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Original Scientific Paper

THE IMPACT OF MIGRAINE ON THE THICKNESS OF THE INNER PLEXIFORM LAYER QUANTIFIED BY OPTICAL COHERENCE TOMOGRAPHY

Anamarija Soldo Koruga[1,2], Nenad Koruga[2,3], Alen Rončević[2,3], Slaven Balog[4], Nina Rotim[5], Silva Butković Soldo[6,7], Davor Jančuljak[1,2], Antonio Kokot[2] and Tatjana Pekmezović[8]

[1]Department of Neurology, University Hospital Center Osijek, Croatia;
[2]Faculty of Medicine, Josip Juraj Strossmayer University of Osijek, Croatia;
[3]Department of Neurosurgery, University Hospital Center Osijek, Croatia;
[4]Ophthalmology Clinic Dr. Balog, Osijek, Croatia;
[5]Faculty of Medicine, University of Zagreb, Croatia;
[6]Faculty of Dental Medicine and Health, Josip Juraj Strossmayer University of Osijek, Croatia;
[7]Faculty of Education, Josip Juraj Strossmayer University of Osijek, Croatia;
[8]Institute of Epidemiology, Faculty of Medicine, University of Belgrade, Serbia

SUMMARY – Introduction: Migraine is one of the most prominent headache disorders with a global burden of over one billion patients and presents as a disabling and painful disorder. The pathophysiological mechanism of migraine is not fully understood, but the most accepted theory suggests that migraine is caused primarily by neural dysfunction leading to secondary changes in cerebral perfusion. Methods: We conducted a case-control study in 175 patients; 88 were migraineurs and 87 were healthy controls. Optical coherence tomography (OCT) was performed in all patients using a spectral-domain OCT device in the same environmental conditions. Inclusion and exclusion criteria were set up to achieve more homogenous groups. OCT was performed to scan all retinal layers with an emphasis on the inner plexiform layer (IPL).

Results: Among the 175 examined subjects, 84% were women. The group of migraine patients consisted of patients without aura (56%) and patients with aura (44%). After collecting tomography data, we did not find subjects with changes of IPL. Nevertheless, only moderate differences in IPL thickness in all quadrants except the central area were found in the group of migraine patients with aura using Pearson's test.

Conclusion: According to the presented results, no significant changes of IPL thickness in migraine patients were found. We have to emphasize the need for further investigation of the relationship between average migraine headache days per month and IPL thickness.

Keywords: *case-control studies; migraine with aura; migraine without aura; optical coherence tomography; retina*

Correspondence to: *Anamarija Soldo Koruga,*
Department of Neurology, University Hospital Center Osijek,
31000 Osijek, Croatia
e-mail. anamarijasoldo@gmail.com

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Introduction

Migraine is a primary headache disorder which lasts up to 72 hours and is usually characterized by moderate or severe recurrent episodes of pulsating pain with a mostly unilateral appearance and a range of accompanying symptoms. Its first appearance in patients is most frequent in puberty, it peaks between 35 and 45 years and is more frequent in the female population. As a global burden, it presents in approximately 15% of the population[1-3]. In 2018, it was classified into three main groups according to the International Headache classification, 3rd edition[4]: migraine without aura (MwoA), migraine with aura (MwA) and chronic migraine. Aura represents transient focal neurological symptoms that usually precede, but sometimes accompany the headache phase of migraine attacks and is present in one third of the individuals who have migraine. Migraine is one of the most prevalent neurological diseases and, although its pathogenesis is not fully understood, an increasing amount of evidence supports the involvement of the neurovascular system in the development of the disease. Previous knowledge, based on Wolff's and Graham's vascular theory of the pathophysiology of migraine in which the aura and headache are caused by the vasoconstriction and vasodilatation of blood vessels of the brain, are not accepted in their entirety today, but they are not abandoned completely. According to the neurogenic (or neurovascular) theory, migraine patients in a state without headache have a hyper excitability of the neurons of the cerebral cortex, especially the occipital cortex. Observing the pathophysiology of MwA, the theory of cortical spreading depression explains the symptoms of aura, which are followed by the activation of the trigeminal vascular system (TGVS) and the development of the next phase of the migraine attack — pain. The release of neurotransmitters and vasoactive intestinal peptides is increased with the activation and sensitization of TGVS. This activation of injurious receptors results in the transmission of pain signals via the trigeminal nociceptive afferent fibers, hence causing pain[5-8]. Visual aura is known as the most common aura symptom during migraine attacks. Transient vasospasm during visual aura or even MwoA can cause a decreased blood flow and perfusion deficit in the ocular vasculature. Cerebral and ocular vascular changes and the subsequent transitory constriction of the retinal and ciliary arteries lead to possible ischemic damage of the optic nerve, retina or choroid, causing thickness changes in retinal layers[9]. OCT is a simple and noninvasive procedure to assess such morphological retinal and choroidal changes in individuals with neurodegenerative diseases — but also in migraine patients — by utilizing infrared wavelengths with a sensitivity of 8-10 micrometers[10-12]. OCT can be used to quantitatively and qualitatively detect and measure the thickness of retinal layers, such as the retinal nerve fiber layer (RNFL), ganglion cell layer (GCL) and inner plexiform layer (IPL). Several recent clinical studies have reported alterations in retinal perfusion, microvascular alterations and consequent changes in the thickness of specific retinal layers in migraine individuals, but different and even contradictory results have been obtained. We performed an extensive clinical study and investigated the impact of MwA or MwoA on possible changes in the thickness of seven retinal layers, but in this paper we will focus primarily on IPL.

Methods

This case-control study was conducted on 175 participants; 88 individuals had the diagnosis of migraine and 87 were healthy/control participants. The study protocol adhered to the tenets of the Declaration of Helsinki and was previously approved by the hospital's Ethics Board. Each participant was informed about the study and signed an informed consent form before participation. The 88 participants who had the diagnosis of MwA or MwoA were treated by a neurologist in the Department of Neurology, University Hospital Center Osijek, Croatia. Key (general) criteria for participants with migraine were: over 18 years of age, the diagnosis of MwA or MwoA confirmed by a neurologist, no pathological findings on native magnetic resonance imaging (MRI) of the brain and brain blood vessels including white matter lesions, and no pathological findings in a neurological examination. Exclusion criteria for the group of cases, i.e. participants with migraine, were: an inability to cooperate due to cognitive or psychiatric disorders, a suspected or confirmed neurodegenerative or demyelinating

disease of the central nervous system, head trauma, nystagmus verified by a neurological examination, myopia or hyperopia > 2 diopters, intracranial operations, eye diseases, ocular hypertension (> 20 mm Hg), history of glaucoma, diabetic maculopathy, diabetic retinopathy, epiretinal membrane, vitreomacular traction, any other pathology of the posterior pole of the eye, post-traumatic eye events, eye infections, optic nerve conduction disorders verified by visual evoked potentials, systemic diseases affecting the brain, optic nerve and eye (diabetes mellitus, hypertension or low blood pressure, dyslipidemia, coagulation disorders, cardiopathy) or active therapy with medication that could affect retinal thickness. Subjects who met all the inclusion criteria and none of the exclusion criteria were invited to participate in the study by undergoing an OCT scan after providing a written informed consent. A total of 87 participants were healthy individuals who also had to be older than 18 years and able to sign the informed consent form. The same exclusion criteria were established for the group of healthy controls and the study groups were matched by sex and age.

Procedure: The study was conducted using a high-resolution spectral-domain scan Heidelberg Spectralis® OCT device. It provides a capture rate of 40,000 A-scans per second with an image magnification of 10 micrometers per pixel. IPL measurements were obtained in the same room, under the same conditions during the morning hours in all participants. In all subjects with migraines, the OCT scan was performed a minimum of 48 hours after the last acute migraine attack and the application of acute therapy for the migraine attack. All the scans were performed by the same ophthalmic technician. The system was centered on fovea and papilla; all scans were fully automatically aligned and focused using real-time eye tracking. All captured scans were automatically saved and analyzed by the OCT software.

Statistical analysis: Data processing was done in the statistical program PSPP v.3. The statistical methods used were the Student's t-test and Pearson's coefficient correlations. The defined level of significance was $P < 0.05$. The normality of the distribution of OCT was checked with the Kolmogorov-Smirnov test. The dispersion of the results was measured with the arithmetic mean and standard deviation.

Results

Overall, 175 respondents participated in the research; 146 (84%) of them were female with an average age of 38 ± 11 and 29 (16%) were male patients with an average age of 36 ± 9.88 (50.3%). A total of 88 respondents had previously been diagnosed with migraine and 87 (49.7%) were healthy/control subjects. In patients with migraine, 49 (56%) reported MwoA and 39 (44%) patients had MwA. Furthermore, a positive family history of migraine was documented in 31 (35%) patients; in 26 (84%) subjects, migraine was found in their parents, and in 5 patients (16%) it was documented in siblings. In the same group, a detailed analysis revealed the incidence of MwA in 11 (28%) patients, and MwoA in 20 (41%) patients. All participants with MwA reported visual aura; 51% had fortification spectrum and 39% partial vision loss (scotoma). The characteristics of pain and the localization of migraine are shown in Table 1.

Table 1. Descriptive data presenting the type of migraine pain and its localization.

Type of pain	**n**	**%**
Pulsatile	77	87.5
Pressure	1	1.1
Blunt	2	2.3
Stabbing	8	9.1
Localization	**n**	**%**
Left-sided hemicrania	25	28.4
Right-sided hemicrania	32	36.4
Both-sided diffuse headache	31	35.2

A Student's t-test was performed in independent samples for each eye separately to analyze any differences in IPL thickness. Two groups of subjects were analyzed: patients with migraine and healthy patients. OCT was performed in both groups and the results did not reveal any statistically significant difference between the groups (Tables 2 and 3). Furthermore, the same test was performed in groups of subjects with MwA and MwoA. The statistical analysis did not reveal any difference in IPL thickness between the two groups (Tables 4 and 5).

Table 2. IPL thickness of the right eye measured by OCT in migraine patients and healthy subjects.

Group		Number of patients	Arithmetic mean (standard deviation)	Difference	95% Confidence interval		*P*-value*
					From	To	
OCT right eye lower temporal area	Migraine	88	26.01 (2.19)	0.13	-0.64	0.67	>0.05
	Healthy	87	26.00 (2.21)				
OCT right eye lower nasal area	Migraine	88	24.69 (2.25)	0.01	-0.64	0.66	>0.05
	Healthy	87	24.68 (2.14)				
OCT right eye central area	Migraine	88	35.55 (3.47)	-0.66	-1.04	0.91	>0.05
	Healthy	87	35.61 (3.07)				
OCT right eye upper temporal area	Migraine	88	27.41 (2.25)	-0.18	-0.85	0.50	>0.05
	Healthy	87	27.59 (2.24)				
OCT right eye upper nasal area	Migraine	88	25.50 (2.42)	-0.01	-0.69	0.67	>0.05
	Healthy	87	25.51 (2.14)				

Table 3. IPL thickness of the left eye measured by OCT in migraine patients and healthy subjects.

Group		Number of patients	Arithmetic mean (standard deviation)	Difference	95% Confidence interval		*P*-value*
					From	To	
OCT left eye lower temporal area	Migraine	88	26.14 (2.30)	0.20	-0.48	0.88	>0.05
	Healthy	87	25.94 (2.25)				
OCT left eye lower nasal area	Migraine	88	24.85 (2.24)	-0.16	-0.83	0.51	>0.05
	Healthy	87	25.01 (2.23)				
OCT left eye central area	Migraine	88	35.19 (3.44)	-0.37	-1.32	0.57	>0.05
	Healthy	87	35.56 (2.89)				
OCT left eye upper temporal area	Migraine	88	27.49 (2.30)	0.21	-0.47	0.90	>0.05
	Healthy	87	27.28 (2.31)				
OCT left eye upper nasal area	Migraine	88	25.68 (2.40)	-0.13	-0.83	0.57	>0.05
	Healthy	87	25.81 (2.25)				

Table 4. OCT-measured IPL thickness of the right eye in patients with MwA and MwoA.

Group		Number of patients	Arithmetic mean (standard deviation)	Difference	95% Confidence interval		*P*-value*
					From	To	
OCT right eye lower temporal area	Without aura	49	25.86 (2.03)	-0.34	-1.28	0.59	>0.05
	With aura	39	26.20 (2.38)				
OCT right eye lower nasal area	Without aura	49	24.45 (2.10)	0.54	-1.50	0.42	>0.05
	With aura	39	24.99 (2.42)				
OCT right eye central area	Without aura	49	35.26 (2.93)	-0.65	-2.14	0.82	>0.05
	With aura	39	35.91 (4.05)				
OCT right eye upper temporal area	Without aura	49	27.19 (2.05)	-0.51	-1.47	0.45	>0.05
	With aura	39	27.69 (2.49)				
OCT right eye upper nasal area	Without aura	49	25.30 (2.24)	-0.45	-1.49	0.58	>0.05
	With aura	39	25.76 (2.62)				

We performed two separate Pearson's tests in order to evaluate the association between the average number of migraine headache days per month and changes in IPL thickness using OCT in subjects with migraine. The group of patients with MwoA and the group of patients with MwA underwent separate tests. The results in the first group did not reveal a statistically significant difference. However, a statistically significant moderate negative correlation in all quadrants of both eyes except the central area was found in the group of patients with MwA. The results indicate a proportion of average migraine headache days per month and changes in IPL thickness, which is presented in Tables 6 and 7.

Table 5. OCT-measured IPL thickness of the left eye in patients with MwA and MwoA.

Group		**Number of patients**	**Arithmetic mean (standard deviation)**	**Difference**	**95% Confidence interval**		***P*-value***
					From	**To**	
OCT left eye lower temporal area	Without aura	49	26.04 (2.18)	-0.23	-1.21	0.76	>0.05
	With aura	39	26.27 (2.47)				
OCT left eye lower nasal area	Without aura	49	24.72 (2.06)	-0.29	-1.25	0.67	>0.05
	With aura	39	25.01 (2.47)				
OCT left eye central area	Without aura	49	34.71 (2.98)	-1.08	-2.54	0.38	>0.05
	With aura	39	35.79 (3.90)				
OCT left eye upper temporal area	Without aura	49	27.29 (2.16)	-0.44	-1.43	0.54	>0.05
	With aura	39	27.75 (2.46)				
OCT left eye upper nasal area	Without aura	49	25.53 (2.32)	-0.34	-1.37	0.69	>0.05
	With aura	39	25.87 (2.52)				

Table 6. Pearson's test for IPL thickness in all quadrants for both eyes in patients with MwoA.

Right eye	**LT**†	**LN**‡	**C**§	**UT**¶	**UN**††
Average migraine headache days per month	-.011	-.067	.088	-.102	-.147
Left eye	**LT**†	**LN**‡	**C**§	**UT**¶	**UN**††
Average migraine headache days per month	.016	.074	.145	-.071	-.134

† lower temporal (LT), ‡ lower nasal (LN), § central (C), ¶ upper temporal (UT), †† upper nasal (UN)

Table 7. Pearson's test for IPL thickness in all quadrants for both eyes in patients with MwA.

Right eye	**LT**†	**LN**‡	**C**§	**UT**¶	**UN**††
Average migraine headache days per month	-.417**	-.516**	-.182	-.408**	-.428**
Left eye	**LT**†	**LN**‡	**C**§	**UT**¶	**UN**††
Average migraine headache days per month	-.488**	-.383*	-.194	-.413**	-.487**

† lower temporal (LT), ‡ lower nasal (LN), § central (C), ¶ upper temporal (UT), †† upper nasal (UN)

Discussion

Our study was conducted in order to find potential changes in retinal layers in a specific layer of IPL which consists of synaptic connections between the axons of bipolar cells and the dendrites of ganglion cells. IPL contains the synapse between the second-order and third-order neuron in the visual pathway and a potential gap in the examination of migraine patients in this layer emerged. Migraines mostly affect the female population[13], which was also seen in the sample of subjects included in our study; the proportion of women in the sample was 84%. The first onset of migraine can occur at any age, but the first notable crisis usually occurs during adolescence with the peak occurring between 30 and 50 years of age. Migraine usually becomes significantly less intense and frequent during the following decades. Our study confirmed these claims as the mean age of our patients was 38 ± 11 and the presence of the male sex was negligible. Migraine is one of the most prevalent neurological conditions worldwide, with an estimated yearly occurrence of more than one billion cases[14]. As a cyclic disorder, it has different phases: premonitory phase, transient neurological symptoms, intense headache attack and postdrome phase[15,16].

The underlying theory of the development and etiology of migraines remains to be elucidated despite recent advancements in diagnostic procedures, even though neurogenic and vasogenic theories have been proposed. The vasogenic theory in the development of migraine prevailed during the 20th century and proposed vasodilation as the cause of migraine pain. Still, the neurogenic theory has gained more momentum lately and postulates that an incorrect cerebral interpretation of normal sensory input in the trigeminal sensory system is the source of migraine discomfort[17].

Regardless of theory, studies of decreased retinal thickness in migraine patients were recently conducted. The use of OCT as the main tool in the investigation of retinal pathology provides pertinent information regarding the diagnosis and diversity of conditions that lead to retinal tissue degeneration and alterations. This technique allows for an accurate measurement of RNFL, GCL or macular volume. As a non-invasive technique based on the principle of low-coherence light interferometry, it became a generally accepted method increasingly used in the analysis of the anterior eye segment. Technically, OCT uses infrared light reflection in order to acquire axial cross-sectional images with a resolution of less than 10 microns[18]. The importance of OCT becomes more obvious as the retina is an extension of the central nervous system and provides a corridor into abnormal brain processes. Lately, OCT has been introduced as a diagnostic tool that has provided concrete proof of RNFL thinning and GCL abnormalities in a variety of neurological conditions involving disorders of the nervous system. The findings of these studies suggest a relationship between the thickness of specific retinal layers, brain atrophy and clinically manifested visual dysfunction, making the eye a useful model for researching multiple sclerosis and neurodegenerative diseases such as Parkinson's disease and Alzheimer's disease[12,19-21]. Structural changes of the optic nerve in migraine are caused by compromised choroidal blood flow which leads to focal ischemic damage of the optic disk. In order to confirm the connection between migraine and retinal layer changes, several studies have been conducted so far in the last decade, with inconsistent results.

As expected, we found a female predominance among the subjects in our study. Also, MwoA was found in 56% and MwA in 44% of the patients. Familiar inheritance in MwA was found in 85% of subjects and chronic migraine was found in only 6% of our subjects. We examined 175 subjects, i.e. 350 eyes, and the results of the OCT scanning of the IPL of the left and right eye did not reveal significant differences among the previously defined groups of subjects. Also, comparing the groups of patients with and without aura with OCT analysis did not reveal any significant differences between the groups.

The association between the frequency of migraine and thickness of IPL among groups of subjects with MwoA and MwA according to Pearson's test did not reveal any statistically significant differences in the group of subjects with MwoA. Nevertheless, a statistically significant moderate negative correlation in all quadrants of both eyes except the central area was found in subjects with MwA.

In 2023, Liinamaa et al. published the largest cohort study on retinal neural tissue and vascular changes[22]. They did not find any statistically significant differences in average RNFL between healthy controls and 375 migraine patients. They also emphasized that migraine

did not have any impact on the subfields of other retinal layers. This study should be taken seriously due to its total number of migraine subjects and healthy controls. Our results are consistent with the aforementioned study, although we worked with a notably smaller sample size. Other studies reported lesser RNFL thickness in migraine patients when compared to healthy controls and some of them observed thinner RNFL in specific quadrants. In 2023, Raga-Martinez et al. reported lesser thickness of the superior quadrant of the peripapillary RNFL; the same study also reported reduced mean thickness of GCL-IPL in chronic migraine patients[12]. Similar to our results, Tak *el al*. detected no significant differences of IPL among the investigated groups[23]. In 2023, Jie et al. published a meta-analysis of sixteen identified studies in which they found a decreasing of RNFL in subjects with MwA and MwoA; also, retinal microvascular impairments were found in subjects with MwA[5]. We can conclude that our results display a similar outcome with the aforementioned meta-analysis; we would also like to emphasize that the number of eyes examined in our study was notably higher in comparison to any single study included in the previously mentioned meta-analysis.

To summarize, we did not find significant changes of IPL in migraine patients. Moreover, the advantage of our study was a completely homogenous group of patients and healthy controls with a relatively large number of subjects. Also, as the first study conducted in the Republic of Croatia, we compared our results with previously conducted studies with great interest. According to the presented results, we can conclude that migraine had no influence on the changes of the examined IPL; although we need to highlight the need for further investigations of the relationship between average migraine headache days per month and IPL thickness.

References

1. Ashina M. Migraine. N Engl J Med. 2020;383(19):1866–1876. doi:10.1056/NEJMra1915327.
2. Eigenbrodt AK, Ashina H, Khan S, Diener HC, Mitsikostas DD, Sinclair AJ, et al. Diagnosis and management of migraine in ten steps. Nat Rev Neurol. 2021;17(8):501–514. doi:10.1038/s41582-021-00509-5.
3. GBD 2016 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 328 diseases and injuries for 195 countries, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet. 2017;390(10100):1211-1259. doi:10.1016/S0140-6736(17)32154-2.
4. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1-211. doi: 10.1177/0333102417738202. PMID: 29368949.
5. Liu Z, Jie C, Wang J, Hou X, Zhang W, Wang J, Deng Y and Li Y. Retina and microvascular alterations in migraine: a systemic review and meta-analysis. Front Neurol. 2023;14:1241778. doi: 10.3389/fneur.2023.1241778.
6. Reggio E, Chisari CG, Ferrigno G, Patti F, Donzuso G, Sciacca G, et al. Migraine causes retinal and choroidal structural changes: evaluation with ocular coherence tomography. J Neurol. 2017;264(3):494–502. doi:10.1007/s00415-016-8369-2.
7. Shayestagul NA, Christensen CE, Amin FM, Ashina S, Ashina M. Measurement of blood flow velocity in the middle cerebral artery during spontaneous migraine attacks: a systematic review. Headache. 2017;57(6):852–861. doi:10.1111/head.13061.
8. Zhang X., Levy D., Kainz V., Noseda R., Jakubowski M., Burstein R. Activation of central trigeminovascular neurons by cortical spreading depression. Ann Neurol. 2011;69(5):855–865. doi: 10.1002/ana.22329.
9. Abdelghaffar M, Hussein M, Thabet NH, Elkady A, Aly M, El-Kholy NA, et al. The potential impact of migraine headache on retinal nerve fiber layer thickness. Egypt J Neurol Psychiatry Neurosurg. 2022;58(1):141. https://doi.org/10.1186/s41983-022-00570-x.
10. Goadsby PJ, Charbit AR, Andreou AP, Akerman S, Holland PR. Neurobiology of migraine. Neuroscience 2009;161(2):327-341. doi:10.1016/j.neuroscience.2009.03.019.
11. Gabriele ML, Wollstein G, Ishikawa H, Kagemann L, Xu J, Folio LS, Schuman JS. Optical coherence tomography: History, current status, and laboratory work. Invest Ophthalmol Vis Sci. 2011;52(5):2425–2436. doi:10.1167/iovs.10-6312.
12. Raga-Martínez I, Povedano-Montero FJ, Hernández-Gallego J, López-Muñoz F. Decrease Retinal Thickness in Patients with Chronic Migraine Evaluated by Optical Coherence Tomography. Diagnostics (Basel). 2023;13(1):5. doi:10.3390/diagnostics13010005.
13. Allais G, Chiarle G, Sinigaglia S, Airola G, Schiapparelli P, Benedetto C. Gender-related differences in migraine. Neurol Sci. 2020;41(Suppl 2):429-436. doi: 10.1007/s10072-020-04643-8. PMID: 32845494; PMCID: PMC7704513.

14. Safiri S, Pourfathi H, Eagan A, Mansournia MA, Khodayari MT, Sullman MJM, et al. Global, regional, and national burden of migraine in 204 countries and territories, 1990 to 2019. Pain. 2022;163(1):e293–e309. doi:10.1097/j.pain.0000000000002275
15. Sacco S, Braschinsky M, Ducros A, Lampl C, Little P, van den Brink AM, et al. European headache federation consensus on the definition of resistant and refractory migraine. J Headache Pain. 2020;21(1):76. doi:10.1186/s10194-020-01130-5
16. Andreou AP, Edvinsson L. Mechanisms of migraine as a chronic evolutive condition. J Headache Pain. 2019;20(1):117. doi:10.1186/s10194-019-1066-0.
17. Shibata Y. Migraine Pathophysiology Revisited: Proposal of a New Molecular Theory of Migraine Pathophysiology and Headache Diagnostic Criteria. Int J Mol Sci. 2022;23(21):13002. doi: 10.3390/ijms232113002. PMID: 36361791; PMCID: PMC9653875.
18. Razaghi, G., Hedayati, E., Hejazi, M. *et al.* Measurement of retinal nerve fiber layer thickness with a deep learning algorithm in ischemic optic neuropathy and optic neuritis. Sci Rep. 2022;12(1):17109. https://doi.org/10.1038/s41598-022-22135-x
19. Maldonado RS, Mettu P, El-Dairi M, Bhatti MT. The application of optical coherence tomography in neurologic diseases. Neurol Clin Pract. 2015;5(5):460–469. doi: 10.1212/CPJ.0000000000000187.
20. Saidha S, Al-Louzi O, Ratchford JN, Bhargava P, Oh J, Newsome SD, et al. Optical coherence tomography reflects brain at-rophy in multiple sclerosis: A four-year study. Ann Neurol. 2015;78(5):801–813. doi: 10.1002/ana.24487.
21. Ascaso FJ, Cruz N, Modrego PJ, Lopez-Anton R, Santabárbara J, Pascual LF, et al. Retinal alterations in mild cognitive impairment and Alzheimer's disease: An optical coherence tomography study. J Neurol. 2014;261(8):1522–1530. doi: 10.1007/s00415-014-7374-z.
22. Ristioja S, Leiviskä IL, Saarela VO, Liinamaa MJ. Retinal neural tissue and vascular calibres in migraine: the Northern Finland Birth Cohort Eye Study. Acta Ophthalmol. 2023;101(7):1–10. doi:10.1111/aos.16617.
23. Tak AZA, Sengul Y, Bilak Ş. Evaluation of white matter hyperintensities and retinal fiber layer, ganglion cell layer, inner-plexiform layer, and choroidal layer in migraine patients. Neurological Sci. 2018;39(3):489–496. doi:10.1007/s10072-017-3228-5.

Sažetak

UTJECAJ MIGRENE NA DEBLJINU UNUTARNJEG PLEKSIFORMNOG SLOJA MJEREN OPTIČKOM KOHERENTNOM TOMOGRAFIJOM

A. Soldo Koruga, *N. Koruga*, *A. Rončević*, *S. Balog*, *N. Rotim*, *S. Butković Soldo*, *D. Jančuljak*, *A. Kokot* i *T. Pekmezović*

Uvod: Migrena je jedan od najistaknutijih oblika glavobolje s globalnim opterećenjem od preko milijardu pacijenata i prezentira se kao onesposobljavajući bolni poremećaj. Patofiziološki mehanizam migrene nije u potpunosti razjašnjen, ali najprihvaćenija teorija sugerira da je migrena prvenstveno uzrokovana neuronskom disfunkcijom koja dovodi do sekundarnih promjena u cerebralnoj perfuziji.

Metode: Autori su proveli studiju slučaj-kontrola na 175 pacijenata; 88 su bili oboljeli od migrene, a 87 zdrave kontrolne skupine. Optička koherentna tomografija (OCT) provedena je kod svih pacijenata korištenjem spektralno-domenskog OCT uređaja u istim uvjetima okoline. Uspostavljeni su uključni i isključni kriteriji kako bi se postigle homogenije skupine. OCT procedura provedena je skeniranjem svih slojeva mrežnice s naglaskom na unutarnji pleksiformni sloj (IPL).

Rezultati: Među 175 pregledanih ispitanika, 84% bile su žene. Skupinu pacijenata s migrenom činili su pacijenti bez aure (56%) i pacijenti s aurom (44%). Nakon prikupljanja podataka tomografije nismo pronašli ispitanike s promjenama IPL-a. Ipak, u skupini pacijenata s migrenom i aurom, Pearsonovim testom pronađene su samo umjerene razlike u debljini IPL-a u svim kvadrantima osim središnjeg područja.

Zaključak: Prema prikazanim rezultatima, nisu pronađene značajne promjene debljine IPL-a kod pacijenata s migrenom. Nužna je potreba za daljnjim istraživanjem odnosa između prosječnog broja dana migrenske glavobolje u mjesecu i debljine IPL-a.

Ključne riječi: *studije slučaj-kontrola; migrena s aurom; migrena bez aure; optička koherentna tomografija; mrežnica*