Research Article / Araştırma Makalesi

Effect of Vitamin D Deficiency on Peripapillary Retinal Nerve Fiber Layer and Choroidal Thickness: A Cross-Sectional Study

D Vitamini Eksikliğinin Peripapiller Retina Sinir Lifi Tabakası ve Koroid Kalınlığı Üzerindeki Etkisi: Kesitsel Bir Çalışma

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Abstract

Background: To evaluate the effect of vitamin D deficiency (VDD) on the choroidal thickness (CT) and the peripapillary retinal nerve fiber layer thickness (RNFLT).
Materials and Methods: This study involved 60 patients with VDD (Group 1) and 50 healthy controls (Group 2). Group 1 was further divided into three subgroups—severe deficiency (Group 1a), deficiency (Group 1b), and insufficiency (Group 1c)—based on serum vitamin D levels. Optical coherence tomography (OCT) was utilized to measure the subjects' subfoveal CT, nasal CT, temporal CT, and peripapillary RNFLT.
Results: The subfoveal, temporal, and nasal CTs were observed to be significantly thinner in Group 1 than in Group 2 ($p < 0.001$). However, peripapillary RNFLTs did not differ significantly between Groups 1 and 2 in all quadrants (all $p > 0.05$). Subfoveal, temporal, and nasal CTs were significantly thinner in Group 1a than in Groups 1b and 1c (all $p < 0.001$). However, peripapillary RNFLTs were comparable across subgroups in all quadrants (all $p > 0.05$). Vitamin D levels correlated positively with subfoveal, temporal, and nasal CTs among patients with VDD.
Conclusions: Patients with VDD exhibited decreased CTs, and those with severe deficiency showed an even more dramatic decrease. Therefore, VDD may cause choroidal pathologies.

Keywords: Vitamin D Deficiency, Choroidal Thickness, Retinal Nerve Fiber Layer, Optical Coherence Tomography

Öz

Amaç: D vitamini eksikliğinin (DVE) koroid kalınlığı (KK) ve peripapiller retina sinir lifi tabakası kalınlığı (RSLTK) üzerindeki etkisini değerlendirmek.
Materyal ve Metod: Çalışma grubu, DVE'si olan 60 hastadan (Grup 1) ve 50 sağlıklı kontrolden (Grup 2) oluşuyordu. Alt gruplama için serum D vitamini düzeyleri kriter alınarak Grup 1'den şiddetli eksiklik (Grup 1a), eksiklik (Grup 1b) ve yetersizlik (Grup 1c) olmak üzere üç alt grup oluşturuldu. Deneklerin subfoveal, nazal, temporal KK ve peripapiller RSLTK'sini ölçmek için optik koherens tomografi (OKT) (Heidelberg Spectralis OKT) kullanıldı.
Bulgular: Grup 1'in subfoveal, temporal ve nazal KK ölçümlerinin Grup 2'ninkinden önemli ölçüde daha ince olduğu görüldü (p<0,001). Grup 1 ve Grup 2'nin tüm kadranlarda Peripapiller RSLTK'leri benzerdi (her biri için p>0,05). Subfoveal, temporal ve nazal KK ölçümleri Grup 1a'da Grup 1b ve Grup 1c'den önemli ölçüde daha inceydi (her biri için p<0,001). Alt grupların peripapiller RSLTK'leri tüm kadranlarda benzerdi (her biri için p>0,05). DVE'li hastalarda subfoveal KK, temporal KK ve nazal KK skorları ile D vitamini seviyeleri arasında pozitif korelasyonlar bulundu.
Sonuç: DVE'li hastalarda KK'de azalma izlendi ve şiddetli eksikliği olanlarda daha da belirgin bir azalma mevcuttu. DVE koroidal patolojilere neden olabilir.

Anahtar Kelimeler: D Vitamini Eksikliği, Koroid Kalınlığı, Retina Sinir Lifi Tabakası, Optik Koherens Tomografi

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Introduction

Vitamin D deficiency (VDD) is a relatively common condition, affecting nearly half of the global population (1). In addition to being obtained from various foods, vitamin D is also created endogenously by the body through several processes (2). Its function in numerous tissues is comparable to that of hormones, and its chemical structure is comparable to that of traditional steroid hormones (3). Vitamin D's primary roles include controlling calcium-phosphorus balance and, consequently, bone metabolism (4). Vitamin D's physiologically active form, 1,25-dihydroxyvitamin D, has also been demonstrated to possess anti-inflammatory, antiangiogenic, and anticarcinogenic qualities (5). Recent considerations suggest that serum levels of 25-hydroxyvitamin D [25(OH)D], the main circulating form of vitamin D, may serve as the best predictor of the body's vitamin D supply. The Endocrine Society's Clinical Practice Guidelines classify serum 25(OH)D concentrations as insufficient within the range of 20 to 30 ng/mL, deficient between 10 and 20 ng/mL, and severely deficient when below 10 ng/mL (4).

Numerous eye tissues have been found to contain the vitamin D receptor (VDR) and regulatory enzymes. Numerous ocular conditions and disorders have been linked to serum 25(OH)D levels, including myopia, age-related macular degeneration, diabetic retinopathy, high intraocular pressure (IOP), and uveitis (6–9). Additionally, it has been demonstrated that choroidal endothelial cells and retinal vascular cells express the VDR (10). A lower serum level of 25(OH)D has been associated with numerous alterations in retina and choroid structures.

The advancement of retinal imaging techniques has made it possible to use optical coherence tomography (OCT) to investigate the retinal and choroidal layers in greater depth and to learn more about the alterations in the retina and choroids that arise in systemic and ocular disorders. Therefore, this study aimed to assess the choroidal thickness (CT) and peripapillary retinal nerve fiber layer (RNFL) thickness (RNFLT) of patients with vitamin D insufficiency, deficiency, and severe deficiency based on their serum 25(OH)D levels using OCT. To our knowledge, this study is the first to investigate the retina and choroid in subgroups of adult patients with VDD.

Materials and Methods

This prospective cross-sectional study was conducted in the ophthalmology department of a tertiary care clinic. The study protocol was approved by the local ethics committee of Adıyaman University Clinical Research Ethics Committee (date:23/06/2022; decision number: 2022-01-02). This study adhered to the ethical principles outlined in the Declaration of Helsinki, and all participants provided informed consent.

This study examined 110 right eyes, comprising 60 right eyes from 60 patients with low serum vitamin D levels and 50 right eyes from 50 healthy individuals. Patients presenting with various problems in the internal medicine clinic, and exhibiting serum 25(OH)D levels below 30 ng/mL during routine examinations, were referred to the ophthalmology department (Group 1). The control group comprised healthy individuals whose serum 25(OH)D levels were within the normal limits (30–50 ng/mL) in the preceding month (Group 2). The patients in Group 1 were divided into three subgroups based on their serum 25(OH)D levels (4): severely deficient (<10 ng/mL, Group 1a), deficient (10–20 ng/mL, Group 1b), and insufficient (20–30 ng/mL, Group 1c).

All participants underwent a thorough ophthalmological examination under the same room settings by the same ophthalmologist. The ophthalmological examination involved examining the anterior segment with a slit lamp, measuring IOP via Goldman applanation tonometry, cycloplegic refraction, measuring visual level with the Snellen chart (cases with best corrected visual acuity of 20/20 were included), examining the dilated fundus to detect chorioretinal pathologies (90D lens), and OCT examination. The participants' demographic characteristics, such as gender and age, and clinical characteristics, such as spherical equivalent, were recorded. Individuals with a history of ocular surgery, with spherical refraction >±3.00 diopters, with eye diseases affecting the retina and choroid (e.g., uveitis, central serous chorioretinopathy, glaucoma, retinal vascular occlusion), with corneal scaring, with cataracts, with heavy alcohol and caffeine consumption, who smoked, who were pregnant, who were receiving vitamin D treatment (0–12 months, excluding prophylactic treatment), and who were aged under 18 years were excluded from this study.

The peripapillary RNFLT was measured using a spectral domain (SD) OCT (Heidelberg Engineering, Heidelberg, Germany). The participant's blood pressure was measured before the OCT procedure began. Following pupil dilation, OCT measurements were taken in the same setting and by the same ophthalmologist between 9:30 a.m. and 11.30 a.m., and those with a quality of >20 were used. The peripapillary RNFLT was measured using fast optic disc scan procedures. The Fast RNFL mode enables automated computation of the peripapillary RNFLT in high-speed mode, which was accomplished by enabling the automated real-time function and setting the frame rate to 16fps. The protocol for fast optic disc included six radial line scans along with three subalternate 360° circular scans with 3.46 mm diameters around the optic disc. This program ensures the standard 12° circular scan has a thickness profile spanning the temporal-superior-nasal-inferior regions. The software computes the average thickness (in μm) both globally and in six sectors centered on the optic disc. Figure 1 shows these sectors, including the temporal, temporal superior, temporal inferior, nasal, nasal inferior, and nasal superior sectors.

The enhanced depth imaging (EDI) configuration of the OCT was used to determine the CT. The fundus image acquired using EDI SD-OCT from the outer border of the retinal pigment epithelium (RPE) to the choroid-scleral junction was

measured manually at three points by the same ophthalmologist (Figure 2): subfoveal (SFCT), 500 µm from the fovea nasally (NCT), and 500 µm from the fovea temporally (TCT).

Statistical analysis

Statistical analyses were conducted with SPSS Statistics software for Windows (version 24.0; IBM, Armonk, NY, US). The data were summarized using descriptive statistics, which were presented in a tabular format. Continuous (numerical) variables are presented as the mean ± standard deviation and the median (minimum−maximum), and categorical variables are presented as the number (percentage). The normality of the numerical variables was assessed with appropriate tests and visual representations, depending on the sample size and data characteristics. While the Shapiro–Wilk test was preferred for comparisons with small samples (n < 50), the Kolmogorov–Smirnov and Anderson–Darling tests were preferred for comparisons with large samples ($n \geq 50$). Categorical variables were compared between groups using Pearson's chi-square test. Numerical variables were compared between two groups using the independent samples t-test if normally distributed and the Mann–Whitney U test if non-normally distributed. Numerical variables were compared between more than two groups using one-way analysis of variance (ANOVA) if normally distributed or the Kruskal–Wallis H test if non-normally distributed. Non-parametric pairwise comparisons were conducted using the Dwass–Steel–Critchlow–Fligner test. When numerical variables did not exhibit a normal distribution, Spearman's rank correlation coefficient was used to examine the relationship between the numerical variables. $p < 0.05$ was considered statistically significant.

Results

This study involved 110 subjects (60 in Group 1 and 50 in Group 2). Group 1 consisted of subjects with VDD, comprising 22 (46.7%) males and 38 (53.3%) females. Group 2 consisted of healthy controls, comprising 24 (48.0%) males and 26 (52.0%) females.

The mean age was 34.6 ± 8.1 (19–50) years in Group 1 and 33.9 ± 7.9 (18–49) years in Group 2. Gender, age, IOP, and spherical equivalent did not differ significantly between groups ($p = 0.315$, $p = 0.659$, $p = 0.182$, $p = 0.727$, respectively). In contrast, serum vitamin D levels did differ significantly between groups ($p < 0.001$, Table 1).

Table 1. Comparison of Demographic Characteristics of Groups

	Group 1 (n=60)	**Group 2 (n=50)**	***p***
Age (year) †	34,6 ± 8,1	33,9 ± 7,9	0,659*
Gender ‡ Male Female	 22 (46,7) 38 (53,3)	 24 (48,0) 26 (52,0)	0,315**
Serum Vitamin D Level (ng/mL) §	13,5 (4,8 – 28,9)	39,8 (31,8 – 48,0)	**<0,001*** **
IOP (mmHg) §	16,0 (1,0 – 20,0)	15,0 (12,0 – 19,0)	0,182***
Spherical equivalent (Diopter) §	-0,38 (-2,50 – +2,00)	-0,38 (-2,75 – +2,25)	0,727***

IOP: İntraocular pressure
†: Mean ± Standard deviation, ‡: n (%), §: Median [Min.-Max.],
** Independent Samples T-Test.*
*** Pearson Chi-Square test.*
**** Mann-Whitney U test.*
Bold values indicate p < 0.05.

SFCT, TCT, and NCT were significantly thinner in Group 1 than in Group 2 (all $p < 0.001$, Figure 3). However, peripapillary RNFLT did not differ significantly between Groups 1 and 2 in all quadrants (both $p > 0.05$, Table 2). In patients with VDD, serum vitamin D levels correlated positively with the SFCT ($r = 0.651$, $p < 0.001$), TCT ($r = 0.660$, $p < 0.001$), and NCT ($r = 0.676$, $p < 0.001$). In contrast, serum vitamin D levels were not significantly correlated with peripapillary RNFLT.

Regarding the subgroups in Group 1, Group 1a comprised 7 males and 13 females, Group 1b comprised 8 males and 12 females, and Group 1c comprised 7 males and 13 females. The mean age was 32.7 ± 8.6 years in Group 1a, 35.4 ± 7.3 years in Group 1b, and 35.6 ± 8.4 years in Group 1c. Gender, age, IOP, and spherical equivalent did not differ significantly among the subgroups ($p = 0.931$, $p = 0.466$, $p = 309$, and $p = 0.762$, respectively). However, serum vitamin D levels did differ significantly among the subgroups ($p < 0.001$).

SFCT, TCT, and NCT differed significantly between the subgroups (all $p < 0.001$). However, peripapillary RNFLT did not differ significantly among subgroups in all quadrants (all $p > 0.05$, Table 3). Pairwise comparisons of the subgroups revealed that serum vitamin D levels were significantly lower in Group 1a than in Groups 1b and 1c ($p<0.001$ for both). Similarly, SFCT, TCT, and NCT were significantly thinner in Group 1a than in Groups 1b and 1c (all $p < 0.05$) but did not differ significantly between Groups 1b and 1c (all $p > 0.05$, Table 4).

Table 2. Comparison of Peripapillary Retinal Nerve Fiber Layer and Choroidal Thicknesses of The Groups

	Group 1 (n=60)	**Group 2** (n=50)	***p***
Subfoveal CT (µm) §	330,0 (284 – 360)	388,5 (338,0 – 420,0)	**<0,001***
Temporal CT (µm) §	310,5 (250 – 331)	363,0 (312,0 – 391,0)	**<0,001***
Nasal CT (µm) §	309,0 (255 – 328)	354,5 (315,0 – 384,0)	**<0,001***
G-RNFL (µm) §	98,0 (78,0 – 114)	98,0 (93,0 – 107,0)	0,112*
N-RNFL (µm) §	75,0 (62,0 – 86,0)	76,0 (65,0 – 87,0)	0,489*
NS-RNFL (µm) §	110,0 (99,0 – 130,0)	111,0 (98,0 – 129,0)	0,550*
TS-RNFL (µm) §	135,0 (109 – 158)	135,0 (115– 163)	0,509*
T-RNFL (µm) §	75,0 (60 – 88)	76 (63,0 – 85,0)	0,127*
TI-RNFL (µm) †	144,4 ± 9,3	146,4 ± 9,1	0,274**
NI-RNFL (µm) §	120,0 (107,0 – 138,0)	120,0 (101,0 – 131,0)	0,374*

CT:Choroidal thickness, µm: Micrometer, RNFL: Retinal Nerve Fiber Layer, G:Global, N:Nasal, NS: Nasal-Superior, TS: Temporal-Superior, T: Temporal, TI:Temporal-Inferior, NI: Nasal-Inferior

§: Median [Min.-Max.], †: Mean ± Standard deviation.

**. Mann-Whitney U test. ** Independent Samples T-Test.*

Bold values indicate p < 0.05.

Table 3. Comparison of Subgroups in Patients With Vitamin D Deficiency

	Patient (Subgroup)			***p***
	Group 1a (n=20)	**Group 1b (n=20)**	**Group 1c (n=20)**	
Age(year) †	32,7 ± 8,6	35,4 ± 7,3	35,6 ± 8,4	0,466*
Gender ‡ Male Female	 7 (35,0) 13 (65,0)	 8 (40,0) 12 (60,0)	 7 (35,0) 13 (65,0)	0,931**
Serum Vitamin D Level (ng/mL) §	7,2 (4,8 – 9,1)	13,5 (11,6 – 19,0)	25,9 (22,9 – 28,9)	**<0,001*****
IOP (mmHg) §	16,5 (11,0 – 20,0)	15,5 (12,0 – 18,0)	16,0 (11,0 – 20,0)	0,309***
Spherical equivalent (Diopter) §	-0,5 (-2,5 – +2,0)	-0,4 (-2,0 – +2,0)	-0,1 (-2,2 – +2,0)	0,762***
Subfoveal CT (µm) §	296,5 (284,0 – 315,0)	336,5 (315,0 – 355,0)	338,0 (318,0 – 360,0)	**<0,001*****
Temporal CT(µm) §	275,5 (250,0 – 291,0)	318,5 (305,0 – 331,0)	318,5 (305,0 – 331,0)	**<0,001*****
Nasal CT (µm) §	272,0 (255,0 – 288,0)	313,5 (302,0 – 328,0)	316,0 (301,0 – 325,0)	**<0,001*****
G-RNFL (µm)§	97,5 (78,0 – 108,0)	98,0 (85,0 – 109,0)	97,0 (79,0 – 114,0)	0,960***
N-RNFL (µm) §	75 (66,0 – 86,0)	77,0 (65,0 – 86,0)	74,5 (62,0 – 86,0)	0,939***
NS-RNFL (µm) §	110,0 (92,0 – 129,0)	109,5 (95,0 – 132,0)	110,0 (88,0 – 128,0)	0,965***
TS-RNFL (µm) †	134,3 ± 10,9	134,6 ± 11,9	133,8 ± 11,1	0,977*
T-RNFL (µm) §	75,5 (65,0 – 85,0)	75,0 (60,0 – 88,0)	76,5 (64,0 – 82,0)	0,733***
TI-RNFL (µm) †	143,7 ± 10,5	144,3 ± 8,4	145,4 ± 9,3	0,842*
NI-RNFL (µm) §	120,0 (113,0 – 129,0)	119,5 (109,0 – 135,0)	120,0 (112,0 – 138,0)	0,790***

CT:Choroidal Thickness, IOP: İntraocular Pressure RNFL: Retinal Nerve Fiber Layer, G:Global, N:Nasal, NS: Nasal-Superior, TS: Temporal-Superior, T: Temporal, TI:Temporal-Inferior, NI: Nasal-Inferior

†: Mean ± Standard Deviation, ‡: N (%), §: Median [Min.-Max.], µm: Micrometer

** One-Way ANOVA Test. ** Pearson Chi-Square *** Kruskal Wallis-H Test.*

Bold Values İndicate p< 0.05.

Table 4. Pairwise comparison of subgroups with vitamin D deficiency

	p*
Serum Vitamin D Level (ng/mL)	
Group 1a- Group 1b	**<0.001**
Group 1a- Group 1c	**<0.001**
Group 1b- Group 1c	**<0.001**
Subfoveal Choroidal Thickness (µm)	
Group 1a- Group 1b	**<0.001**
Group 1a- Group 1c	**<0.001**
Group 1b- Group 1c	0.907
Temporal Choroidal Thickness (µm)	
Group 1a- Group 1b	**<0.001**
Group 1a- Group 1c	**<0.001**
Group 1b- Group 1c	0.918
Nasal Choroidal Thickness (µm)	
Group 1a- Group 1b	**<0.001**
Group 1a- Group 1c	**<0.001**
Group 1b- Group 1c	0.541

*Dwass-Steel-Critchlow-Fligner test.

Bold values indicate p < 0.05.

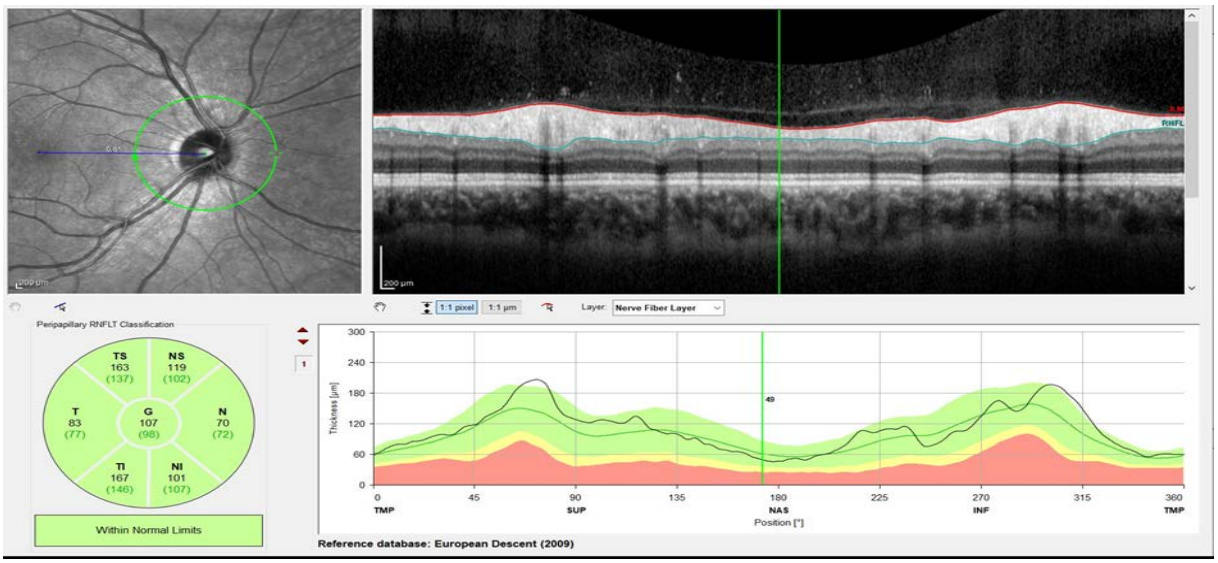

Figure 1. Peripapillary retinal nerve fiber layer thickness analysis of a case is shown.
Measurement was made in the global, temporal(T), temporal superior(TS), temporal inferior(TI), nasal(N), nasal inferior(NI) and nasal superior(NS) quadrants at the center of the optic disc.

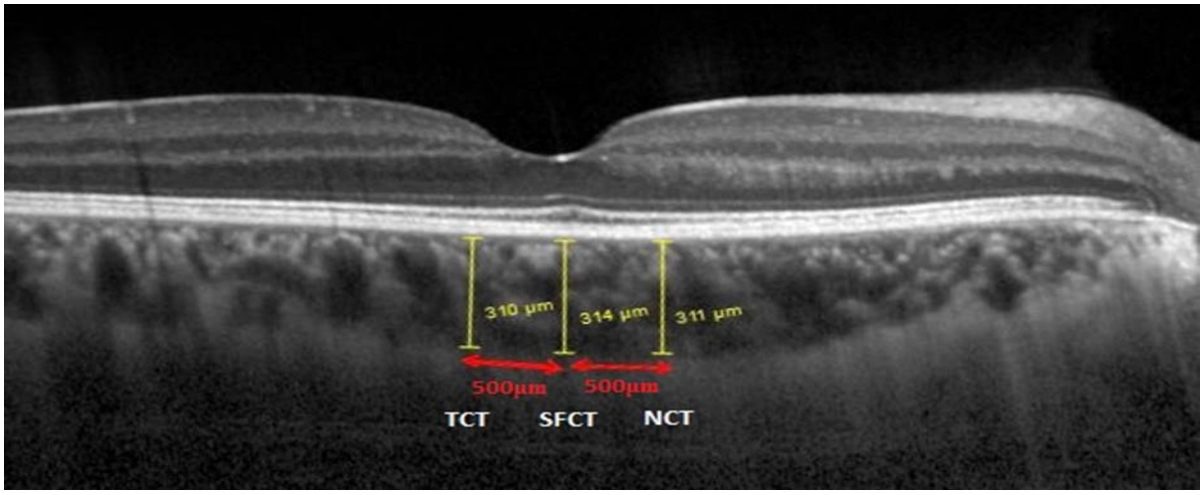

Figure 2. The choroidal thickness was measured manually at three points: subfoveal region, 500 µm from the fovea temporally, and 500 µm from the fovea nasally.
SFCT: subfoveal choroidal thickness, NCT: nasal choroidal thickness, TCT: temporal choroidal thickness, µm: micrometer.

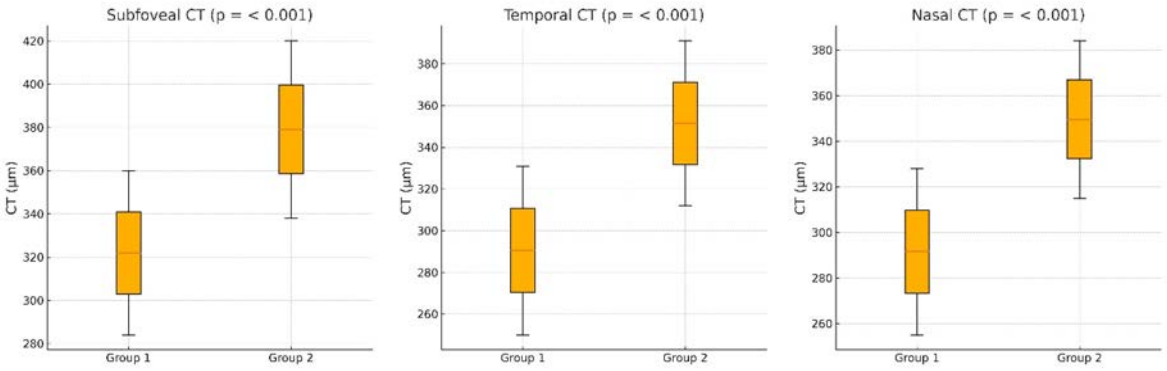

Figure 3. Box plot images showing the difference in subfoveal choroidal thickness, temporal choroidal thickness, and nasal choroidal thickness between Group 1 and Group 2. The line inside the box indicates the median value, and the upper and lower bars indicate the maximum and minimum values, respectively.
CT: Choroidal thickness

Discussion

The choroid is a highly vascularized tissue within the eye. While the vascular endothelial growth factor (VEGF) stimulates sprouting, elongation, and proliferation of endothelial cells, vitamin D serves as a potent inhibitor of these processes (11). It also inhibits the proliferation of vascular smooth muscle cells and attenuates the vascular mitogenic response to stimulatory factors (12). Additionally, it influences the renin-angiotensin system and enhances endothelial cell-dependent vasodilation (13,14). VDD may result in endothelial dysfunction because it reduces oxidative stress and lipid peroxidation in the vascular endothelium (15,16). VDD has also been associated with increased vascular calcification, platelet aggregation, and thrombogenesis (17). Considering the systemic effects of VDD, we hypothesized that it may cause choroidal thinning in the eye.

In our study, we observed significantly thinner SFCT, TCT, and NCT in patients with VDD than in healthy controls. In addition, serum vitamin D levels were negatively correlated with SFCT, TCT, and NCT. Vural et al. found significantly thinner subfoveal, peripapillary nasal, and peripapillary inferior CTs in individuals with VDD, reporting that this choroidal thinning correlated with serum vitamin D levels (18). In another study, the subfoveal, nasal, and temporal CTs were significantly thinner in patients with VDD than in controls, and the CTs increased after vitamin D treatment (19). Gurbostan et al. found a significantly thinner subfoveal CT in patients with VDD than in controls (20). The results of these studies conducted in adult patients are consistent with our study. However, unlike these studies, our study divided the patients with VDD into subgroups according to their serum vitamin D level. In the studies by Kocaay et al. and Aydemir et al. involving pediatric patients, the CT was significantly thinner in patients with VDD (21,22), which is consistent with our study.

Vitamin D limits damage to cellular and mitochondrial membranes by reducing the production of reactive oxygen species (ROS) and activating nitric oxide synthases (23). It also protects neurons against ROS that have already been generated by inhibiting gamma-glutamyl transferases in the brain, which play crucial roles in glutathione metabolism and neurotrophin expression (24). This evidence suggests vitamin D has neuroprotective effects on the nervous system.

Disruptions in neuroprotective and immune regulatory mechanisms may cause neurodegenerative damage to optic nerve axons and ganglion cell bodies in patients with VDD (8). Our study found no significant difference in peripapillary RNFLT between patients with VDD and healthy controls in all quadrants. Other studies have revealed that the RNFL may be affected by factors other than increasing age (13,18). Additionally, the RNFL may be lost as the duration of VDD increases, and it is unknown how long the patients included in our study had experienced VDD; therefore, more comprehensive studies are needed. Vural et al. found no significant difference in the RNFL between patients with VDD and controls in all quadrants (18). Another study on pediatric patients found no significant difference in RNFL except in the nasal superior quadrant between those with VDD and controls (22). Similarly, Ozturk et al. found no significant difference in mean RNFLT between patients with VDD and controls. Therefore, our study is supported by these findings (25).

Our study had some limitations. Firstly, we could not assess the choroid using swept-source OCT technology or OCT angiography, which would facilitate enhanced visualization of the choroid and yield more precise measurements. Although the same ophthalmologist measured the CT in all subjects in our study, these measurements may contain some errors as they were performed manually. Secondly, we do not know how long the subjects included in our study had experienced VDD. Thirdly, while the subjects included in our study were similar in terms of race (Caucasian), age, gender, refraction, and IOP, the sample size was small. To our knowledge, our study is the first to examine the choroid and RNFL in adult patients with VDD, categorizing them into subgroups representing insufficiency, deficiency, and severe deficiency.

In conclusion, the patients with VDD showed decreased CTs, with the greatest thinning observed in those with severe deficiency. A comprehensive assessment of the choroid is crucial for patients with VDD, and it is important to take into account the possible involvement of VDD in the etiopathogenesis of chorioretinal pathology.

Ethical Approval: *Ethical approval of the study was obtained with the decision of Adıyaman University Clinical Research Ethics Committee (date: 23.06.2022; decision number: 2022-01-02).*

Author Contributions:
Concept: B.Ö
Literature Review: B.Ö, Ö.U
Design : B.Ö, Ö.U
Data acquisition: B.Ö, Ö.U
Analysis and interpretation: B.Ö, Ö.U
Writing manuscript: B.Ö
Critical revision of manuscript: B.Ö, Ö.U
Conflict of Interest: *The authors have no conflicts of interest to declare.*
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References

1. Hilger J, Friedel A, Herr R, Rausch T, Roos F, Wahl DA, et al. A systematic review of vitamin D status in populations worldwide. Br J Nutr. 2014;111(1):23-45.
2. Misra M, Pacaud D, Petryk A, Collett-Solberg PF, Kappy M. Vitamin D Deficiency in Children and Its Management: Review of Current Knowledge and Recommendations. Pediatrics. 2008;122(2):398–417.
3. Norman AW. Sunlight, season, skin pigmentation, vitamin D, and 25-hydroxyvitamin-D:integral components of the vitamin D endocrin system. Am J Clin Nutr 1998;67:1108–10.
4. Holick MF, Binkley NC, Bischoff-Ferrari HA, Gordon CM, Hanley DA, Heaney RP, et al. Evaluation, treatment, and prevention of vitamin D deficiency: an endocrine society clinical practice guideline. J Clin Endocrinol Metab. 2011; 96: 1911–1930.
5. Alsalem JA, Patel D, Susarla R, Coca-Prados M, Bland R, Walker EA, et al. Characterization of vitamin D production by human ocular barrier cells. Invest Ophthalmol Vis Sci. 2014;55(4):2140-7.
6. Reins RY, McDermott AM. Vitamin D: Implications for ocular disease and therapeutic potential. Exp Eye Res. 2015;134:101–10.

7. Seyyar SA, Tokuc EO, Tıskaoğlu NS, Karabaş VL, Güngör K. Do serum vitamin D levels correlate with macular edema or with diabetic retinopathy? Eur J Ophthalmol. 2022;32(6):3592–3598.
8. Kim HT, Kim JM, Kim JH, Lee MY, Won YS, Lee JY, et al. The relationship between vita- min D and glaucoma: a Kangbuk Samsung health study. Korean J. Ophthalmol. 30 (6) (2016) 426–433.
9. Goncalves A, Milea D, Gohier P, Jallet G, Leruez S, Baskaran M, et al. Serum vitamin D status is associated with the presence but not the severity of primary open angle glaucoma. Maturitas. 2015;81(4):470–474.
10. Choi D, Appukuttan B, Binek SJ, Planck SR, Stout JT, Rosenbaum JT, et al. Prediction of cis-regulatory elements controlling genes differentially expressed by retinal and choroidal vascular endothelial cells. J Ocul Biol Dis Infor 2008;1:37–45.
11. Mantell DJ, Owens PE, Bundred NJ, Mawer EB, Canfield AE. 1alpha,25-dihydroxyvitamin D(3) inhibits angiogenesis in vitro and in vivo. Circ Res. 2000;87(3):214–220.
12. Zittermann A, Koerfer R. Protective and toxic effects of vitamin D on vascular calcification: clinical implications. 2008;29(6):423–432
13. Yang L, Ma J, Zhang X, Fan Y, Wang L. Protective role of the vitamin D receptor. Cell Immunol. 2012;279(2):160–166.
14. Wang Y, Chiang YH, Su TP, Hayashi T, Morales M, Hoffer BJ, et al. Vitamin D3 attenuates cortical infarction induced by middle cerebral arterial ligation in rats. Neuropharmacology. 2000;39(5):873–880.
15. Pilz S, Tomaschitz A, März W, Drechsler C, Ritz E, Zittermann A, et al. Vitamin D, cardiovascular disease and mortality. Clin Endocrinol. 2011;75:575–84.
16. Tarcin O, Yavuz DG, Ozben B, Telli A, Ogunc AV, Yuksel M, et al. Effect of vitamin D deficiency and replacement on endothelial function in asymptomatic subjects. J Clin Endocrinol Metab. 2009;94:4023–30.
17. Brewer LC, Michos ED, Reis JP. Vitamin D in atherosclerosis, vascular disease, and endothelial function. Curr Drug Targets 2011;12:54–60.
18. Vural E, Hazar L, Çağlayan M, Şeker Ö, Çelebi ARC. Peripapillary choroidal thickness in patients with vitamin D deficiency. Eur J Ophthalmol. 2021;31(2):578-583
19. Öncül H, Alakus MF, Çağlayan M, Öncül FY, Dag U, Arac E.. Changes in choroidal thickness after vitamin D supplementation in patients with vitamin D deficiency. Can J Ophthalmol. 2020;55(6):486–491.
20. Gürbostan Soysal G, Berhuni M, Özer Özcan Z, Tıskaoğlu NS, Kaçmaz Z. Decreased choroidal vascularity index and subfoveal choroidal thickness in vitamin D insufficiency. Photodiagnosis Photodyn Ther. 2023;44:103767.
21. Kocaay F, Bilen A, Asik A, Çınar ŞŞ, Aydemir E, Aydemir GA, et al. Changes in choroidal tissue post-supplementation with vitamin D in pediatric patients who are deficient in vitamin D. Int Ophthalmol. 2023;43(10):3767-3775.
22. Aydemir E, Ilhan C, Aksoy Aydemir G, Bayat AH, Bolu S, Asik A. Evaluation of Retinal Structure in Pediatric Subjects With Vitamin D Deficiency. Am J Ophthalmol. 2022;233:30-37.
23. Wrzosek M, Łukaszkiewicz J, Wrzosek M, Jakubczyk A, Matsumoto H, Piątkiewicz P, et al. Vitamin D and the central nervous system. Pharmacol Rep. 2013;65(2):271–278.
24. Kalueff AV, Eremin KO, Tuohimaa P. Mechanisms of neuroprotective action of vitamin D3. Biochemistry (Mosc). 2004;69(7):738–741.
25. Ozturk E, Cankaya C. Effect of Vitamin D Deficiency on Contrast Sensitivity Function. Curr Eye Res. 2020;45(12):1619-1624.