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One-Year Follow-up of Retinal Nerve Fiber Layer Changes in Fibromyalgia Patients: A Retrospective Cohort Study

Fibromiyalji Hastalarında Retina Sinir Lif Tabakası Değişikliklerinin Bir Yıllık Takibi: Retrospektif Kohort Çalışması

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ABSTRACT

OBJECTIVE: To retrospectively evaluate retinal nerve fiber layer (RNFL) changes over one year in fibromyalgia (FM) patients compared to healthy controls, assess correlations between clinical parameters and RNFL measurements, and investigate potential treatment-related effects.

MATERIALS and METHODS: This retrospective cohort study analyzed medical records of 44 female FM patients and 44 age-matched healthy controls who underwent serial optical coherence tomography (OCT) examinations. RNFL thickness measurements obtained using Canon-OCT HS100 at baseline and one-year follow-up were extracted from the database. Clinical data including Visual Analog Scale (VAS), Fibromyalgia Impact Questionnaire (FIQ), disease duration, and medication use were collected from patient records. Statistical analyses included Mann-Whitney U tests, Wilcoxon signed-rank tests, Kruskal- Wallis tests, and Spearman correlations.

RESULTS: No significant baseline differences in RNFL parameters were observed between groups. However, FM patients showed significantly greater RNFL thinning in nasal (Δ: -1.51±1.01 vs - 0.74±0.66 µm, p<0.001) and superior (Δ: -2.49±1.66 vs -1.09±1.08 µm, p<0.001) sectors compared to controls. Disease duration correlated positively with average (r=0.308, p=0.042) and temporal (r=0.342, p=0.023) RNFL change. Pregabalin users showed greater RNFL reduction in nasal (p=0.016) and inferior (p=0.031) sectors.

CONCLUSION: FM patients demonstrate accelerated RNFL thinning over one year, particularly in nasal and superior sectors. Disease duration influences this progression. These findings may be consistent with the hypothesis of ongoing neurodegenerative processes in FM, though the small effect sizes warrant cautious interpretation. The results highlight OCT as a potential monitoring tool.

KEYWORDS: Fibromyalgia, retinal nerve fiber layer, optical coherence tomography, neurodegeneration

ÖZ

AMAÇ: Fibromiyalji (FM) hastalarında sağlıklı kontrol grubuyla karşılaştırmalı olarak bir yıl boyunca RNFL değişikliklerini retrospektif olarak değerlendirmek, klinik parametreler ile retinal sinir lifi tabakası (RNFL) ölçümleri arasındaki korelasyonları değerlendirmek ve potansiyel tedaviye bağlı etkileri araştırmak.

GEREÇ ve YÖNTEM: Bu retrospektif kohort çalışmada, seri optik koherens tomografisi (OCT) muayeneleri yapılan 44 kadın FM hastası ve 44 yaş eşleştirilmiş sağlıklı kontrolün tıbbi kayıtları analiz edildi. Başlangıçta ve bir yıllık takipte Canon-OCT HS100 kullanılarak elde edilen RNFL kalınlığı ölçümleri veri tabanından çıkarıldı. Görsel Analog Ölçeği (VAS), Fibromiyalji Etki Anketi (FIQ), hastalık süresi ve ilaç kullanımı gibi klinik veriler hasta kayıtlarından toplandı. İstatistiksel analizler Mann-Whitney U testleri, Wilcoxon işaretli sıra testleri, Kruskal-Wallis testleri ve Spearman korelasyonlarını içeriyordu.

BULGULAR: Gruplar arasında RNFL parametrelerinde başlangıçta önemli bir fark gözlenmedi. Ancak, FM hastaları, kontrollere kıyasla nazal (Δ: -1,51±1,01 vs -0,74±0,66 µm, p<0,001) ve superior (Δ: -2,49±1,66 vs -1,09±1,08 µm, p<0,001) sektörlerde anlamlı olarak daha fazla RNFL incelmesi gösterdi. Hastalığın süresi, ortalama (r=0,308, p=0,042) ve temporal (r=0,342, p=0,023) RNFL değişikliği ile pozitif korelasyon gösterdi. Pregabalin kullanıcıları, nazal (p=0,016) ve inferior (p=0,031) sektörlerde daha fazla RNFL azalması gösterdi.

SONUÇ: FM hastaları, özellikle nazal ve superior sektörlerde, bir yıl boyunca hızlanan RNFL incelmesi göstermiştir. Hastalık süresi bu ilerlemeyi etkilemektedir. Bu bulgular, FM'de devam eden nörodejeneratif süreçler hipotezi ile uyumlu olabilir; ancak küçük etki büyüklükleri dikkatli yorumlanmayı gerektirmektedir. Bu sonuçlar OCT'yi potansiyel bir izleme aracı olarak öne çıkarmaktadır.

ANAHTAR KELİMELER: Fibromiyalji, retina sinir lifi tabakası, optik koherens tomografi, nörodejenerasyon

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INTRODUCTION

Fibromyalgia (FM) is a chronic pain disorder that affects about 2-4% of the general population, with a significantly higher prevalence in females (1). It is characterized by widespread musculoskeletal pain, fatigue, sleep disturbances, and cognitive issues, which can make diagnosis and treatment difficult (1,2). Although the exact cause of FM remains unknown, it is increasingly thought to involve central nervous system (CNS) sensitization, altered pain processing, and neuroinflammatory mechanisms (3). Recent advances in neuroimaging have uncovered structural and functional brain abnormalities in FM patients, including reductions in gray matter volume within pain-processing areas(1,3). The retina, as an accessible extension of the central nervous system, provides a unique opportunity to assess neurodegenerative changes through optical coherence tomography (OCT) (4–6). Cross-sectional studies have shown retinal nerve fiber layer (RNFL) thinning in FM patients compared to healthy controls, indicating subclinical axonal damage (7). Garcia-Martin et al. (8) first reported significant RNFL thinning in FM patients, especially in the temporal sectors, with more notable changes in specific FM subtypes. Later studies have confirmed these findings and provided additional evidence of ganglion cell layer involvement (6,9,10). However, the current literature is mostly cross-sectional, which limits our understanding of RNFL changes over time and how they relate to disease features and treatment. The relationship between FM medications and RNFL changes remains poorly understood. Pregabalin, an alpha-2-delta calcium channel ligand, and duloxetine (SNRI) are among the most prescribed medications for FM. While these drugs provide symptomatic relief, their potential effects on retinal structure have not been systematically investigated. One study suggested that long-term pregabalin use might be associated with RNFL thinning, but longitudinal data are limited (11). This retrospective cohort study aimed to evaluate one-year RNFL thickness changes in FM patients compared to healthy controls using existing clinical data. It also explored the relationships between clinical parameters (disease duration, severity, pain) and RNFL measurements, as well as examined whether different treatment regimens are linked to varying RNFL changes.

MATERIALS and METHODS

This retrospective cohort study was carried out by reviewing medical records and OCT databases at Uşak Training and Research Hospital. The study protocol received approval from the local ethics committee (approval number: 1077-1077-06) and was performed in accordance with the Declaration of Helsinki.

We identified patients from the rheumatology clinic database who had been diagnosed with FM according to the 2016 American College of Rheumatology criteria and had undergone serial OCT examinations approximately one year apart as part of routine clinical care. Age-matched healthy controls were selected from the ophthalmology database among individuals who had undergone routine eye examinations during the same period. Medical records of 44 female patients with FM diagnosis who had complete OCT data at two time points, baseline and approximately one-year follow-up, were included. Forty-four age-matched healthy female subjects with similar serial OCT examinations served as controls. Inclusion criteria for FM patients included: age 18-60 years, confirmed FM diagnosis in the medical record, and availability of two OCT examinations approximately 12 months apart. Exclusion criteria included: documented history of ocular disease (glaucoma, macular degeneration, optic neuropathy), diabetes mellitus, uncontrolled hypertension, neurodegenerative diseases, refractive error >6 diopters, and previous ocular surgery as recorded in the medical history.

Clinical data were collected from electronic medical records. Demographic information included age, sex, body mass index (BMI), marital status, education level, smoking status, and alcohol use. Disease-related data covered disease duration at baseline, Visual Analog Scale (VAS) pain scores, and Fibromyalgia Impact Questionnaire (FIQ) scores recorded closest to the baseline OCT exam. Medication data, such as use of SNRI (duloxetine), pregabalin, and tricyclic antidepressants (TCA), were documented based on prescriptions at baseline. Cardiometabolic comorbidities were identified from the problem list in the medical records.

RNFL thickness measurements were obtained from the OCT database. All examinations were performed using the Canon OCT-HS100 (Ver. 4.5.3, Canon, Japan) as part of routine clinical practice. The peripapillary RNFL thickness in four quadrants (superior, inferior, nasal, and temporal), the average RNFL thickness, and the RNFL symmetry index were recorded. Data from the right eye of each participant were used for analysis. Examinations with signal strength less than 6 were

excluded. The change in RNFL thickness (delta) was calculated as the difference between the one-year follow-up and baseline measurements.

Statistical Analyses

Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY). Continuous variables were tested for normality with the Shapiro-Wilk test. Variables with a normal distribution are presented as mean±standard deviation, while non-normally distributed variables are shown as median (interquartile range). Between-group differences were analyzed using either the independent samples t-test or the Mann-Whitney U test, as appropriate. Changes within groups over time were assessed with paired t-tests or Wilcoxon signed-rank tests. Correlations between clinical parameters and RNFL measurements were evaluated using Pearson or Spearman correlation coefficients. Multiple group comparisons were conducted with the Kruskal-Wallis test. A p-value<0.05 was considered statistically significant. No formal correction for multiple comparisons (e.g., Bonferroni or false discovery rate) was applied, given the exploratory nature of the secondary analyses; however, primary between-group RNFL change comparisons yielding p<0.001 would remain significant even under conservative correction thresholds.

RESULTS

The study involved 88 female participants, with 44 having FM and 44 healthy controls. The FM group had an average age of 45.09±5.07 years. The mean BMI was 27.37 ±4.59 kg/m². Most were married (86.4%), and 54.5% had only completed primary school. Smoking was reported by 43.2%, and alcohol consumption by 4.5%. Cardiometabolic comorbidities were found in 43.2% of FM patients (Table 1). The mean disease duration was 15.34±17.34 months (median: 12.0 months). The average VAS score was 6.77±2.14, and the average FIQ score was 62.86±20.17, indicating a moderate to severe impact of the disease. Regarding treatment, 63.6% were on SNRI (duloxetine), 31.8% on pregabalin, and 11.4% on TCA. The most common treatment pattern was SNRI monotherapy (54.5%), followed by pregabalin monotherapy (29.5%).

Table 1. *Demographic and clinical characteristics of fibromyalgia patients (n=44)*

Characteristic	Value
Age (years), mean±SD	45.09±5.07
BMI (kg/m²), mean±SD	27.37±4.59
Disease duration (months), median (IQR)	12.0 (5.0-16.2)
VAS score, mean±SD	6.77±2.14
FIQ score, mean±SD	62.86±20.17
Married, n (%)	38 (86.4%)
Smoker, n (%)	19 (43.2%)
Cardiometabolic comorbidity, n (%)	19 (43.2%)
SNRI (duloxetine) use, n (%)	28 (63.6%)
Pregabalin use, n (%)	14 (31.8%)
TCA use, n (%)	5 (11.4%)

BMI: Body mass index, FIQ: Fibromyalgia Impact Questionnaire, IQR: Interquartile range, SD: Standard deviation, SNRI: Serotonin-nor-epinephrine reuptake inhibitör, TCA: Tricyclic antidepressant, VAS: Visual Analog Scale

At baseline, there were no statistically significant differences in RNFL thickness between FM patients and healthy controls across any sector (Table 2). The average RNFL thickness was 91.89±10.51 µm in FM patients compared to 92.93±12.66 µm in controls (p=0.528). Likewise, the temporal (p=0.262), nasal (p=0.950), superior (p=0.616), and inferior (p=0.174) sectors showed no significant differences between the groups.

Table 2. Baseline RNFL measurements: FM patients vs. healthy controls

RNFL sector (µm)	FM (n=44)	Controls (n=44)	p-value
Average	91.89±10.51	92.93±12.66	0.528
Temporal	77.70±8.36	79.59±7.26	0.262
Nasal	76.89±8.93	77.07±9.71	0.950
Superior	118.61±15.18	117.00±14.89	0.616
Inferior	112.14±19.22	117.25±16.52	0.174
Symmetry	0.78±0.09	0.80±0.08	0.385

Values are mean±SD. p-values from Mann-Whitney U test or independent t-test as appropriate
RNFL: Retinal nerve fiber layer

Both groups exhibited significant RNFL thinning across all sectors over one year (all p<0.001, Wilcoxon signed-rank test), consistent with expected age-related decline. However, the extent of change varied significantly between groups in specific sectors (Table 3). FM patients showed significantly more RNFL thinning compared to controls in the nasal sector (Δ: -1.51±1.01 vs - 0.74±0.66 µm, p<0.001) and superior sector (Δ: -2.49±1.66 vs -1.09±1.08 µm, p<0.001). A trend toward greater thinning was seen in the temporal sector (Δ: -1.59±1.13 vs -1.15±0.88 µm, p=0.061). There were no significant differences in average RNFL change and inferior sector change between groups.

Table 3. *One-year RNFL changes (delta): FM patients vs. healthy controls*

RNFL sector (µm)	FM (n=44)	Controls (n=44)	p-value
Δ Average	-1.75±1.39	-1.36±1.20	0.217
Δ Temporal	-1.59±1.13	-1.15±0.88	0.061
Δ Nasal	-1.51±1.01	-0.74±0.66	<0.001*
Δ Superior	-2.49±1.66	-1.09±1.08	<0.001*
Δ Inferior	-2.56±1.68	-2.71±1.71	0.735
Δ Symmetry	+0.01±0.11	-0.01±0.11	0.372

Values are mean±SD. *p<0.05 (Mann-Whitney U test). Δ=Change from baseline to 1-year follow-up.
RNFL: Retinal nerve fiber layer

In the FM group, disease duration showed significant positive correlations with the average RNFL change (r=0.308, p=0.042) and in the temporal sector (r=0.342, p=0.023), suggesting that longer disease duration was linked to less RNFL thinning (Table 4). At baseline, age was positively correlated with the temporal RNFL (r=0.398, p=0.007), and BMI was positively correlated with the superior RNFL (r=0.335, p=0.026). VAS and FIQ scores showed no significant correlations with RNFL measurements or changes. No significant differences in baseline RNFL measurements or RNFL changes were observed among the three main treatment groups (SNRI, pregabalin, TCA) using the Kruskal-Wallis test (all p>0.05). However, when comparing pregabalin users (n=14) to non-users (n=30) within the FM group, pregabalin users showed significantly greater RNFL thinning in the nasal sector (Δ: -1.96±1.01 vs -1.30±0.96 µm, p=0.016) and the inferior sector (Δ: -3.28±1.36 vs -2.22±1.73 µm, p=0.031) (Table 5).

Table 4. *Significant correlations between clinical parameters and RNFL in FM patients*

Variables	r	p-value	Method
Age vs. RNFL-temporal (baseline)	0.398	0.007	Pearson
BMI vs. RNFL-superior (baseline)	0.335	0.026	Pearson
Disease duration vs. Δ RNFL-average	0.308	0.042	Spearman
Disease duration vs. Δ RNFL temporal	0.342	0.023	Spearman
Only statistically significant correlations (p<0.05) are shown. Δ=Change from baseline to follow-up.			
FM: Fibromyalgia			

Table 5. *RNFL changes by pregabalin use in FM patients*

RNFL change (μm)	Pregabalin users (n=14)	Non-users (n=30)	p-value
Δ Average	-2.12±1.51	-1.58±1.32	0.187
Δ Temporal	-1.82±1.20	-1.48±1.11	0.330
Δ Nasal	-1.96±1.01	-1.30±0.96	0.016*
Δ Superior	-2.34±1.78	-2.56±1.63	0.622
Δ Inferior	-3.28±1.36	-2.22±1.73	0.031*
Values are mean±SD. *p<0.05 (Mann-Whitney U test).			
RNFL: Retinal nerve fiber layer			

DISCUSSION

This retrospective cohort study provides preliminary evidence suggesting accelerated RNFL thinning in FM patients over a year. While initial RNFL measurements were similar between FM patients and healthy controls, the decline rate was notably higher in FM patients, especially in the nasal and superior sectors. These results may be consistent with the hypothesis of ongoing neurodegenerative processes in FM and contribute to the growing research on retinal structural changes associated with this condition.

The lack of significant initial differences in RNFL thickness between groups contrasts with several previous cross-sectional studies that reported RNFL thinning in FM patients. Garcia-Martin et al. (8) demonstrated notable RNFL thinning in FM patients, particularly in the temporal sectors. However, our results align with more recent studies that found no significant vascular or neurodegenerative changes in FM patients at baseline (6,7,9,10,12,13). This discrepancy may be

due to differences in disease duration, severity, or patient selection. Our group had a median disease duration of 12 months, indicating an early stage of the disease.

The discovery of accelerated RNFL thinning over time in FM patients has important implications. The significantly greater decline in the nasal (2.04-fold) and superior (2.29-fold) sectors compared to controls suggests sector-specific vulnerability that may reflect underlying pathophysiological mechanisms. The nasal quadrant mainly consists of papillomacular bundle fibers that project to the temporal retina, while the superior sector receives fibers from the superior retina. The selective involvement of these sectors may suggest differential susceptibility to possible neurodegenerative processes in FM, although alternative explanations, including subclinical vascular or inflammatory mechanisms, cannot be excluded.

It should be noted that the statistically significant RNFL differences observed between groups were relatively small in magnitude (about 1–2 μm). Studies evaluating the reproducibility of SD- OCT RNFL measurements have reported test-retest variability ranging from approximately 1.8 to 4.5 μm, depending on the sector and instrument used. While the between-group differences in our study exceeded the coefficient of variation reported for most SD-OCT devices, the overlap between measurement variability and observed effect sizes calls for cautious interpretation. Future studies with larger sample sizes and longer follow-up periods will be

necessary to determine whether these subclinical differences lead to meaningful retinal dysfunction. Additionally, since multiple RNFL sectors and several correlation analyses were tested, the potential for increased type I error due to multiple comparisons should be acknowledged. Although our primary findings in the nasal and superior sectors remained significant at very conservative thresholds ($p<0.001$), some secondary findings (e.g., pregabalin subgroup analyses and individual correlations) should be viewed as exploratory and hypothesis-generating rather than definitive.

The positive correlation between disease duration and RNFL change, with less thinning over longer disease duration, is counterintuitive and needs careful interpretation. One possible explanation is that patients with longer disease duration may have already experienced significant RNFL loss, reaching a floor effect that prevents further measurable decline. Alternatively, this result could reflect survivor bias, where patients with stable retinal structure are more likely to continue follow-up. This observation warrants further investigation in larger longitudinal cohorts with extended follow-up periods.

The exploratory analysis of pregabalin use and RNFL thinning yielded notable findings that should be interpreted with caution given the limited sample size of the pregabalin subgroup (n=14). Pregabalin, an alpha-2-delta calcium channel ligand, is a first-line treatment for FM that decreases glutamate release and reduces neuronal hyperexcitability. Biçer et al. (11) previously reported that longer duration of pregabalin use was connected to RNFL thinning in FM patients. Our results are broadly consistent with this observation, with pregabalin users showing notably greater RNFL decline in the nasal and inferior sectors. The exact mechanism behind this association remains unknown. Possible explanations include direct effects on retinal neurons, selection bias, or confounding by indication. However, due to the small sample size of pregabalin users, these findings should be regarded as exploratory and hypothesis-generating; no causal relationship can be established from these data. Importantly, this should not discourage clinicians from prescribing pregabalin when necessary, but it suggests that eye monitoring may be wise for long-term users, especially those with pre-existing retinal conditions.

The absence of a link between VAS/FIQ scores and RNFL measurements indicates that retinal structural changes might occur independently of perceived symptom severity. This discrepancy between subjective symptoms and objective structural markers aligns with the idea that FM involves multiple underlying mechanisms, with central sensitization likely playing a larger role in pain perception while peripheral neurodegenerative processes appear as measurable tissue changes (1,2).

Several mechanisms could explain the accelerated RNFL loss observed in FM patients. Small fiber neuropathy (SFN) has been identified in a significant number of FM patients, showing reduced intraepidermal nerve fiber density on skin biopsies. The retinal nerve fibers originate from the same embryological source as peripheral nerves, and similar pathological processes may impact both tissues. Additionally, central sensitization and neuroinflammation may promote retinal neuronal damage through the release of inflammatory mediators and oxidative stress. Autonomic dysfunction, often linked to FM, might also influence retinal perfusion and neuronal health (4,7,14).

Study Limitations

This study has several limitations due to its retrospective design. First, data were gathered from existing medical records, which might have incomplete documentation of clinical variables and possible confounders. Second, the timing of OCT exams was based on clinical practice rather than a standardized protocol, potentially causing variability in follow-up intervals. Third, medication dosages and adherence could not be confirmed, and changes in treatment during follow-up were not systematically recorded. Fourth, selecting patients with available serial OCT data may have introduced selection bias, as those with more consistent follow-up might differ systematically from those with irregular attendance. Fifth, the primarily female cohort limits the applicability of findings to male patients. Sixth, although the sample size was sufficient to detect significant differences, it might have limited power for subgroup analyses. Finally, the observational nature of the study prevents making causal claims about treatment effects on RNFL. Importantly, our analyses were predominantly based on univariate comparisons, and a multivariate regression model adjusting for potential confounders such as age, BMI, smoking status, and cardiometabolic comorbidities was not performed. Although we found no significant baseline differences in RNFL between groups and the within-subject longitudinal design partially controls for stable individual characteristics, the lack of multivariate adjustment limits our ability to isolate the

independent contribution of FM to RNFL change. Additionally, detailed clinical characteristics of the control group (e.g., BMI, smoking status, comorbidities) were not systematically documented, which precludes a comprehensive between-group comparison of potential confounding variables. Future prospective studies should incorporate multivariate models to better account for these factors.

Despite these limitations, the study provides valuable real-world evidence of RNFL changes in FM patients over time. The use of routine clinical data enhances the external validity of our findings and reflects typical patient populations encountered in clinical practice.

Future prospective studies should incorporate standardized follow-up intervals, larger sample sizes, thorough medication exposure assessments, and combine functional measures (e.g., visual field testing) with structural OCT parameters. Examining ganglion cell layer changes, retinal vascular parameters using OCT angiography, and their correlations with brain imaging results would offer a more complete understanding of neuro-ophthalmic involvement in FM.

CONCLUSION

This retrospective cohort study suggests that FM patients experience faster RNFL thinning over one year compared to healthy controls, especially in nasal and superior sectors. Disease duration and pregabalin use are linked to RNFL changes, while symptom severity has no correlation with retinal structural measures. These results may point to possible neurodegenerative processes in FM and indicate that OCT could be a useful, non-invasive tool for monitoring. However, the observed RNFL changes are small in magnitude and approach the limits of OCT measurement variability, necessitating cautious interpretation. Longer-term prospective studies are needed to confirm these findings and explore the clinical significance of retinal changes in FM.

Ethics: This retrospective cohort study was carried out by reviewing medical records and OCT databases at Uşak Training and Research Hospital. The study protocol received approval from the local ethics committee (approval number: 1077-1077-06) and was performed in accordance with the Declaration of Helsinki.

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Author contribution status; The concept of the study; IMUB, design; IMUB, AB. literature review; AB. collecting and processing data; IMUB, AB. statistics; IMUB. writing phase; IMUB, AB.

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REFERENCES

1. Sarzi-Puttini P, Giorgi V, Marotto D, Atzeni F. Fibromyalgia: an update on clinical characteristics, aetiopathogenesis and treatment. Nat Rev Rheumatol. 2020; 16: 645-660. doi: 10.1038/s41584-020-00506-w
2. García-Domínguez M. Fibromyalgia and inflammation: unrevealing the connection. Cells. 2025; 14: 271. doi: 10.3390/cells14040271
3. Martínez-Lavín M. Fibromyalgia and small fiber neuropathy: the plot thickens! Clin Rheumatol. 2018; 37: 3167-3171. doi: 10.1007/s10067-018-4300-2
4. Bambo MP, Garcia-Martin E, Gutierrez-Ruiz F, Magallon R, Roca M, Garcia-Campayo J, et al. Study of perfusion changes in the optic disc of patients with fibromyalgia syndrome using new colorimetric analysis software. J Fr Ophtalmol. 2015; 38: 580-587. doi: 10.1016/j.jfo.2015.01.010
5. Sevimli N, Kurna SA, Şilte Karamanlıoğlu AD, Ünlü Özkan F, Aktaş İ, Çakır M. Characteristics of the retina and choroid in fibromyalgia patients and their correlation with disease severity and quality of life. Photodiagnosis Photodyn Ther. 2023; 44: 103819. doi: 10.1016/j.pdpdt.2023.103819
6. Boquete L, Vicente MJ, Miguel-Jiménez JM, Sánchez-Morla EM, Ortiz M, Satue M, et al. Objective diagnosis of fibromyalgia using neuroretinal evaluation and artificial intelligence. Int J Clin Health Psychol. 2022; 22: 100294. doi: 10.1016/j.ijchp.2022.100294
7. Öztürk GY, Emekli DT, Sahutoglu E, Kocyigit BF. Evaluation of ophthalmic vascular and neuroretinal alterations in fibromyalgia syndrome: a cross-sectional comparative study. Rheumatol Int. 2024; 44: 1757-1765. doi: 10.1007/s00296-024-05662-w
8. Garcia-Martin E, Garcia-Campayo J, Puebla-Guedea M,

Ascaso FJ, Roca M, Gutierrez-Ruiz F, et al. Fibromyalgia is correlated with retinal nerve fiber layer thinning. PLoS One. 2016; 11: e0161574. doi: 10.1371/journal.pone.0161574

9. Cordón B, Orduna E, Viladés E, Garcia-Martin E, Garcia-Campayo J, Puebla-Guedea M, et al. Analysis of retinal layers in fibromyalgia patients with premium protocol in optical tomography coherence and quality of life. Curr Eye Res. 2022; 47: 143-153. doi: 10.1080/02713683.2021.1951301
10. Garcia-Martin E, Tello A, Vilades E, Perez-Velilla J, Cordon B, Fernandez-Velasco D, et al. Diagnostic ability and capacity of optical coherence tomography-angiography to detect retinal and vascular changes in patients with fibromyalgia. J Ophthalmol. 2022; 2022: 3946017. doi: 10.1155/2022/3946017
11. Biçer GY, Zor KR, Biçer KE, Küçük E, Küçük EB. Can the long term using of pregabalin in fibromyalgia affect the choroid and retinal nerve fiber layer? Eye (Lond). 2022; 36: 2318-2322. doi: 10.1038/s41433-021-01864-9
12. Urfalıoglu S, Berk E. Assessment of retinal nerve fiber thickness and optic nerve head blood flow in female patients diagnosed with fibromyalgia syndrome. Arq Bras Oftalmol. 2022; 85: 30-36. doi: 10.5935/0004-2749.20220005
13. Gezginaslan TA, Limon U, Kaleoğlu Ö, Saygın lÖ, Bozkurt E, Akçay BİS. Evaluation of optical coherence tomography and optical coherence tomography angiography findings of fibromyalgia. Int Ophthalmol. 2024; 45: 12. doi: 10.1007/s10792-024-03363-8
14. Karl F, Bischler T, Egenolf N, Evdokimov D, Heckel T, Üçeyler N. Fibromyalgia vs small fiber neuropathy: diverse keratinocyte transcriptome signature. Pain. 2021; 162: 2569-2577. doi: 10.1097/j.pain.0000000000002249