causes a decrease in the choroidal thickness, which was persistent, even 6 months after treatment [66]. Endo et al. 2018 in the DM treatment group, who received continuous systemic medication treatments including oral hypoglycemic agents with/without subcutaneous insulin therapy for DM there were no significant differences from the control group regarding choroidal layer thicknesses in all stages of DR [65].

These findings prove that the choroidal thickness suffers a

decrease in all areas after anti-VEGF injection. It is not known if that decrease is due to pathological process of the diabetic retinopathy itself or if it's related to the anti-VEGF injection on the vascular endothelium. Further research is warranted to compare both groups of patients and conclude a follow up of patients using OCT to determine which of the two groups would suffer a greater decrease in choroidal thickness.

Our study is limited by a relatively low sample size, but its strength is a low p value ($p = .000$). Further studies are recommended in order to ascertain the relationship between diabetic retinopathy & choroidal thickness.

Conclusion

We examined 40 eyes of 40 patients with diabetic retinopathy who received anti-VEGF injections before and after the procedure. OCT was examined to measure the choroidal thickness in the temporal, nasal, and subfoveal areas. The temporal, nasal, and subfoveal choroid showed a decrease in thickness after receiving anti-VEGF injections in DME patients. The mean decrease in nasal choroidal thickness was 14.4 (SD = 4.3, 95% CI = 13.02:15.78, p value = .000). The mean decrease in subfoveal choroidal thickness was 12.25 (SD = 3.59, 95% CI = 11.1:13.4, p value = .000). The mean decrease in temporal choroidal thickness was 13.33 (SD = 4.2, 95% CI = 11.98:14.67, p value = .000). Our sample size was limited but our results were statistically significant. Further studies are recommended in order to ascertain the relationship between diabetic retinopathy, anti- VEGF injections & choroidal thickness.

Summary

Introduction: The choroid is a vascularized tissue that plays a vital role in providing metabolic support to the outer retina. We are concerned with studying the changes in choroidal thickness related to anti-VEGF injection in diabetic patients.

Methods: This was a prospective cohort interventional study conducted at South Valley University Hospital, Ophthalmology department and included 40 eyes from 40 patients with diabetic macular edema. Inclusion criteria included adult phakic patients with diabetic macular edema receiving anti vascular endothelial growth factors injections without prior anti-VEGF therapy. All the patients underwent SD-OCT, visual acuity assessment. OCT Measurements were performed before and six months after anti-VEGF treatment.

Results: The mean decrease in nasal choroidal thickness was 14.4 (SD = 4.3, 95% CI = 13.02:15.78, p value = .000). The mean decrease in subfoveal choroidal thickness was 12.25 (SD = 3.59, 95% CI = 11.1:13.4 , p value = .000). The mean decrease in

temporal choroidal thickness was 13.33 (SD = 4.2, 95% CI = 11.98:14.67, p value = .000).

Conclusion: The temporal, nasal, and subfoveal choroid showed a decrease in thickness after receiving anti-VEGF injections in DME patients. The mean decrease in nasal choroidal thickness was 14.4 (SD = 4.3). The mean decrease in subfoveal choroidal thickness was12.25 (SD = 3.59). The mean decrease in temporal choroidal thickness was13.33 (SD = 4.2). Our sample size was limited but our results were statistically significant (p value = .000). Further studies are recommended in order to ascertain the relationship between diabetic retinopathy, anti-VEGF injections & choroidal thickness.

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الملخص العربي

مقدمة: المشيمية عبارة عن نسيج وعائي يلعب دورا حيويا في توفير الدعم الايضي للشبكية. نحن مهتمون بدراسة التغيرات في سمك المشيمية المرتبطة بحقن مضاد عامل النمو الوعائي فى مرضى السكرى.
الطريقه:
كانت هذه دراسة تداخلية جماعية محتملة أجريت في مستشفى جامعة جنوب الوادى ،قسم طب وجراحه العيون ،وشملت 40عينا من40 مريضا يعانون من الوذمه البقعيه السكريه.

تضمنت معايير اشتمال المرضى البالغين المصابين بالوذمة البقعية السكرية الذين يتلقون حقن مضاد عوامل نمو بطانة الوعائية الدموية دون علاج مسبق بمضادات عامل النموالوعائي.
خضع جميع المرضى للألشعة المقطعية على شبكية العين وتقييم النظر قبل الحقن وبعد . الحقن بسته اشهر
النتائج :كان متوسط النقص فى سمك المشيمه الانفيه الداخليه 14.4ميكرون وكان متوسط النقص فى سمك المشيمه السفليه لمركزالشبكيه 12.25ميكرون وكان متوسط النقص فى سمك المشيمه الصدغيه الخارجيه 13.33ميكرون .
الخالصه :
إن المشيمية الصدغية والانفية والتحت مركزية أظهرت انخفاضا في السمك بعد الحقن بمضادات عامل النمو الوعائي في مرضى االارتشاح الشبكي السكري.

تقييم سمك مشيمة العين قبل وبعد حقن الجسم الزجاجى بالعوامل المضادة للوعائية كعلاج للارتشاح السكرى بمركز الابصار باستخدام التصوير المقطعى البصرى.

رسالة عملية مقدمة من

الطبيب / نها محمود احمد محمد

بكالوريوس الطب والجراحة

توطئة للحصول على درجة الماجستير في طب وجراحة العيون

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