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Original Article

Evaluation of Retinal Layers and Choroidal Structures Using Optical Coherence Tomography in Alopecia Areata

Alopesi Areatada Optik Koherens Tomografi Kullanılarak Retina Tabakaları ve Koroid Yapılarının Değerlendirilmesi

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ABSTRACT

Objective: To evaluate the macula, retinal nerve fiber layer (RNFL), retinal layers, and choroidal thickness (CT) using spectral domain optical coherence tomography (SD-OCT) in patients with alopecia areata (AA).

Methods: The right eyes of 42 AA patients (17 women, 25 men) and 42 controls (18 women, 24 men) were included in the study. Each subject underwent thorough ophthalmic examination and SD-OCT (Heidelberg Engineering) measurements. Central macular thickness (CMT), RNFL, the average thicknesses of the ganglion cell layer (GCL), inner plexiform layer (IPL), inner nuclear layer (INL), outer plexiform layer (OPL), outer nuclear layer (ONL), retinal pigment epithelium (RPE), inner retinal layers (IRL), photoreceptor layers (PRL) as well as subfoveal, temporal and nasal CT were measured.

Results: In all sectors, no significant difference was observed between the AA group and the control group with regard to the mean values for CMT and RNFL ($p>0.05$, for all). There was not a significant difference between the AA group and the control group with regard to the thickness of the GCL, IPL, INL, OPL, ONL, RPE, IRL, and PRL ($p>0.05$ for all). CT at the subfoveal, temporal, and nasal regions was significantly thicker in the AA group than in the control group ($p<0.05$ for all).

Conclusions: Along with T-lymphocyte-mediated hair follicle damage, choroidal melanocyte damage and inflammation can also be observed in AA patients. CT may increase secondary to melanocyte inflammation in AA patients.

Keywords: Alopecia areata, choroidal thickness, retinal nerve fiber layer, optical coherence tomography

ÖZ

Amaç: Alopesi areatalı (AA) hastalarda makula, retina sinir lifi tabakası (RSLT), retina tabakaları ve koroid kalınlığını (KK) spektral domain optik koherens tomografi (SD-OKT) ile değerlendirmektir.

Yöntemler: Çalışmaya 42 AA hastasının (17 kadın, 25 erkek) ve 42 kontrol (18 kadın, 24 erkek) grubunun sağ gözü dahil edildi. Her deneğe kapsamlı bir oftalmik muayene ve SD-OKT (Heidelberg Engineering) ölçümleri uygulandı. Santral maküla kalınlığı (SMK), RSLT, gangliyon hücre tabakası (GHT), iç pleksiform tabaka (IPT), iç nükleer tabaka (INT), dış pleksiform tabaka (DPT), dış nükleer tabaka (DNT), retina pigment epiteli (RPE), iç retinal tabaka (IRT), fotoreseptör tabakaları (FRT) ile subfoveal, temporal ve nazal KK ölçüldü.

Bulgular: Tüm kadranlarda SMK ve RSLT ortalama değerleri açısından AA grubu ile kontrol grubu arasında anlamlı bir fark gözlenmedi (tümü için $p>0,05$). GHT, IPT, INT, DPT, DNT, RPE, IRT, FRT kalınlıkları açısından AA grubu ile kontrol grubu arasında anlamlı fark yoktu (tümü için $p>0,05$). AA grubunda subfoveal, temporal ve nazal bölgelerdeki KK kontrol grubuna göre anlamlı olarak daha kalındı (tümü için $p<0,05$).

Sonuçlar: AA hastalarında T-lenfosit aracılı kıl folikülü hasarının yanı sıra koroidal melanosit hasarı ve enflamasyon da görülebilmektedir. AA hastalarında melanosit enflamasyonuna sekonder KK artabilir.

Anahtar kelimeler: Alopesi areata, koroidal kalınlık, retina sinir lifi tabakası, optik koherens tomografi

INTRODUCTION

Alopecia areata (AA) is a skin disease caused by an immune response that affects hair follicles and causes patchy hair loss without scarring[1]. Numerous hypotheses have been proposed for enigmatic pathogenesis, but there appears to be compelling evidence of an autoimmune mechanism mediated by T-cells in individuals with a genetic predisposition[1]. The progression profile of AA is significantly influenced by the T-cell-mediated increase in the follicular plane and upstream pathways[1,2]. Patients of all ages are affected equally, including children[1].

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Ocular abnormalities have been previously reported in patients with AA[2-16]. Cataracts resulting from lens involvement have been reported in AA patients[5-11,16]. Oltulu et al.[12] evaluated the ocular surface via impression cytology and showed a correlation between significant disturbances in conjunctival cytology and tear function with AA. Tosti et al.[14] reported that AA may be linked to the involvement of the retinal pigment epithelium (RPE) via the same mechanism that might harm pigment cells in the skin. It has been hypothesized that melanocyte modification or their participation as a result of subsequent harm causes RPE malfunction[15]. A recent study reported retinal changes such as peripheral drusen, white-without-pressure changes, and peripheral retinal degenerations[16].

There is an ongoing debate regarding the importance and etiopathogenesis of chorioretinal alterations in AA patients. Recent research used spectral domain optical coherence tomography (SD-OCT, software version 6.5.2; Heidelberg Engineering, Heidelberg, Germany) to compare RPE and choroidal thickness (CT) in patients with AA and healthy controls[17]. They emphasized that monitoring CT through SD-OCT may provide perspective into the prognosis of the disease in patients with AA[17]. However, evaluation of the macula, measurement of the thickness of the retinal nerve fiber layer (RNFL), and measurement of the thickness of all retinal layers have never been performed using SD-OCT.

From this perspective, this research planned to assess the macula, thickness of the RNFL, the thickness of all retinal layers as well as CT by SD-OCT in patients with AA.

MATERIALS and METHODS

This observational and prospective clinical study was conducted at the ophthalmology and dermatology departments of a tertiary hospital between July 2021 and July 2022 after receiving approval from the Adıyaman University Clinical Researches Ethics Committee (decision no: 2021-7-1, date: June 11, 2021). Patients were asked to sign informed consent prior to submission. The principles outlined in the Helsinki Declaration served as the basis for the development of the protocol that will be followed in this investigation.

The AA group consisted of patients who were admitted to the hospital after the onset of symptoms, were newly diagnosed, had disease activity confirmed by a positive pull test around the lesion, and had a <50% non-severe Severity of Alopecia Tool score. Healthy people who sought out the ophthalmology clinic for a regular eye exam were randomly assigned to the control group. In the study, data from only the participants' right eyes were collected from each individual. Individuals with poor OCT quality, a history of hypertension, diabetes, pregnancy, known atherosclerotic disease, cigarette use, antihypertensive drug use, neurodegenerative diseases (like Alzheimer's or Parkinson's disease) previous ocular surgery or trauma, glaucoma, choroidal or macular pathology, high refractive error (patients with more than ±3.00 diopters as cycloplegic spherical equivalent), axial length more than 25 mm and patients who suffer from a systemic illness other than AA were not included in this study.

Detailed ophthalmological examination findings and demographic characteristics including sex, age, and occupation was recorded. Before a thorough eye examination, systemic immunosuppressive medications such as steroids were not administered to AA patients. Each subject underwent a thorough ophthalmic examination, which included measuring intraocular pressure with a pneumotonometer, testing best corrected visual acuity with the Snellen chart (6 m), slit lamp biomicroscopy for anterior segment evaluation, dilated fundus examination, and SD-OCT examination.

After pupillary dilation, SD-OCT was used to obtain tomographic images of all patients. Before beginning the SD-OCT procedure, the blood pressure was checked. An ophthalmologist conducted all examinations at the facility, while another ophthalmologist evaluated them both while concealing the patients' diagnoses. To reduce any diurnal variations, OCT measurements for all patients were carried out between 9:00 and 11:00 a.m. The study only used high-quality scans, defined as continuous scan patterns without artifacts or blank spaces, well-focused pictures, and signal strengths of more than 20 (40 being the maximum).

The rapid macular thickness technique involved six radial lines that were spaced 30 degrees apart and had a 6 mm diameter, all centered on the fovea. The fast-macular thickness map was created using a 25-line horizontal raster scan that covered an area of 20 20° and was centered on the fovea. The scans were completed in a high-speed mode thanks to an automated real-time feature. The macular thickness map's concentric rings had widths of 1, 3, and 6 mm for macular scans. The inner and outer macula four quadrant thicknesses were assessed in addition to the central macular thickness (CMT) (Figure 1). The CMT was established as the macular ring center, a 1 mm diameter circle. The inner macula, which has a 3 mm diameter, and the outer macula, which has a 6 mm diameter, are the rings that surround the foveal area.

Figure 1 illustrates the division of the inner and outer rings into four portions that correspond to the nasal, superior, temporal, and inferior. The CMT, outer nasal, outer superior, outer temporal and outer inferior, inner nasal, inner superior, inner temporal, inner inferior macula SD-OCT thickness parameters were calculated. The average thicknesses of the photoreceptor layers, inner retinal layers, RPE, outer nuclear layer, outer plexiform layer, inner nuclear layer, inner plexiform layer, and ganglion cell layer were also measured (Figure 2). Only information from the middle ring was examined and evaluated in the examination of each retinal layer.

The enhanced depth image processing mode of the SD-OCT was used to measure the CT. Using the SD-OCT device measuring line, the distance between the outer border of the RPE and the scleral border at the end of the choroid was manually measured. Measurements were taken from the subfoveal and from 5 different points, nasal [n500 µm (Nasal CT 1), n1000 µm (Nasal CT 2)] and temporal [t500 µm (Temporal CT 1), t1000 µm (Temporal CT 2)] areas from the fovea (Figure 3).

Circular scans with a diameter of 12° were used to obtain circumpapillary RNFL thickness measurements around the optic nerve. The standard summary printout includes the profile of the straightened raw scan in addition to the delineated RNFL boundaries and global mean and average sectoral thickness values for the six regions (nasal, temporal, inferonasal, inferotemporal, superonasal, and superotemporal).

Statistical Analysis

To perform the statistical analyses, IBM SPSS Statistics for Windows 24.0 (IBM Corp., Armonk, NY, USA) were used. The right eye of each subject was considered when conducting the statistical analyses. Descriptive statistics were presented in the form of the mean ± standard deviation (minimum-maximum). The Pearson chi-square test was used in the evaluation of the categorical variables. Student's t-test was used for normally distributed variables in pairwise comparisons of study groups. Statistical significance was set as $p<0.05$.

RESULTS

The study included 42 AA patients (17 women, 25 men) and 42 healthy subjects (18 women, 24 men). The mean age of the subjects was 32.64±8.14 (21-54) and 33.66±10.78 (20-53) years in the AA and control groups, respectively ($p>0.05$). The male-to-female ratio was 17/25 and 18/24 in the AA and control groups, respectively ($p>0.05$). Age, gender, refraction, and axial length measures of the subjects did not differ statistically from one another (Table 1).

The AA and control groups did not exhibit a statistically significant difference in terms of the mean macular thicknesses ($p>0.05$, for each) (Table 2). There was not a significant difference between the AA group and the control group in terms of the thickness of any of the retinal layers ($p>0.05$ for each) (Table 3). There was not a significant difference in the peripapillary RNFL thicknesses between the groups ($p>0.05$ for each) (Table 4). When compared to the control group, subfoveal CT,

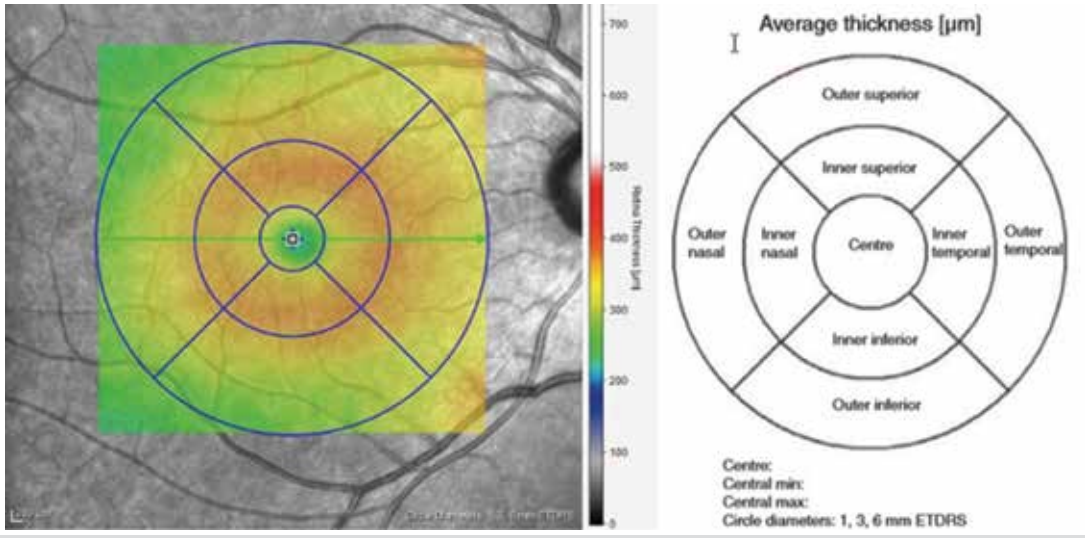

Figure 1. Analyses of macular thickness measurements are displayed. The measurement was carried out in the inner (3 mm) and outer (6 mm) rings surrounding the central macular region, which is 1 mm in diameter. The temporal, nasal, superior, and inferior areas were defined by the division of the inner and outer rings into four quadrants.

nasal CT1, nasal CT2, temporal CT1, and temporal CT2 values were significantly higher in the AA group ($p<0.05$, for each) (Table 4).

DISCUSSION

Ocular findings accompanying AA have been the subject of many studies. Although chorioretinal changes have been reported in AA patients, their detailed evaluation with OCT draws attention as a missing area in the literature[16]. In recent years, with the developments in OCT technology, it has been possible to examine the retina and choroid in detail. From this perspective, we examined retinal and choroidal structures in AA patients. While no difference was observed between the two groups in the macula, RNFL, and retinal layers, we evaluated the choroid as significantly thicker in newly diagnosed AA cases compared with the control group.

Numerous investigations have described retinal abnormalities that occur in AA patients. According to research using fluorescein angiography of the fundus, AA patients experienced retinal changes noticeably frequent than controls did[15]. Recupero et al.[8] found that patients with AA had a statistically significant prevalence of RPE changes compared with controls. Tosti et al.[13] showed a significantly higher frequency of retinal changes in AA patients compared with controls. In another study, retinal function was evaluated electrophysiologically. Electroretinography

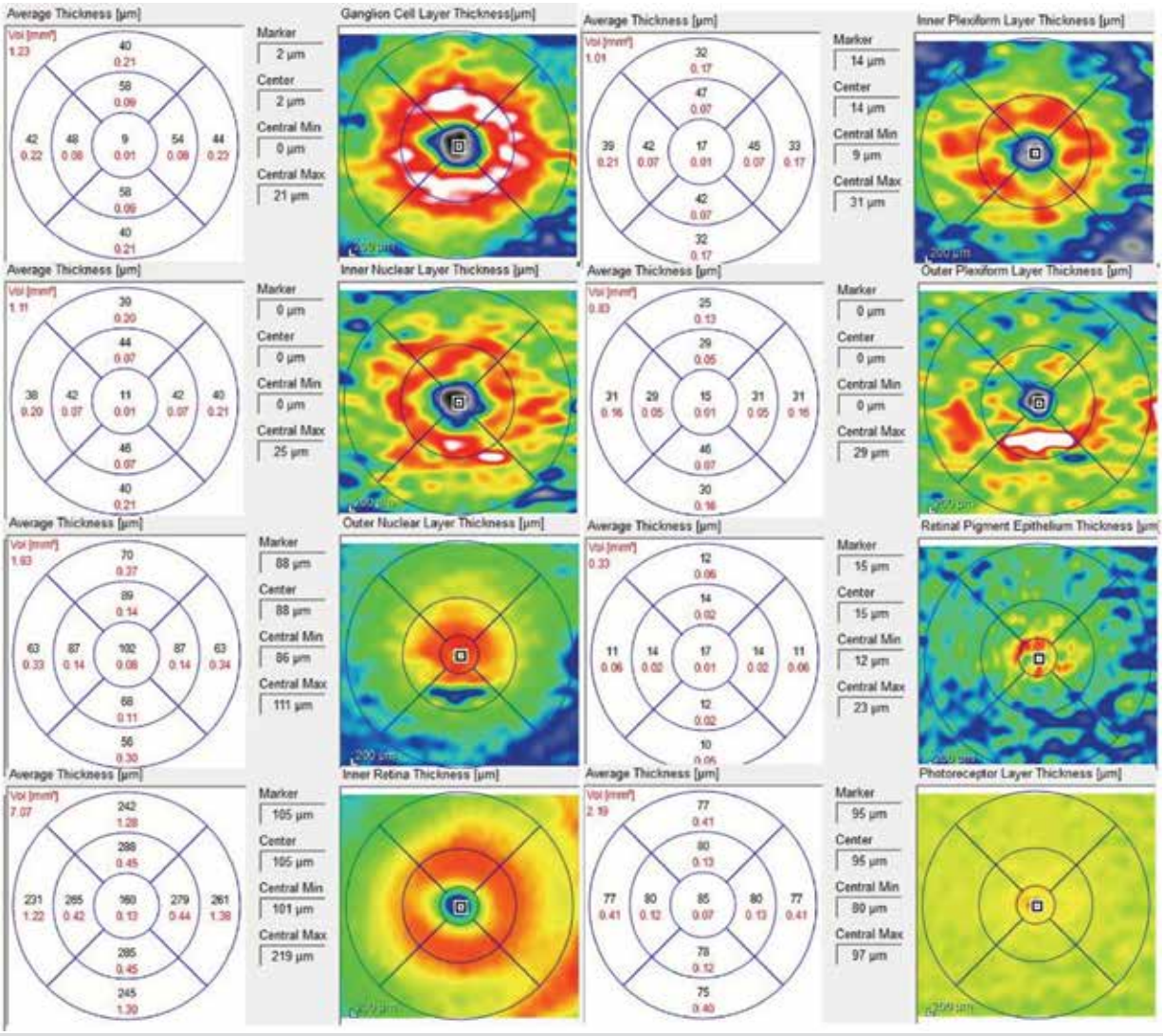

Figure 2. The inner retina layers, the photoreceptor layer, the ganglion cell layer, the inner plexiform layer, the inner nuclear layer, the outer plexiform layer, the outer nuclear layer, and the retinal pigment epithelium layer are all visible in this thickness analysis of optical coherence tomography.

(ERG) was normal in those undergoing ERG, but in electrooculography (EOG) the Arden ratio was significantly reduced in AA patients[14]. A hypothesis was made that RPE malfunction could be indicated by normal photoreceptor function and lowered EOG levels[14]. Strong evidence points to the involvement of T-lymphocyte activity against hair follicle components in AA[18,19]. The RPE may also be predominantly affected by changes in melanocytes or may be implicated as a result of a secondary insult. According to clinical evidence such as damage to the pigment epithelium of the hair and RPE of the retina, this component

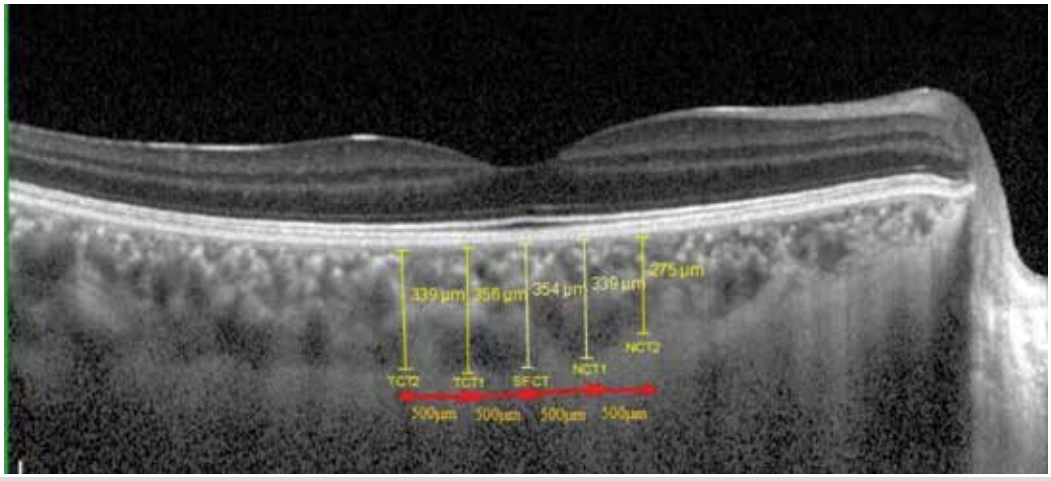

Figure 3. Spectral optical coherence tomography measurement of subfoveal, temporal, and nasal choroidal thickness. From five distinct locations within the fovea and from the fovea to the nasal and temporal areas, measurements were made at 500 m intervals.

Table 1. Demographic characteristics of the groups.

	Alopecia areata (n=42)	Control (n=42)	p-value
Age (year)	32.64±8.14 (21-54)	33.66±10.78 (20–53)	0.625[b]
Male/female	17/25	18/24	0.825[a]
Visual acuity (logMAR)	0.08±0.23	0.06±0.43	0.856[b]
Spherical equivalent (D)	0.07±1.19	0.21±1.15	0.538[b]
Axial length (mm)	23.48±1.05	23.59±1.19	0.875[b]
Intraocular pressure (mmHg)	13.15±1.56	13.51±1.53	0.276[b]

[a]Chi-square test, [b]Student t-test.

Table 2. Comparison of mean macular thicknesses between alopecia areata and control groups in different regions.

	Alopecia areata (n=42)	Control (n=42)	p-value[a]
Central macular thickness (µm)	225.69±13.83	232.97±29.45	0.151
Temporal inner macula (µm)	334.97±20.82	332.78±15.98	0.590
Superior inner macula (µm)	350.16±16.58	347.40±16.43	0.445
Nasal inner macula (µm)	345.04±20.81	347.26±14.99	0.577
Inferior inner macula (µm)	350.11±15.73	346.28±15.21	0.260
Temporal outer macula (µm)	294.21±19.33	289.80±13.76	0.233
Superior outer macula (µm)	303.19±16.24	304.19±12.78	0.755
Nasal outer macula (µm)	315.28±21.75	322.95±14.81	0.063
Inferior outer macula (µm)	294.83±16.28	297.28±14.30	0.466

µm: Micrometer, [a]Student t-test.

may be melanocytes. In our study, we elaborately evaluated the macula, RNFL, and each of the retinal layers separately by SD-OCT. We could not detect any difference in macula and RNFL thickness in patients with AA compared with controls. In addition, we did not find any statistically significant differences between the groups with regard to any of the retinal layers, most notably the thickness of the RPE. Considering that the main pathophysiology in AA is T lymphoid-mediated inflammation against melanocytes, the fact that we were unable to identify any significant difference in thickness between the macula and RNFL can be attributed to the absence of melanocytes in the macula and RNFL. Since the retinal layers other than the RPE do not contain melanocytes, it is expected that there is no difference in these layers. Previous research has demonstrated that under various light frequencies, the RPE related melanosomes in some animal species were displaced. The melanosomes moved to the RPE cells' basal ends in the dark and to their apical protrusions in the light[20,21]. Thus, the amount of melanin pigment and its position and distribution in RPE may impact OCT images[22]. This may be the reason why there was no difference in RPE between the groups. In the follow-up

Table 3. Comparison of mean retinal layer thicknesses between alopecia areata and control groups.

	Alopecia areata (n=42)	Control (n=42)	p-value[a]
Ganglion cell layer (µm)	15.97±3.82	17.92±6.51	0.098
Inner plexiform layer (µm)	21.78±3.10	22.47±3.71	0.358
Inner nuclear layer (µm)	18.57±5.85	20.45±5.27	0.126
Outer plexiform layer (µm)	24.23±7.10	25.28±6.41	0.480
Outer nuclear layer (µm)	90.64±9.38	89.19±10.51	0.506
Retinal pigment epithelium (µm)	16.57±1.93	16.69±1.70	0.766
Inner retinal layers (µm)	178.57± 22.28	187.14±21.11	0.074
Photoreceptor layers (µm)	86.83±3.32	86.50±2.57	0.609

µm: Micrometer, [a]Student t-test.

Table 4. Comparison of peripapillary nerve fiber layer and choroidal thicknesses between alopecia areata and control groups.

	Alopecia areata (n=42)	Control (n=42)	p-value[a]
Global RNFL	100.90±7.87	101.26±7.50	0.832
Nasal RNFL	75.33±15.40	73.54±11.24	0.546
Nasal superior RNFL	118.57±16.98	118.8±25.33	0.159
Temporal superior RNFL	139.38±19.74	140.88±14.27	0.691
Temporal RNFL	71.71±8.94	74.14±9.84	0.240
Temporal inferior RNFL	147.93±18.89	153.51±16.87	0.156
Nasal inferior RNFL	115.23±15.80	113.35±24.26	0.675
Subfoveal CT (µm)	361.76±66.63	323.92±87.79	**0.029**
Nasal CT 1 (µm)	351.28±66.95	313.19±86.09	**0.026**
Nasal CT 2 (µm)	343.45±64.07	287.38±84.97	**0.001**
Temporal CT 1 (µm)	355.14±70.40	318.69±84.09	**0.034**
Temporal CT 2 (µm)	356.80±70.28	299.07±90.10	**0.002**

RNFL: Retinal nerve fiber layer, CT: Choroidal thickness, µm: Micrometer

[a]Student t-test.

Bold values indicate p<0.05.

Nasal CT 1: 500 micrometers nasal to the fovea for choroidal thickness

Nasal CT 2: 1000 micrometers nasal to the fovea for choroidal thickness

Temporal CT 1: 500 micrometers temporal to the fovea for choroidal thickness

Temporal CT 2: 1000 micrometers temporal to the fovea for choroidal thickness

of newly diagnosed patients, a change in RPE thickness may be observed with an increase in inflammation. To determine this, studies involving the long-term follow-up of this patient group will be needed.

One of the body's tissues with maximum vascularization, the choroid, which covers the outer retina. The retina is nourished and oxygenated by this tissue which also controls its temperature[17]. The stroma and suprachoroidal layer of the choroid contain vascular formations, smooth muscle cells, fibroblasts, collagen and elastic fibers, and melanocytes. Previous studies suggested that systemic diseases and dermatological conditions may have influenced choroid circulation[23]. In studies evaluating the choroid in vitiligo and albinism patients, choroidal thinning due to the absence of melanocytes has been reported[24,25]. Since the inflammatory process is chronic in vitiligo with melanocyte loss, a decrease in CT has been reported. Therefore, despite the presence of melanocyte damage, the CT of vitiligo patients is not expected to increase[26]. Patients with psoriasis were found to have thicker choroid than healthy controls in a previous study. According to the results of this study, elevated levels of pro-inflammatory cytokines are linked to increase in CT in these patients[23]. A study that evaluated CT in AA patients reported no significant difference between AA and controls[17]. However, they emphasized that patients with poor prognostic criteria had significantly thinner CT scans than other patients[17]. In our study, unlike the other studies, CT was considerably higher in every location among the AA group versus the control group. Recent research into the pathogenesis of AA has highlighted the significance of cytokine release at both systemic and local levels. Higher levels of cytokines such as IL-6, IL-5, and IL-2 were found in serum of AA patients compared to those of healthy controls[27]. Therefore, we proposed that the increase in CT in AA patients may be due to inflammation caused by T-lymphocyte activity toward components of hair follicles as well as choroidal melanocytes. We attribute this result to the same mechanism of CT increase seen in Vogt Kayanagi Harada disease, which occurs because of the development of inflammation against melanocytes[28]. The fact that our patients were newly diagnosed and at the beginning of the disease may explain the inflammatory-induced CT increase. CT may change in the long-term follow-up of AA patients. However, long-term follow-up and histopathological studies are needed to clarify this question. A number of factors, including age, axial length, and refractive errors, can impact CT of the eye[29,30]. In our study, no discernible difference existed between the patient and control groups with respect to such potential CT-affecting factors. According to some researches, hormone status and gender may affect choroidal blood flow and cause changes in CT[31,32]. There was no significant gender difference between the AA and control groups. However, we did not investigate the amounts of androgen found in the serum of these individuals. This is a limitation associated with our investigation.

To the best of our knowledge, our study is the first to use SD-OCT to examine the macula, RNFL, and all retinal layers in AA patients. Our study has some shortcomings due to its limited sample size and the absence of software for automatic choroidal measurement, which cause some errors depending on the person performing the measurement.

CONCLUSION

It was discovered that the CT of AA patients had a thicker structure than the control group. Along with T-lymphocyte-mediated hair follicle damage, choroidal melanocyte damage and inflammation can also be observed in AA patients. Therefore, AA patients should be closely monitored by ophthalmologists for possible posterior ocular segment disorders.

Ethics

Ethics Committee Approval: Ethical approval for the research was obtained from the Clinical Researches Ethics Committee of Adıyaman University, approval date: June 11, 2021, decision number: 2021-7-1. The study was conducted in accordance with the Declaration of Helsinki.

Informed Consent: Written informed consent was obtained from the volunteers.

Peer-review: Externally and internally peer-reviewed.

Author Contributions

Surgical and Medical Practices: S.D., Concept: B.O., Design: B.O., Data Collection and/or Processing: S.D., H.K., Analysis and/or Interpretation: G.A.A., Literature Search: S.D., H.K., Writing: B.O.

Conflict of Interest: The authors have no conflict of interest to declare.

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