Evaluation of the Retina and Optic Discs of Patients with Chronic and Episodic Migraine using Optical Coherence Tomography and Optical Coherence Tomography Angiography

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Abstract

Objective: Migraine is a recurrent neurovascular disorder that is typically characterized by unilateral pulsating pain ranging between moderate and severe intensity and might be accompanied by transient neurological, autonomic, cognitive, and emotional symptoms [1]. Having a prevalence of 14.7% (18.8% in women and 10.7% in men), it is the third-most common disease worldwide. Although the pathophysiology of migraine has not been fully understood yet, there are two main theories in the current literature: the trigeminovascular theory and the cortical spreading depression (CSD) [2]. Both of these theories assume the development of cerebral hypoperfusion. Therefore, the probability of migraine causing ischemic complications is high. Vascular abnormalities seen in migraine might cause decreased perfusion in the retina and optic nerve heads [3]. It was argued that migraine might be a risk factor for both vascular and neuronal density loss in these regions [4]. The present study aims to determine the effects of migraine on the retina and optic disk by comparing the data obtained from Optical Coherence Tomography (OCT) and Optical Coherence Tomography Angiography (OCTA) in patients with chronic and episodic migraine.

Introduction and Objective

Migraine is a recurrent neurovascular disorder that is typically characterized by unilateral pulsating pain ranging between moderate and severe intensity and might be accompanied by transient neurological, autonomic, cognitive, and emotional symptoms [1]. Having a prevalence of 14.7% (18.8% in women and 10.7% in men), it is the third-most common disease worldwide. Although the pathophysiology of migraine has not been fully understood yet, there are two main theories in the current literature: the trigeminovascular system and the cortical spreading depression (CSD) [2]. Both of these theories assume the development of cerebral hypoperfusion. Therefore, the probability of migraine causing ischemic complications is high. Vascular abnormalities seen in migraine might cause decreased perfusion in the retina and optic nerve heads [3]. It was argued that migraine might be a risk factor for both vascular and neuronal density loss in these regions [4]. The present study aims to determine the effects of migraine on the retina and optic disk by comparing the data obtained from Optical Coherence Tomography (OCT) and Optical Coherence Tomography Angiography (OCTA) in patients with chronic and episodic migraine.
to those of healthy controls, as well as shedding light on new studies on early diagnosis, monitoring, and treatment of the disease.

**Materials and Method**

In total, 60 migraine patients (50 women, 10 men) aged between 18 and 45 years, who applied to the neurology clinic and were diagnosed with migraines and had their types determined by using the criteria set by the International Headache Society (IHS) in 2013, were involved in the present study. The control group consisted of 48 healthy volunteers (34 women, 14 men) between the ages of 18 and 45, who applied to the ophthalmology clinic. Neurological and physical examinations were performed for all participants. Following the neurological examination during the routine outpatient controls, optical coherence tomography (OCT) and optical coherence tomography angiography (OCTA) were performed for migraine patients. Images of the patients’ retina and optic disc were taken by using OCT and OCTA and were compared to those of the control group.

All participants in the present study underwent a full ophthalmic examination, including visual acuity, intraocular pressure (IOP) measurement, and anterior and posterior segment examinations. OCT and OCTA imaging were performed using an Optovue RTVue XR Avanti spectral domain OCT device with AngioVue software version 3.5 (Optovue Inc., Fremont, CA, USA).

For both eyes, optical coherence tomography angiography images were obtained from a 6 x 6 mm cube of the macula and a 4.5 x 4.5 mm cube of the optic nerve. The retinal vascular network was analyzed using the perfusion density (PD). The quantitative analysis of PD was performed using PD maps of the macula (ETDRS schema) and optic nerve head (S/I and TSNIT charts). By using macrocular OCTA scans, the foveal avascular zone (FAZ) area on the superficial capillary plexus (SCP) and PD values of the entire SCP, SCP inner (0.5-1.5 mm) and outer (1.5-3.00 mm) ETDRS sectors, nine ETDRS sectors, the entire deep capillary plexus (DCP), and DCP inner and outer ETDRS sectors were collected. PD values of the retinal peripapillary capillary plexus (RPCP) were also recorded for the superior/ inferior (S/I) sectors and optic nerve head (TSNIT) sectors.

OCT and OCTA parameters of chronic and episodic migraine patients were compared to those of healthy control groups. The perfusion densities of the outer (1.5-3.00 mm) ETDRS sectors, nine ETDRS sectors, the entire deep capillary plexus (DCP), and DCP inner and outer ETDRS sectors were obtained. The PD values of the foveal avascular zone and the entire SCP and inner SCP (0.5-1.5 mm retinal peripapillary capillary plexus) on the superficial capillary plexus (SCP) were also recorded for upper and lower sectors and TSNIT sectors.

**Statistical methodology**

The distributions of individuals constituting the study group by various demographic characteristics were determined by preparing frequency tables, whereas crosstabs were used in determining the distributions of some characteristics of migraine patients by categorical variables. The Chi-square test was used in determining if the frequency distributions in the crosstabs were statistically different, while Fisher's exact Chi-square test was used in the analysis of 2x2 crosstabs.

The normality of the distribution of the measurement results for the parameters used in the study was tested by using the Kolmogorov-Smirnov test and considering the skewness-kurtosis coefficients. Differences between the mean values of the groups were examined using the independent groups t-test to determine if the differences were statistically significant. The significance level was set at 0.05 for two-tailed hypothesis tests and p < 0.05 was accepted that there was a statistically significant difference between the mean values of the groups.

**Results**

The patient group consisted of 83.3% women and 16.7% men with migraine, whereas the control group consisted of 70.8% female and 29.2% male healthy individuals. There was no statistically significant difference between the gender distributions of the experimental and control groups (p>0.05).

Among the migraine patients, 28.3% were in the age group of 18-30 years, 46.7% in the age group of 31-40 years, and 25.0% in the age group of 41-45 years. In the control group, however, 27.1% of the participants were in the age group of 18-30 years, 39.6% in the age group of 31-40 years, and 33.3% in the age group of 41-45 years. No statistically significant difference in age distribution was found between the experimental and control groups (p>0.05).

It was determined that the mean vascular densities in the parafoveal (para.sup, para.inf) and perifoveal (peri.sup.hemi, peri.sup, peri.nasal) subregions of the superficial layer of the right eye retina were significantly lower in the experimental group when compared to the control group (p<0.05). There was no statistically significant difference in the mean vascular densities in other subregions of the superficial layer of the right eye retina between the experimental and control groups (p>0.05) (Table 1).

No statistically significant difference was found in the mean vascular density values in all subregions of the superficial layer of the left eye retina between the experimental and control groups (p>0.05) (Table 2).

It was determined that only the mean thickness of the superficial layer of the right eye's retina in the Parfovea (Para. Nasal) sub-region was significantly higher in the experimental group compared to the control group (p<0.05) (Table 3). No statistically significant differences were found between the mean thickness values of the other sub-regions in the experimental and control groups (p>0.05).

In the left eye, there was no statistically significant difference experimental and control groups in terms of the mean thickness values of all sub-regions in the superficial layer of the retina (p>0.05) (Table 4).

It was seen that the mean vascular density values of the whole region (Sup.Hemi, Inf.Hemi) and Parafovea (Para.Sup.Hemi, Para.Inf.Hemi, Para.Temporal, Para.Sup, Para.Nasal, Para.Inf) and PariFovea (Peri.Sup.Hemi, Peri.Inf.Hemi, Peri.Sup, Peri.Nasal, and Peri.Inf) sub-regions in the deep layer of the right eye’s retinawere significantly lower in the experimental group when compared to the control group (p<0.05) (Table 5). No significant difference was found between the groups in terms of the mean vascular density of the Fovea and PariFovea (Peri.Temporal) sub-regions in the deep layer of the right eye's retina (p>0.05).

The mean vascular density values in the whole (inf. heme) and parafovea (para. sup. heme, para. inf. heme, para. sup. para. nasal, para. inf.), and perifovea (peri. sup. heme, peri. inf. heme, peri.
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Table 2. Retina Yüzeyel Katmanının Damar Dansitesi (VD) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)
### Table 3. Retina Yüzeyel Katmanının Kalınlık (Thickness) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sağ Göz)

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### Table 4. Retina Yüzeysel Katmanının Kalınlık (Thickness) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)

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### Table 6. Retina Derin Katmanının Damar Dansitesi (VD) Ortalamalarının Hasta ve Kontrol Grubuna GöreKarşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)

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<td>t</td>
<td>p</td>
</tr>
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<td>Kontrol</td>
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</table>

Table 8. Retina Derin Katmanının Kalınlık (Thickness) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)

<table>
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<tr>
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<th>Grup</th>
<th>N</th>
<th>Ort.</th>
<th>ss.</th>
<th>t</th>
<th>p</th>
</tr>
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<tbody>
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<td>17,57</td>
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<td>Kontrol</td>
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<td>12,59</td>
<td></td>
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</tr>
<tr>
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<td>12,95</td>
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</table>

Table 9. Retina Derin Katmanının Kalınlık (Thickness) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sağ Göz)
<table>
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<tr>
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<th>N</th>
<th>Ort.</th>
<th>ss.</th>
<th>t</th>
<th>p</th>
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</table>

**Table 10. Retina Sinir Lifi Dansitesi (RNFL) Ortalamalarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)**

<table>
<thead>
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<th>N</th>
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**Table 11. Optik Diskte Cup Disk Oranlarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sol Göz)**

<table>
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<th>Ort.</th>
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<td>0,24</td>
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**Table 12. Optik Diskte Cup Disk Oranlarının Hasta ve Kontrol Grubuna Göre Karşılaştırılmasına İlişkin t Testi Sonuçları (Sağ Göz)**

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Discussion

Although the pathophysiology of migraine has not been fully understood yet, it is considered as a systemic neurovascular disorder, and ischemic events in migraine are not limited to the brain. Other systemic ischemic conditions such as stroke, angina, and myocardial infarction are also observed more frequently in these patients when compared to healthy subjects [5,6]. Two main theories have been proposed for the pathophysiology of migraine, which are the trigeminovascular model and the cortical spreading depression (CSD) [7]. In both theories, it is thought that cerebral hypoperfusion develops during an attack. The trigeminovascular system activates during a migraine attack. Vasoactive substances are secreted upon the activation of nociceptive neurons around the blood vessels. This process triggers mast cell degranulation and causes sterile neurogenic inflammation in the dura mater. In addition, the monosynaptic reflex arc between the trigeminocervical complex and the superior salivatory nucleus activates the parasympathetic nerve endings around the dural blood vessels. Following this process, vasodilator agents are released and vasodilation occurs [8]. In summary, migraine attacks alter both intracerebral and extracerebral vascular regulations. Therefore, migraine is likely to cause the development of ischemic complications. Decreased perfusion in the optic nerve head and retina might be observed in migraine due to these vascular disorders.

Although the vascular abnormalities observed in cerebral and retrobulbar arteries are temporary, the chronic nature of migraine, which is characterized by recurring attacks, might lead to permanent retinal damage [9]. As can be seen, migraine is considered as a risk factor for ischemic optic neuropathy [10]. Kara et al. [11], supporting this hypothesis, investigated retrobulbar circulation and hemodynamic changes among patients having migraine by using color Doppler sonography. They revealed that central retinal artery and posterior ciliary artery resistances were higher in migraine patients during headache-free periods. Greven et al. [12] reported that 25% of young adults with retinal artery occlusion had a history of migraine, and none of them were acutely symptomatic during the event.

There are only few studies that examine the effects of migraine on retinal vascular structure by using OCTA. Chang et al. [13] compared the macular and optic nerve vessel densities (VD) and reported that the foveal avascular zone (FAZ) area was significantly larger, as well as a decreased foveal VD, in migraine patients when compared to healthy control participants. Ulusoy et al. [14] reported thinner retinal nerve fiber layer thickness (RSLT) and wider FAZ area in migraine patients. Lower superficial and deeper foveal VDs were found using OCTA but there was no statistically significant difference in parafoveal VDs.

In previous studies, macular parameters such as RSLT thickness, choroidal thickness, ganglion cell layer (GCL) thickness, and foveal thickness among migraine patients were evaluated by making use of the OCT findings. Gipponi et al. [15] reported a decrease in RSLT thickness in the upper retinal quadrant of migraine patients when compared to normal subjects, regardless of the duration or frequency of the disease. Reggio et al. [16] reported a thinner RSLT in migraine patients in comparison to the control group. Acer et al. [17] found no significant difference between patients and controls in terms of GCL and macular thickness. Salman et al. [18] also determined that there was no statistically significant difference between the RSLT thickness of the migraine group and the control group. In the present study, there also was no significant difference in RSLT and the thickness of the superficial and deep retinal layers between the groups. These controversial results are thought to be related to focal perimetric changes and differences in the sensitivity of retinal axons to ischemia [19,20].

Karaca et al. [21] analyzed choroidal thickness in 32 migraine patients during the headache-free period. They concluded that migraine causes a decrease in choroidal thickness, as well as vasoconstriction and ischemia, during the headache-free period. As a result of these studies, it was concluded that migraine is a vasoconstrictive disease even during headache-free periods. In the present study, it was determined that the vascular densities (VD') of the foveal, parafoveal, perifoveal, superficial capillary plexus (SCP), and deep capillary plexus (DCP) were low in the patient group when compared to the control group during the headache-free period. This finding suggests that the low vascular density (VD') in the experimental groups during headache-free periods might be because of the vasoconstriction originating from the autonomic nervous system dysfunction.

Aoe et al. [22] reported significant changes in the thickness of the RSLT and choroid and emphasized that they might be associated with cortical spreading depression. Phelps and Corbett [23] revealed that the incidence of low-tension glaucoma in migraine patients was higher and it was attributed to the
narrowing of retrobulbar arteries. Grosberget al. [24] evaluated 46 patients having retinal migraine and they found permanent visual loss due to long-term vascular damage in 21 (46%) of those 46 patients. The mean age of their patient cohort was 25 years (range 7-54 years) and they had a very low incidence of vascular risk factors.

As mentioned in these studies, repeated vasospasm and focal ischemia during attacks can lead to optic nerve and retinal damage, ultimately contributing to ocular diseases such as ischemic optic neuropathy and glaucoma. In our study, the lower vertical and horizontal cup-to-disc (c/d) ratio at the optic nerve head compared to the control group supports the vascular theory in migraine pathophysiology. These studies suggest that migraine is not a central but a systemic disease.

Conclusion

It is thought that OCT and OCTA findings in migraine patients might be associated with an increased risk of ocular and systemic vascular events. OCT and OCTA may provide potential benefits as a non-invasive, rapid, and relatively inexpensive biomarkers in migraine patients.

Ethical approval

Ethical approval was obtained from the Ethics Committee of the Medical Faculty of Kocaeli University (Decision Number: 2020/271).

References