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ARAŞTIRMA / RESEARCH

Relationship between headache, corpus callosum, and deep white matter lesions in patients with migraine

Migrenli hastalarda başağrısı, korpus kallozum ve derin beyaz madde lezyonları arasındaki ilişki

Mehmet Onay[1], Aydın Talip Yıldoğan[2], Fettah Eren[3]

[1]Dr. ErsinArslan Training and Research Hospital, Department of Radiology, [2]Department of Neurology, Gaziantep,Turkey
[3]Selcuk University Medical Faculty Hospital, Department of Neurology, Konya, Turkey

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Abstract

Purpose: The aim of the study was to examine the relationship of white matter hyperintensities (WMHs) and measurements of corpus callosum (CC) with migraine characteristics.

Materials and Methods: This study was conducted as a case-control and prospective study that included 50 migraine patients and 40 individuals in the control group. The severity and frequency of headaches and headache pain characteristics were questioned. The effects of the disease were determined using the migraine disability rating scale (MIDAS). WMH lesions were evaluated with the FAZEKAS scale. Morphometric measurements of CC were performed. The relationship between pain characteristics and measurements was examined.

Results: There were 50 migraine patients (44 women, 6 men) with a mean age of 30,86±8,64 (18-51) years. The frequency of multi-point WMHs was higher in migraine patients compared to the control group. There were no differences in the measured values of CC between the two groups. WMHs were more common in patients with nausea complaints. Measured CC genu values were lower in patients with phonophobia and visual aura symptoms.

Conclusion: This study has revealed that patients with migraines have a high rate of WMHs, and this rate is even higher when nausea accompanies attacks. No relationships were found between the measured values of CC and migraines.

Keywords:. Migraine, magnetic resonance imagining (MRI), corpus callosum.

Öz

Amaç: Çalışmanın amacı, beyaz cevher hiperin tensiteleri (BCH) ve korpuskallozum (KK) ölçümlerinin migren özellikleri ile ilişkisini incelemektir.

Gereç ve Yöntem: Bu çalışma, 50 migren hastası ve control grubundaki 40 bireyi içeren, vaka-kontrol ve ileriye dönük bir çalışma olarak yapılmıştır. Başağrısının şiddeti ve sıklığı ile başağrısı ağrı özellikleri sorgulandı. Hastalığın etkileri migren özürlülük derecelendirme ölçeği (MIDAS) kullanılarak belirlendi. WMH lezyonları FAZEKAS skalasiiledeğerlendirildi. KK'un morfometrik ölçümleri yapıldı. Ağrı özellikleri ile ölçümler arasındaki ilişki incelendi.

Bulgular: Yaş ortalaması 30,86±8,64 (18-51) yıl olan 50 migrenhastası (44 kadın, 6 erkek) vardı. Migren hastalarında çok noktalı BCH'lerin sıklığı control grubuna göre daha yüksekti. İki grup arasında ölçülen KK değerlerinde fark yoktu. Bulantı şikayetleri olan hastalarda BCH'ler daha yaygındı. Fonofobi ve görsel aura semptomları olan hastalarda ölçülen KK genu değerleri dahadüşüktü.

Sonuç: Bu çalışma, migrenli hastalarda BCH oranının yüksek olduğunu ve ataklara bulantı eşlik ettiğinde bu oranın daha da yüksek olduğunu ortaya koymuştur. CC'nin ölçülen değerleri ile migren arasında herhangi bir ilişki bulunamadı.

Anahtarkelimeler: Migren, manyetik rezonans görüntüleme (MRG), korpuskallozum

YazışmaAdresi/Address for Correspondence: Dr. Mehmet Onay, Dr. ErsinArslan Training and Research Hospital, Department of Radiology, Gaziantep, Turkey E-mail: mehmetonay079@hotmail.com
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INTRODUCTION

Migraine is a genetically inherited disease, characterized by moderate-to-severe headache attacks associated with increased sensitivity to light and sound, often accompanied by nausea. Migraine headaches are usually unilateral[1]. Neuroimaging studies have frequently revealed white matter hyperintensities (WMHs), silent infarct-like lesions, ischemic lesions, as well as volumetric changes in gray and white matter in migraine patients[2-5]. WMHs are usually small punctiformhyperintense lesions localized to deep, subcortical, periventricular, and infratentorial structures, and have no mass effects. WMHs are typically seen on MRI in T2 and FLAIR (fluid-attenuated inversion rescue weighted) sequences. They are suggested to be consisting of gliosis, demyelination, and characterized by loss of axons, possibly resulting from microvascular injury[3,6,7]. The incidence of WMHs in elderly individuals has gradually increased[8].WMHs are often detected in migraine patients[9].In adult migraine patients, structural changes are observed in white matter and gray matter, with volumetric changes in the processing areas of the brain responsible for nociception and visual and sensorimotor functions.Corpus callosum (CC) is the largest white matter commissure connecting and coordinating the cerebral hemispheres[10]. There are very few studies on CC measurements in migraine patients. Although some authors noted the presence of a smaller CC in patients with migraine compared to controls, the role of the CC in the pathophysiology of migraine has not been fully understood[10,11]. A better understanding of the brain changes of patients with migraines will not only aid in diagnosis but may also help explain migraine pathophysiology, leading to the development of targeted anti-migraine therapies.

This study aimed to examine the relationship of WMHs and measured CC values with migraine characteristics.

MATERIALS AND METHODS

This study was performed in the Gaziantep Dr. ErsinArslan Training and Research Hospital in the period between September 2021 and December 2021. Written informed consent was obtained from each participant and all study procedures were performed in compliance with all principles of the Declaration of Helsinki. The study was designed as a prospective case-control study. This study was approved by the Gaziantep University clinical research local ethics committee (No: 2021/238).

Power analysis was performed to find the number of patients required to be included in the study. The power analysis revealed that it would be adequate to include 90 patients (alpha error probe 0.05, power 0.8).

Participants

The study included a patient group and a control group. The patient group consisted of migraine patients, who were admitted to the neurology outpatient clinic of the hospital. Migraine patients with cognitive dysfunction, central nervous system disease (congenital brain anomaly, mass lesions in the brain, stroke, metabolic and infective diseases, etc.), hypertension, cardiovascular disease, renal disease, endocrine/metabolic disorder, alcohol/substance abuse, a history of a psychiatric disorder, and migraine patients, who were using antidepressant-antipsychotic medications, under the age of 18, and over the age of 65, were excluded from the study. Large vessel disease was excluded by carotid and vertebral ultrasonography and cardiac diseases were excluded by echocardiography and electrocardiography in patients with ischemic lesions in the brain. Migraine was diagnosed using the International Headache Society's criteria.

To homogenize patients' demographics, a control group was included, consisting of individuals with age and gender distribution similar to the migraine patients group. Patients in the control group were selected according to their medical records. Patients without neurological deficits or complaints were selected to be included in the control group of the study.The participants to be included in the patient and control groups of the study were selected by a neurologist (ATY) with at least 5 years of experience.

Age, gender, number of children, and number of sisters and brothers of the patients were recorded. Educational statuses were grouped as primary school, high school, and university. The income levels of the patients and the absence or the presence of a history of migraines in the family and, if any, the family members with migraines were recorded. The migraine disease duration was recorded in years.

Headache assessments

Headache characteristics of the patients were questioned. Patients were asked how many hours their headaches lasted without the use of analgesics and after taking analgesics. Then, patients were divided into groups according to their answers (shorter than 4 hours, 4-24 hours, 24-72 hours, and longer than 72 hours). The severity of headaches was divided into four groups as mild, moderate, severe, and unbearable. Pain lateralization was grouped as unilateral or bilateral. According to the pain characteristics, patients were grouped as those having throbbing-pulsating or continuous-constant headaches, or headaches with other features. The relationship of headache pain with physical activity (worsening, resolving, unaffected) was examined. Symptoms accompanying headaches (nausea, vomiting, photophobia, visual disturbances, and hypoesthesia-hemiparesis) were determined.

Migraine disability rating scale (MIDAS)

The Migraine Disability Assessment Scale (MIDAS) [12] is used to evaluate the disability status of migraine patients in their work, home, and social lives. The number of headache attacks in the last three months was questioned. Pain intensity was asked to be expressed as a number between 0 (no pain) and 10 (worst possible pain). According to this response, patients were grouped as having severities of 5-6 = moderate, 7-8 = severe, and 9-10 = unbearable pain.

Evaluation of magnetic resonance images

MRI images taken within the last six months were included in the study. Measurements and evaluations were performed by MagnetomAvanto 1.5 T (Siemens Healthcare, Erlangen, Germany). MRI evaluation features included a field of view (FOV) of 230 mm, a matrix of 256 x 256, a thickness of 5 mm, a spacing of 1 mm, the number of excitations (NEX) of 2-3, and an echo and repetition time (TE/TR) of 15/500 ms. WMHs were identified on T2 weighted and FLAIR sequences by the radiologist (MO) with at least 10-year of experience.

CC measurements were performed using TSE/T1 sagittal sequences included in the routine brain MRI protocol. The measurements of CC were illustrated in Fig. 1. These drawings and measurements were performed by the same investigator, who did not know any other patient information. The FAZEKAS scale [13] was used to identify and classify WMHs.Patients were divided into groups consisting of patients either with or without WMHs.

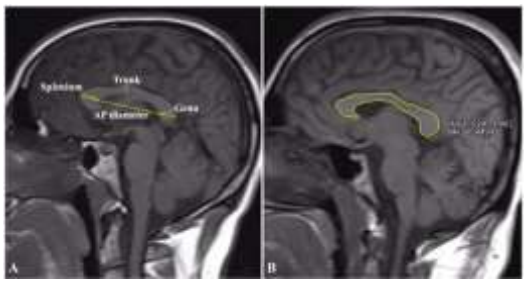

Fig.1. The measurements of the corpus callosum (CC) on sagittal T1-weighted MRI. Fig. 1A shows the anterior-posterior diameter, genu, truncus, and splenium measurements. Fig. 1B shows the CC area on the same plane. It is calculated automatically with a closed-line manual drawn around the CC.

Statistical analysis

Data analysis was performed using The Package for Social Sciences (SPSS) version 16 (IBM, Armonk, NY, USA) software. The conformity of the data to a normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk tests). Arithmetic means, standard deviation, median, and minimum and maximum values were used in the evaluation of numerical data, and frequency distributions and percentages were used to summarize categorical data. Chi-square (χ^2) test was used to compare categorical data. The relationships between numerical data were examined by the Independent Simple t-test and the relationships between categorical data were evaluated by the Man-Whitney U test. The Kruskal Wallis test was used to evaluate three or more groups with numerical data. A post-hoc Man-Whitney U test and Bonferroni correction were performed for paired comparisons between groups with significant Kruskal Wallis test results. Correlations of numerical variables were analyzed with the Pearson correlation coefficient. Pearson Correlation Coefficients of 0.05-0.30, 0.30-0.40, 0.40-0.60, 0.60-0.70, 0.70-0.75, and 0.75-1.00 were considered to indicate low or insignificant, low-moderate, moderate, good, very good, and excellent correlations, respectively. Correlation coefficients with positive signs indicate that the values of variables increase and decrease together. Correlation coefficients with negative signs indicate that, when

the value of one of the variables increases, the other decreases or vice versa.

RESULTS

There were 86 patients, who were diagnosed with migraine to be evaluated for their eligibility for the study. A total of 36 patients, of whom 17 older than 65 years, 9 using antidepressants, 4 with endocrine disorders, and 6 with other reasons, were excluded from the study.

Fifty patients with migraines were included in the study. Of these patients, 88.0% (n=44) were women and 12.0% (n=6) were men, with a mean age of 30.86±8.64 (min:18-max:51) years. Of the 40 control patients, 72.5% (n=29) were women and 27.5% (n=40) were men, with a mean age 28.00±7.08 (min:17-max:43) years. There were no statistical differences between the patient and control groups in terms of gender and age (χ^2=3.485 and p=0.062, t=1.687 and p=0.095, respectively). Sociodemographic characteristics of the patients are summarized in Table 1.

The mean disease duration of migraine was 5.18±6.09 (min:1.00-max:30.00) years. There was no family history of migraines in 23 patients (46%). The pain often persisted for 4-24 hours without analgesics (n=26, 52%). In the presence of analgesics, the most common pain durations were shorter than 4 hours (n=24, 48%) and in the range of 4-24 hours (n=17, 34%). Headache characteristics and accompanying symptoms and signs of the patients are summarized in Table 2.

Table 1. Demographic characteristics of migraine patients

Characteristic	n	%
Marital Status		
Married	29	58.0
Bachelor/Divorced	21	42.0
Educational status		
Illiterate	2	4.0
Primary School	23	46.0
High School	15	30.0
University	10	20.0
Employment status		
Not working	38	76.0
Working at a desk job	1	2.0
Working in a physically demanding job	11	22.0
Income rate		
Below minimum wage	13	26.0
Minimum wage	28	56.0
2 fold of minimum wage	5	10.0
At least 3 fold of minimum wage	4	8.0

Table 2. Headache-related characteristics of patients with migraine

Characteristic	n	%
Presence of migraine family history		
Absence of migraine family history	27	54.0
Present in a first-degree relative	18	36.0
Present in a second-degree relative	3	6.0
Present both in a first and in a second degree relative	2	4.0
Duration of a headache without medicine		
4-24 hours	26	52.0
24-72 hours	17	34.0

Over 72 hours	7	14.0
Duration of a headache with medicine		
Below 4 hours	24	48.0
4-24 hours	17	34.0
24-72 hours	8	16.0
Over 72 hours	1	2.0
Severity of headaches		
Moderate	5	10.0
Severe	22	44.0
Unbearable	23	46.0
Lateralization of headaches		
One-sided on right side	22	44.0
One-sided on left side	3	6.0
Bilateral	25	50.0
Characteristics of headaches		
Throbbing	40	80.0
Continuous	9	18.0
Other	1	2.0
Does the headache affect daily workability?		
No	47	94.0
Yes	3	6.0
Association of headache with physical activity		
Getting worse with physical activity	37	74.0
Not changing with physical activity	13	26.0
Nausea/vomiting accompanying headaches		
None	11	22.0
Present	39	78.0
Photophobia accompanying headaches		
None	13	26.0
Present	37	74.0
Phonophobia accompanying headaches		
None	10	20.0
Present	40	80.0
Visual symptoms accompanying headaches		
None	27	54.0
Present	23	46.0
Sensory symptoms accompanying headaches		
None	25	50.0
Present	25	50.0

According to the FAZEKAS scale, 70.0% (n=35) of the patients had no lesions/a single lesion and 30.0% (n=15) had multiple punctiform lesions. In the control group, 87.5% (n=35) had no lesions/a single lesion and 12.5% (n=5) had multiple point lesions. When the patient and control groups were compared according to FAZEKAS scale findings, the frequency of multiple punctiform lesions was higher in migraine patients than in the control group (X^2=3.938, p=0.047). No statistically significant differences were observed in the diameter and the measured area of CC between migraine patients and the control group.

The measured values of CC in the patient and control groups and the relationship between them are summarized in Table 3. Among migraine patients, there were no differences in the measured values of CC between the FAZEKAS scale groups. The measurements of CC by FAZEKAS scale scores were summarized in Table 4. MIDAS scores were obtained by inquiringmigrainepatients about their headaches in the last 3 months. The mean MIDAS score was found as 18.12±15.44. The number of days with pain in the last three months was 8.50±6.58. According to the visual pain scale, the mean pain severity was 7.80±1.48. When the measured values of CC were examined according to the MIDAS scale scores, a negative correlation was revealed between the genu diameter and impairments in housework performance (p=0.003, r=-0.307), the social or family life (p=0.003, r=-0.330), and MIDAS scores (p=0.022, r= -0.324). As the VAS scores increased, the measured area of CC decreased (p=0.019, r=-0.30). There were no correlations between other MIDAS parameters and the measured values of CC (p>0.05). Relationships between the measured values of CC and MIDAS scale scores were summarized in Table 5.

When the relationship between the pain characteristics and the measured values of CC was examined among migraine patients, no relationships were found between the presence of a family history of migraines and CC measurements (p=0.345, p=0.697, p=0.453, p=0.953 respectively). There were no differences between the duration of pain with or without analgesic use and the measured values of CC (p >0.05). There were no relationships between pain severity, location of the headache, characteristics of pain, the relationship of the headache with physical activity, the presence of photophobia-paresthesia, and the measured values of CC (p >0.05).

Table 3. Findings from corpus callosum measurements compared between patient and control groups

Characteristics	Patient (n=50)		Control (n=40)			
	Mean±SD	Median (min-max)	Mean±SD	Median (min-max)	t*	p
The total area of the corpus callosum (mm^2)	625.62±91.84	619.8 (310.7-791.8)	624.05±74.78	631.15 (450.5-796.6)	0.087	0.931
Genu diameter of the corpus callosum (mm)	11.19±1.38	11.2 (8.2-15.3)	11.32±1.45	11.00 (8.40-14.70)	-0.414	0.680
Corpus callosum truncus diameter (mm)	7.20±1.05	7.3 (4.5-9.7)	7.50±0.85	7.55 (5.20-9.10)	-1.446	0.152
Corpus callosum splenium (mm)	11.63±1.38	11.5 (8.6-14.8)	11.31±1.11	11.25 (9.10-13.90)	1.194	0.236
AP diameter of the corpus callosum (mm)	68.60±4.08	68.55 (59.8-78.5)	68.69±4.29	67.60 (60.90-77.60)	-0.105	0.916

AP: Anteroposterior, *=Independent sample t-test

Table 4. Corpus callosum measurements of migraine patients according to FAZEKAS scale groups

Characteristic	FAZEKAS 0 (n=35)	FAZEKAS 1 (n=15)		
	Mean±SD	Mean±SD	z*	p
Corpus callosum area (mm^2)	624.27±100.48	628.76±70.61	-0.370	0.711
Genu diameter of the corpus callosum (mm)	11.27±1.25	11.02±1.69	-0.721	0.471
Corpus callosum truncus diameter (mm)	7.20±1.03	7.20±1.13	-0.392	0.695
Corpus callosum splenium diameter (mm)	11.46±1.03	12.02±1.30	-1.346	0.178

Anteroposterior diameter of the corpus callosum (mm)	68.65±3.97	68.49±4.46	-0.371	0.711

FAZEKAS 0: no lesion or only one lesion, FAZEKAS 1: multiple punctiform lesions are present, *=Mann-Whitney U test.

Table 5. The relationship between MIDAS scale scores of patients and corpus callosum measurements

	*	Total area (mm^2)	Genu (mm)	Truncus (mm)	Splenium (mm)	AP (mm)
Inability to go to school and work	r	-0.203	-0.132	-0.168	-0.196	0.130
	p	0.156	0.363	0.244	0.057	0.370
Schoolwork productivity	r	0.065	-0.083	-0.051	-0.007	0.204
	p	0.652	0.566	0.724	0.963	0.156
Inability to do housework	r	0.044	-0.208	-0.151	0.078	0.205
	p	0.762	0.147	0.294	0.590	0.153
Housework productivity	r	-0.127	-0.307	-0.255	0.020	0.104
	p	0.378	0.030	0.074	0.888	0.473
Social family life	r	0.150	-0.409	-0.258	-0.088	-0.014
	p	0.298	0.003	0.070	0.542	0.924
Headache day in 3 months	r	-0.088	-0.330	-0.203	0.054	0.089
	p	0.542	0.019	0.157	0.711	0.540
Headache severity	r	-0.330	-0.226	-0.268	0.125	0.023
	p	0.019	0.115	0.060	0.386	0.876
MIDAS SCORE	r	-0.096	-0.324	-0.237	0.034	0.116
	p	0.509	0.022	0.097	0.813	0.421

AP: Anteroposterior, * = Pearson correlation analysis.

Measured genu values were lower in patients suffering only from phonophobia or visual aura symptoms (p=0.039, p=0.040, respectively). Measured values of the trunk of CC were lower in the moderate and severe disability groups as compared to the MIDAS scale scores (p=0.042). There were no statistically significant differences between other measurements (p >0.05). There were no statistically significant differences between MIDAS and the FAZEKAS scores (p=0.356). Similarly, no correlations were found between the severity of pain, the number of days with pain, disease duration of migraine, and FAZEKAS scores (p >0.05).

DISCUSSION

Migraine, the third most common disease worldwide, is a well-known independent risk factor for subclinical focal deep white matter lesions, even in young and healthy individuals without cardiovascular risk factors. Although deep WMHs are common in migraine patients, their pathophysiology in association with migraines is poorly understood[14]. In our study, we found that the prevalence of WMHs in different hyperintensities was 30% in migraine patients. In the literature, the prevalence of WMH was reported in the range of 31.1%-34.8%; which is consistent with our results[9, 15-17]. However, slightly higher rates of prevalence such as 43.2%, 43%, and 43.1% were reported by Le Pira et al.[18], Seneviratne et al.[19], and Negm et al.[17], respectively. This difference may have occurred because of differences in patient selection and the sample size across studies. The probability of detecting WMHs in brain MRI is up to 2-4 times higher in individuals with migraines compared to controls[3,16, 20-22]. In our study, consistently with the literature, the incidence of WMHs in individuals with migraine was 2.4 times more compared to that in controls.Our study did not reveal a statistically significant relationship between gender and the presence of WMHs.This finding is supported by other studies in the literature[15-17, 22-26].

In our study, no statistically significant relationships were found between migraine subtypes (with and without aura) and the presence of WMHs. (p = 0.445). Some authors, too, previously reported in the literature that there were no associationsbetween migraine subtypes and the presence of WMHs[9, 15, 17, 19, 27]. On the other hand, there are other studies in the literature reporting that the presence of WMHs was found to be higher in migraine patients with aura compared to migraine patients without aura[3, 17, 18, 23].

Zhang et al.[16] and Hamedani et al.[21] have shown that migraine patients without aura have a higher WMH burden compared to migraine patients with aura.It is suggested that recurrent aura attacks create diffuse cortical depression, thus causing ischemic damage by affecting microvascular hemodynamics via fluctuations in cerebral blood flow with hyperperfusion or hypoperfusion[28]. This theory may explain why the incidence of WMHs could be higher in migraine patients with aura compared to migraine patients without aura. Although the results from certain studies support this theory, results from the abovementioned studies and our study findingsdo not support this theory, requiring further studies to be conducted about this subject matter.

In our study, a statistically significant difference was found between patients with and without nausea during a migraine attack (p=0.015). Previously, the presence of WMHs was reported to be higher in patients with nausea [17]. In our study, there was not a statistically significant difference between the presence of WMHs and MIDAS scores (p=0.356). Similarly, no correlations were found between the severity of pain, the number of days with pain, and the presence of WMHs (p>0.05). A significant correlation has beenreportedbetween the severity of migraine attacks and the presence of WMHs[17, 25]. This can be explained by the disruption of the blood-brain barrier due torecurrent severe migraine attacks causing hemodynamic changes, neurogenic inflammation, and cortical spreading depression[19].In the study by Le Pira et al.[18], as in our study, a statistically significant relationship was not found between migraine severity and WMHs. This may have occurred because of the small sample size.

In our study, a statistically significant correlation was not found between the duration of migraine disease and the presence of WMHs (p >0.05). This finding is consistent with results from previous studies[16, 17,19]. However, Traininger et al. [21] concluded that patients with migraines for more than 20 years had a higher incidence of WMHs compared to those with migraines with disease duration shorter than 20 years.Our results can be explained by the fact that most of the patients with migraines in our study had disease duration shorter than ten years. In our study, only three patients had disease duration longer than 15 years. We found out in our study that age was not a risk factor for the presence of WMHs in migraine patients. Consistently, there are studies in the literature reporting no relationships between age and WMHs in migraine patients[3,15,26]. This may mean that frequency and severity of migraine attacks decrease with advancing age. Age is a risk factor for the development of WMHs in migraine patients[16,17,19,29].This may be related to the increased number of years with migraines and the consequently increased likelihood of developing WMHs with advancing age.

In our study, no relationships were found between having a family history of migraines and the presence of WMHs. Seneviratneet al.[19] found a statistically significant relationship between the family history of migraines and the presence of WMHs in their study on migraine patients[19]. That difference may have resulted from collecting information about the presence of migraines in the family by patients' self-reports. Furthermore, in our study, there was not a statistically significant relationship between the lateralization of pain (unilateral or bilateral pain) and the presence of WMHs.

No statistically significant differences were observed in the measured diameters and areas of CC between migraine patients and the control group in our study. Measured genu values were lower in patients with phonophobia and visual aura symptoms compared to migraine patients without aura. According to the MIDAS scale, the measured values of the CC trunk were significantly lower in the moderate and severe disability groups. Akin et al.[11] reportedthat the measured values of the CC splenium and genu were smaller in adolescents with migraines compared to healthy adolescents. Demir et al.[30] reported a statistically significant reduction in the CC volume when migraine patients and control groups were compared. This reduction was greater in patients with migraines with aura. Our study together with other studies in the literature may suggest that the migraine itself, the severity of migraine, and the presence of aura may be associated with atrophy of CC either in its entirety or in its certain regions.Since there are very few studies on the biometry of CC in migraine patients, more studies are needed about this subject matter.

There were some limitations in this study. Firstly, the study sample was small because of the strict selection criteria.Secondly, the use of 3-T MRI could better visualize smaller lesions. The major strengths of our study were the use of a clinical-based design, the standardized migraine diagnosis according to International Headache Society criteria, and the precise MRI protocol read by an experienced

neuroradiologist blinded to the clinical data. The use of the precise MRI protocol and image evaluation by the experienced neuroradiologist blinded to the clinical data minimized the risk of misclassification of lesions.Volumetric measurements of the brain were not evaluated in our study. The normal and standard deviation values of the biometric measurements of CC have not been established, yet. Therefore, the measured values were compared with those obtained from control group participants in our study.

In conclusion, WMHs were found at increased rates in migraine patients, while these rates were even higher in patients with nausea accompanying their migraine attacks. Although the measured CC genu values were found to be lower in patients having phonophobia and visual aura symptoms during their attacks, it was determined that migraines did not affect the measured CC values in general.

YazarKatkıları:Çalışmakonsepti/Tasarımı: FE, ATY; Veritoplama: ATY, MO; Verianaliziveyorumlama: ATY, MO; Yazıtaslağı: ATY, FE, MO; İçeriğineleştirelincelenmesi: FE, MO; Son onayvesorumluluk: MO, ATY, FE; Teknikvemalzemedesteği: ATY, MO; Süpervizyon:FE, ATY;Fon sağlama (mevcutise): yok.
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