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Evaluation of Brain Magnetic Resonance Indices in Migraine and Cluster Headache Patients

Migren ve Küme Baş Ağrısı Hastalarında Beyin Manyetik Rezonans İndekslerinin Değerlendirilmesi

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ABSTRACT

Aim: This study aimed to compare neuroanatomical indices measured by magnetic resonance imaging (MRI), including the bicaudate index (BCI), bifrontal index (BFI), Evans index, fourth ventricle index (FVI), and corpus callosum index (CCI), across patients with migraine with and without aura, cluster headache, and healthy controls, to identify structural differences and elucidate the pathophysiological mechanisms underlying these disorders.
Material and Methods: A total of 144 participants aged 20-50 years were retrospectively analyzed and evenly divided into four groups: migraine with aura, migraine without aura, cluster headache, and healthy controls. Neuroanatomical measurements were obtained from archived MRI scans performed using a 1.5 Tesla device. All indices were calculated using standardized linear formulas and verified by a radiologist blinded to the diagnostic groups.
Results: Significant differences were observed among the groups in BCI, BFI, and Evans index (p=0.047, p=0.010, and p=0.010, respectively). The BCI was significantly lower in the migraine with aura group compared to the cluster headache group (p=0.041). BFI (p=0.010) and Evans index (p=0.006) were notably reduced in the migraine without aura group compared to controls. No significant differences were found in FVI (p=0.172) and CCI (p=0.967).
Conclusion: These findings reveal the presence of structural brain differences in patients with migraine and cluster headache. Changes in BCI, BFI, and Evans index may reflect underlying neuroanatomical mechanisms and could be considered potential biomarkers for diagnosis and individualized treatment strategies. They may also support the development of mechanism-based imaging strategies in the clinical evaluation of headache.
Keywords: Migraine; cluster headache; magnetic resonance imaging; neuroanatomy.

ÖZ

Amaç: Bu çalışmada, auralı ve aurasız migren hastaları, küme baş ağrısı hastaları ve sağlıklı kontroller arasında bikaudat indeks (BKİ), bifrontal indeks (BFİ), Evans indeksi, dördüncü ventrikül indeksi (DVİ) ve korpus kallozum indeksi (KKİ) dahil olmak üzere manyetik rezonans görüntüleme (MRG) ile ölçülen nöroanatomik indeksleri karşılaştırarak yapısal farklılıkları belirlemek ve bu hastalıkların altında yatan patofizyolojik mekanizmaları aydınlatmak amaçlanmıştır.
Gereç ve Yöntemler: Yaşları 20-50 arasında değişen toplam 144 katılımcı geriye dönük olarak analiz edilmiş ve auralı migren, aurasız migren, küme baş ağrısı ve sağlıklı kontroller olmak üzere dört gruba eşit şekilde ayrılmıştır. Nöroanatomik ölçümler, 1.5 Tesla cihazı kullanılarak çekilmiş olan arşivlenmiş MRG görüntülerinden elde edilmiştir. Tüm indeksler standart lineer formüller kullanılarak hesaplanmış ve tanı gruplarına kör bir radyolog tarafından doğrulanmıştır.
Bulgular: Gruplar arasında BKİ, BFİ ve Evans indeks (sırasıyla p=0,047, p=0,010 ve p=0,010) açısından anlamlı farklılıklar gözlendi. BKİ, auralı migren grubunda küme baş ağrısı grubuna kıyasla anlamlı şekilde daha düşüktü (p=0,041). BFİ (p=0,010) ve Evans indeksi (p=0,006) aurasız migren grubunda kontrol grubuna kıyasla belirgin şekilde azalmıştı. DVİ (p=0,172) ve KKİ (p=0,967) açısından anlamlı bir farklılık bulunmadı.
Sonuç: Bu bulgular, migren ve küme baş ağrısı hastalarında yapısal beyin farklılıklarının varlığını ortaya koymaktadır. BKİ, BFİ ve Evans indeksindeki değişiklikler, altta yatan nöroanatomik mekanizmaları yansıtabilir ve tanı ile bireyselleştirilmiş tedavi stratejileri için potansiyel biyobelirteçler olarak değerlendirilebilir. Ayrıca, baş ağrısının klinik değerlendirmesinde mekanizmaya dayalı görüntüleme stratejilerinin geliştirilmesini destekleyebilir.
Anahtar kelimeler: Migren; küme baş ağrısı; manyetik rezonans görüntüleme; nöroanatomi.

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INTRODUCTION

Primary headache disorders such as migraine and cluster headache rank among the most prevalent neurological conditions worldwide and have a profound negative impact on quality of life (1). Diagnosis is generally established through a thorough clinical history (2,3).

Cluster headache is characterized by intense, stabbing pain in the trigeminal distribution, accompanied ipsilaterally by parasympathetic reflex activation and related cranial autonomic symptoms. Attacks typically last between 15 and 180 minutes and are frequently accompanied by restlessness and agitation. Patients experience "cluster periods" during which daily headache attacks persist for durations ranging from seven days to one year, followed by pain-free "remission periods" (1-3).

Migraine, which affects females approximately twice as often as males, is a primary headache disorder manifesting as recurrent, unilateral, throbbing headaches lasting 4 to 72 hours. These attacks are commonly associated with nausea, photophobia, phonophobia, and autonomic dysfunction. Migraine is classified into two main subtypes: migraine without aura (common migraine) and migraine with aura (classic migraine). Migraine with aura is defined by one or more transient, fully reversible neurological symptoms, visual, sensory, speech, or motor, that typically precede the headache phase (1,3,4).

Clinical and experimental investigations into the pathophysiology of migraine and cluster headache have revealed similar structural and functional alterations at the level of the brainstem and hypothalamus (5-8). In particular, advanced magnetic resonance imaging (MRI) techniques have demonstrated widespread abnormalities in pain- and multisensory-processing brain regions in both headache types (2,9,10). Moreover, unique structural and functional changes in cortical and subcortical areas have been proposed to underlie the distinct clinical features of these disorders (11,12). Structural MRI findings in chronic cluster headache patients include volumetric changes in the hypothalamus and adjacent regions, disruptions in white matter tracts, and impaired functional connectivity in pain-processing networks (5). In addition to shared biomarkers such as the hypothalamus and periaqueductal gray matter, structural differences in regions like the left thalamus have emerged as critical factors distinguishing these two headache disorders (2,7,8). These data offer insight into the neurophysiological underpinnings of migraine and cluster headache and support the development of mechanism-based, personalized therapeutic approaches.

The aim of the present study was to measure the bicaudate index (BCI), bifrontal index (BFI), Evans index, fourth ventricle index (FVI), and corpus callosum index (CCI) on brain MRI scans of patients with migraine with aura, migraine without aura, cluster headache, and healthy controls. Through comparative analysis of structural and functional differences in these neuroanatomical indices, we sought to identify distinct biomarkers across headache subtypes. Ultimately, the objective of the present study was to make a significant contribution to the literature on the neurophysiological and neuroanatomical bases of primary headaches and to guide the development of mechanism-based diagnostic and therapeutic strategies through index evaluation.

MATERIAL AND METHODS

This retrospective, single-center study included 108 patients aged 18-50 years who presented to our outpatient clinic between January 2020 and January 2025 and were evaluated according to the International Classification of Headache Disorders (ICHD-III) criteria (1), together with a healthy control group of 36 individuals. Patients diagnosed with migraine with and without aura, or cluster headache, who had undergone brain MRI were identified through the hospital records. The healthy control group consisted of individuals with no history of neurological or psychiatric disorders and no International Classification of Diseases (ICD) diagnostic codes, who presented with benign peripheral symptoms such as benign paroxysmal positional vertigo (BPPV) but had normal brain MRI findings and no evidence of central neurological pathology.

In this study, individuals under 20 and over 50 years of age were excluded to minimize potential age-related changes in brain atrophy and corpus callosum development. Additionally, individuals with neurodegenerative or neuropsychiatric disorders, such as hydrocephalus, stroke, schizophrenia, Alzheimer's disease, or Parkinson's disease, were excluded. A retrospective screening identified only 36 patients with cluster headache who met the inclusion criteria. Due to the limited number of patients in the cluster headache group, the sample size of the other groups was matched accordingly to maintain the validity of statistical comparisons. To ensure comparable sample sizes across groups and reduce potential selection bias, the number of participants in the migraine with aura, migraine without aura, and healthy control groups was standardized to 36 individuals each. Consequently, four distinct groups were established, each consisting of 36 individuals. Due to the known epidemiological characteristics of headache disorders, gender distribution was not equal across groups. Cluster headache is more prevalent in males, while migraine, especially with aura, is more common in females according to existing literature (3). As a result, gender matching was not applied, and the natural gender distribution of each diagnostic group was preserved to reflect real-world clinical patterns.

Patients with migraine were assessed separately. Symptoms accompanying migraine attacks (nausea, vomiting, photophobia, phonophobia) were recorded in detail. For patients diagnosed with aura, visual disturbances, altered shape perception, motor/sensory symptoms, and their localization (right/left) were documented. For all migraine patients, monthly pain frequency and pain localization were determined. In the cluster headache group, the localization (right/left) of autonomic symptoms (tearing, eye redness), monthly attack frequency, and duration were recorded. The healthy control group was rigorously confirmed to have no history of neurological symptoms or headaches. All included patients had previously received standard treatment for their respective headache diagnoses, including acute and prophylactic therapies, as documented in hospital records. However, detailed medication data were not analyzed, as the study focused on structural MRI indices rather than pharmacological outcomes.

MRI was performed using a high-resolution 1.5 Tesla MRI device. BCI, BFI, Evans index, and FVI were measured on axial T2-weighted images, while the CCI was calculated on sagittal T1-weighted images. All measurements were

recorded in millimeters and were verified twice by an experienced radiologist who was blinded to the diagnostic classification of participants to ensure both measurement reliability and minimization of assessment bias.

The formulas used to calculate linear ventricular indices were as follow: BFI = f/F x 100, BCI = c/C x 100, Evans index = f/A x 100 (Figure 1A), FVI = v/V x 100 (Figure 1B), and CCI = AA'/AB + BB'/AB + CC'/AB (Figure 1C). Here, v represents the maximum diameter of the fourth ventricle, V the inner skull diameter at the same level, f the interfrontal horn distance, c the intercaudate nucleus distance, and A the maximum inner skull diameter (13).

This study was conducted pursuant to the principles of the Declaration of Helsinki upon approval of Aksaray University Health Sciences Scientific Research Ethics Committee dated 24.04.2025 and numbered 2025/71.

Statistical Analysis

The distribution pattern of the data was evaluated using the Kolmogorov-Smirnov test. Homogeneity of variances was assessed using Levene's test. One-way analysis of variance was used to assess differences across the four groups for normally distributed data, while the Kruskal-Wallis test was used for data did not follow a normal distribution, followed by post-hoc comparisons. Pairwise comparisons were conducted using the Tukey test following one-way analysis of variance, and Bonferroni-corrected p-values were reported following Kruskal-Wallis test. Normally distributed data were presented as mean and standard deviation, and non-normally distributed data were presented as median, 25^{th}-75^{th} percentile, minimum, and maximum. All statistical analyses were conducted using IBM SPSS Statistics for Windows version 30.0. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 144 participants were included in the present study, with 36 in each group. The migraine with aura group included 21 (58.3%) males and 15 (41.7%) females, with a median age of 32 (range, 20-44) years. The migraine without aura group included 20 (55.6%) males and 16 (44.4%) females, with a median age of 30 (range, 21-44) years. The cluster headache group included 36 patients consisted of 25 (69.4%) males and 11 (30.6%) females, with a median age of 37 (range, 20-49) years. Lastly, the control group comprised 36 healthy individuals, 23 (63.9%) males and 13 (36.1%) females, with a median age of 29 (20-46) years. The groups were matched for age, and no statistically significant differences were observed among the groups in terms of age (p=0.116) or gender (p=0.728).

Cluster Headache Group

In the cluster headache group, the pain was localized to the left side in 21 (58.3%) of the patients and to the right side in 15 patients (41.7%). Autonomic symptoms were present in all patients, with eye redness and lacrimation each reported by 18 (50.0%) patients. The median monthly attack frequency was 13 (range, 4-90). The median pain duration was 1 (range, 0.5-3) hours (Table 1).

Migraine with Aura Group

In the migraine with aura group, the pain was localized to the left side in 22 (61.1%) patients and to the right side in 14 (38.9%) patients. All patients in this group experienced aura, with visual disturbances reported by 26 (72.2%) patients, motor symptoms by 6 (16.7%) patients, and altered

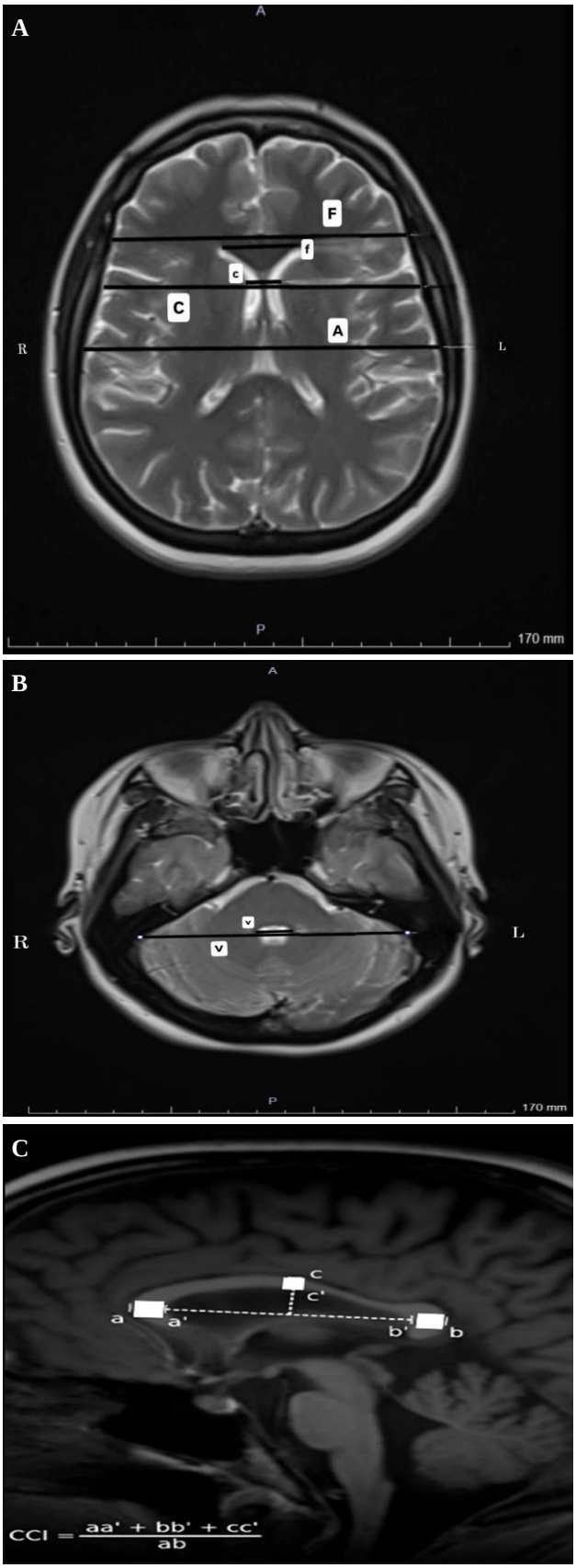


Figure 1. Illustration of the linear measurements used to calculate **A)** bifrontal, bicaudate, and Evans indices on an axial T2-weighted brain magnetic resonance imaging slice, **B)** fourth ventricular index, weight and derived index on an axial T2-weighted brain magnetic resonance imaging slice, and **C)** illustration of the measurements used to calculate corpus callosum index on a sagittal T1-weighted brain magnetic resonance imaging slice

shape perception by 4 (11.1%) patients. The median monthly attack frequency was 3 (range, 1-8), and the median pain duration was 24 (range, 6-72) hours (Table 1).

Migraine without Aura Group

In the migraine without aura group, the pain was localized to the left side in 22 (61.1%) patients and to the right side in 14 (38.9%) patients. A high prevalence of associated symptoms was noted in this group, including photophobia in 22 (62.5%), phonophobia in 21 (60.7%), and nausea and/or vomiting in 19 (53.5%) patients. The median monthly attack frequency was 4 (range, 2-7). The pain duration was 30 (range, 24-72) hours (Table 1).

Comparison Results of the Groups

Statistically significant differences were observed for BCI, BFI, and Evans index (p=0.047, p=0.010, and p=0.010, respectively), while no significant differences were found for FVI and CCI (p=0.172 and p=0.967, respectively) among the groups (Table 2). Post-hoc test results revealed that the mean BCI value was significantly lower in the migraine with aura group compared to the cluster headache group (p=0.041). Additionally, the migraine without aura group showed significantly lower median BFI and Evans index values compared to the control group (p=0.010 and p=0.006, respectively) according to the post-hoc pairwise comparison results.

DISCUSSION

In this study, structural differences in MRI indices among patients with migraine with aura, migraine without aura, cluster headache, and healthy controls were analyzed. The findings offer important insights into the neuroanatomical mechanisms underlying these primary headache disorders. Notably, significant p-values for the BCI, BFI, and Evans index indicate marked structural alterations in migraine patients compared to both control and cluster headache groups. The significantly lower BCI in the migraine with aura group versus the cluster headache group is particularly striking. This may reflect differences in thalamic and ventricular anatomy and provides clues to the complex neuroanatomical basis of migraine pathophysiology. Bertotti et al. (14) reported that altered functional connectivity in the left thalamus is a distinguishing MRI feature between migraine and cluster headache patients. Furthermore, the lower BFI and Evans index in the migraine without aura group relative to controls suggest pathological modifications in frontal brain regions and ventricular structures. This observation aligns with Bertotti et al.'s (14) report of reduced gray matter in the frontal and prefrontal cortices of migraine patients. These findings are consistent with the pathophysiological hypotheses in the literature regarding the role of the hypothalamus, brainstem, and peripheral

Table 1. Clinical characteristics of headache types

	CH (n=36)	MwA (n=36)	MwoA (n=36)
Pain Laterality, n (%)			
Left-sided	21 (58.3)	22 (61.1)	22 (61.1)
Right-sided	15 (41.7)	14 (38.9)	14 (38.9)
Autonomic Manifestations, n (%)	18 (50.0) eye redness 18 (50.0) tearing	-	-
Associated Symptoms, n (%)	36 (100) autonomic symptoms	26 (72.2) visual disturbances 6 (16.7) motor symptoms 4 (11.1) altered shape perception	22 (62.5) photophobia 21 (60.7) phonophobia 19 (53.5) nausea/vomiting
Aura, n (%)	-	36 (100)	-
Monthly Attack Frequency*	13 (10-20) [4-90]	3 (2-4) [1-8]	4 (3-5) [2-7]
Pain Duration (hours)*	1 (1-2) [0.5-3]	24 (24-35) [6-72]	30 (24-48) [24-72]

CH: cluster headache, MwA: migraine with aura, MwoA: migraine without aura, *: descriptive statistics are presented as median (25^{th}-75^{th} percentile) [minimum-maximum]

Table 2. Comparison of neuroanatomical MRI indices among four study groups

	Control	CH (n=36)	MwA (n=36)	MwoA (n=36)	p
Gender, n (%)					
Male	23 (63.9)	25 (69.4)	21 (58.3)	20 (55.6)	0.728
Female	13 (36.1)	11 (30.6)	15 (41.7)	16 (44.4)	
Age (years)*	29 (26-35) [20-46]	37 (26-41) [20-49]	32 (24-37) [20-44]	31 (26-38) [21-44]	0.116
BCI**	8.42±1.65[ab]	9.20±1.90[b]	8.05±2.07[a]	8.26±1.50[ab]	**0.047**
BFI*	27.78 (26.13-29.04) [21.77-33.12][a]	27.33 (25.77-29.26) [14.37-32.25][ab]	27.04 (25.42-28.70) [21.14-30.94][ab]	26.14 (24.84-27.43) [13.96-30.26][b]	**0.010**
Evans Index*	22.27 (21.38-23.49) [17.11-27.12][a]	21.90 (20.65-23.19) [11.81-26.10][ab]	21.62 (20.20-23.20) [17.13-27.13][ab]	21.09 (19.56-21.97) [11.67-24.80][b]	**0.010**
FVI**	14.50±1.77	13.60±2.25	13.89±1.63	13.63±1.98	0.172
CCI**	0.40±0.40	0.40±0.40	0.40±0.38	0.40±0.36	0.967

MRI: magnetic resonance imaging, CH: cluster headache, MwA: migraine with aura, MwoA: migraine without aura, BCI: bicaudate index, BFI: bifrontal index, FVI: fourth ventricular index, CCI: corpus callosum index, descriptive statistics are presented as *: median (25^{th}-75^{th} percentile) [minimum-maximum] or **: mean±standard deviation, [a, b]: groups sharing the same letter do not differ significantly, different superscript letters indicate statistically significant differences based on post-hoc pairwise comparison results: for BCI: control vs CH: p=0.272, control vs MwA: p=0.825, control vs MwoA: p=0.981, CH vs MwA: p=0.041, CH vs MwoA: p=0.130, MwA vs MwoA: p=0.963; for BFI: control vs CH: p=1.000, control vs MwA: p=1.000, control vs MwoA: p=0.010, CH vs MwA: p=1.000, CH vs MwoA: p=0.055, MwA vs MwoA: p=0.405; and for Evan index: control vs CH: p=1.000, control vs MwA: p=1.000, control vs MwoA: p=0.006, CH vs MwA: p=0.666, CH vs MwoA: p=0.140, MwA vs MwoA: p=0.526

structures drawn from advanced MRI studies (2,12). The findings obtained in the present study, consistent with reports in the literature on the neurological mechanisms of migraine, support the notion that structural changes in the frontal lobes play a key role in the pathophysiology of migraine attacks.

BCI is an important biomarker widely used in neurological diseases such as Alzheimer's disease, frontotemporal dementia, Parkinsonism, and epilepsy, assessed via linear measurements of lateral ventricles (15). Messina et al. (2) used MRI biomarkers to differentiate migraine and cluster headache patients, and conducted a detailed analysis of their associations with symptom profiles. Their study emphasized the clinical significance of structural and functional differences in the diagnosis and treatment of patients with migraine and cluster headache. In particular, the left thalamic network was shown to be a distinguishing MRI feature between migraine and cluster headache patients; this observation suggests that the significantly lower BCI in our migraine with aura group compared to the cluster headache group may reflect underlying differences in thalamic and ventricular structures. As reported by Ferraro et al. (5), the central role of the hypothalamus in cluster headache pathophysiology illuminates how dynamic alterations in hypothalamic connections are associated with the characteristic features of the attacks. Similarly, the study by Abagnale et al. (7) on episodic cluster headache highlighted microstructural alterations in the hypothalamus and thalamus, along with differences in functional connectivity within cortical networks. Giorgio et al. (11) further demonstrated that structural and functional modifications in the frontal and lateral occipital cortices support the neurological differentiation between migraine and cluster headache. Taken together, our observation of reduced BCI in the migraine with aura group likely reflects microstructural changes in these key brain regions. This finding suggests that the BCI may serve as a potential biomarker in the diagnosis of primary headache disorders such as migraine and cluster headache, and underscores the need for further research to elucidate how neuroanatomical differences impact clinical outcomes.

Evans index and BFI are critical biomarkers widely used to assess lateral ventricular width on two-dimensional imaging. The Evans index plays a key role in diagnosing conditions such as idiopathic normal pressure hydrocephalus (iNPH) and ventriculomegaly, whereas the BFI is preferred for analyzing neurodegenerative processes like brain atrophy (16,17). The high sensitivity of these measures allows for accurate detection of neuroanatomical changes. In the literature, the frontal cortex is emphasized for its pivotal role in pain perception, processing, and cognitive function; structural modifications in this region have been proposed to contribute significantly to the recurrent, severe pain attacks characteristic of migraine (2,11,12). In the present study, subgroup analysis of migraine patients revealed that significantly lower Evans index and BFI values compared to controls were observed only in the migraine without aura group, whereas no significant differences in these parameters were found in the migraine with aura group. These findings suggest that changes in ventricular system dimensions may be related to cerebrospinal fluid dynamics and intracranial pressure modulation, and that the neuroanatomical profiles of migraine with aura patients more closely resemble those of healthy controls. As reported by Chong et al. (10), weakened connections between the hypothalamus and frontal and temporal cortical areas in migraine patients may predispose to dysfunction within pain-control circuits. Additionally, findings by Coppola et al. (8) and Buture et al. (12) support the hypothesis that integration of the hypothalamus with trigeminovascular and parasympathetic networks triggers central sensitization, forming the neuroanatomical basis of migraine. Bertotti et al. (14) emphasized that disrupted central pain modulation pathways have been demonstrated via neuroimaging in both migraine and cluster headache. Taken together, the particularly low Evans index and BFI values observed in migraine without aura patients indicate that structural alterations among the frontal lobes, hypothalamus, and ventricular system play a decisive role in pain modulation and central sensitization. In patients with episodic migraine without aura, bilateral greater and lesser occipital nerve blocks have been shown to significantly reduce headache frequency, duration, and pain severity, suggesting that neuroanatomical sensitivity in this subgroup may be associated with clinical responsiveness (18). This clinical observation reinforces the hypothesis that structural changes in frontal and ventricular regions, such as those reflected by Evans index and BFI, may not only serve as diagnostic biomarkers but also predict therapeutic outcomes in migraine management.

Limitations of this study include the modest sample size and the restricted age range (20-50 years), which may affect the generalizability of the findings. The use of a 1.5 Tesla MRI scanner may have limited spatial resolution, potentially influencing the accuracy of structural measurements. The classification of migraine patients into only two subtypes (with and without aura) and the absence of attack/remission stratification in the cluster headache group may reduce clinical granularity. Gender distribution was not balanced across groups due to the known epidemiological patterns of headache disorders, cluster headache being more common in males, and migraine with aura in females. Consequently, gender-based subgroup analyses were not feasible, and potential gender-related effects on MRI indices could not be fully assessed. Furthermore, the exclusive focus on structural MRI parameters precluded evaluation of functional or neurophysiological mechanisms. The lack of direct correlation between imaging findings and clinical symptoms limits interpretability. Finally, confounding factors such as stress, lifestyle, genetics, and environmental influences were not controlled, and future studies incorporating these variables are needed to enhance explanatory power.

CONCLUSION

MRI analyses in patients with migraine and cluster headache reveal that neuroanatomical differences shed light on the pathophysiology of these primary headache disorders. The significant differences observed in the BCI, BFI, and Evans index highlight the interplay among the frontal cortex, hypothalamus, and thalamic structures. The low BFI and Evans index values in migraine patients

suggest that changes in ventricular system dimensions may be linked to intracranial pressure modulation and pain mechanisms. The markedly lower BCI in the migraine with aura group compared to the cluster headache group indicates that complex interactions between thalamic and ventricular structures may serve as biomarkers. However, these findings should be interpreted with caution due to factors such as sample size and the technical limitations of the MRI equipment. Large-scale, long-term, and multidisciplinary studies are needed to more comprehensively elucidate the relationship between these neuroanatomical indices and clinical symptoms. In conclusion, the neuroanatomical findings obtained in the present study provide a foundational framework for developing individualized therapeutic approaches and offer new insights in the diagnosis and management of migraine and cluster headache.

Ethics Committee Approval: The study was approved by the Health Sciences Scientific Research Ethics Committee of Aksaray University (24.04.2025, 2025/71).

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