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Association between Migraine Patterns and White Matter Hyperintensities in MRI Brain

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Migraine, White Matter Hyperintensities, MRI, Chronic Migraine, Migraine with Aura, Fazekas Score, Brain Imaging, Headache Disorders

Abstract:

Background: White matter hyperintensities (WMHs) on MRI have been increasingly reported in migraine patients, but their association with specific migraine patterns—such as chronicity, frequency, and aura—remains incompletely understood. This study aimed to evaluate the relationship between migraine subtypes and the prevalence, distribution, and severity of WMHs using MRI brain imaging.

Methods: This cross-sectional observational study included 93 adult migraine patients (aged 18–55 years) diagnosed according to the International Classification of Headache Disorders, 3rd edition (ICHD-3). Participants were categorized into migraine with aura (MA, n=28) and without aura (MO, n=65), and into chronic (n=27) and episodic (n=66) migraine groups. All subjects underwent 1.5T MRI brain scans. WMHs were evaluated using T2-FLAIR sequences and graded using the Fazekas scale. Clinical and demographic data were collected and analyzed. Associations were assessed using chi-square tests, t-tests, Spearman's correlation, and multivariate logistic regression.

Results: WMHs were observed in 38 patients (40.9%). Their prevalence was significantly higher in MA (57.1%) compared to MO (33.8%) ($p=0.031$), and in chronic migraine (66.7%) compared to episodic migraine (30.3%) ($p=0.001$). Deep white matter and bilateral WMHs were more frequently seen in chronic migraine patients. Fazekas scores were significantly higher in MA (1.32 ± 0.89) and chronic migraine groups (1.48 ± 0.62). Spearman's correlation showed significant positive associations between WMH severity and duration of illness ($\rho=0.382$, $p=0.001$), frequency of attacks ($\rho=0.415$, $p<0.001$), and duration of episodes ($\rho=0.336$, $p=0.004$). Multivariate analysis identified chronic migraine (OR: 3.39, $p=0.012$), ≥5 years duration (OR: 2.87, $p=0.021$), and ≥4 attacks/month (OR: 3.14, $p=0.014$) as independent predictors of WMHs.

Conclusion: The presence and burden of WMHs are significantly associated with migraine chronicity, attack frequency, and aura status. These findings suggest that higher migraine burden may contribute to structural brain changes and underscore the importance of early and sustained migraine control to prevent potential long-term neurovascular consequences.

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Introduction

Alopecia refers to “baldness” or loss of hair. The Migraine is a prevalent and disabling neurological disorder affecting an estimated 1 in 7 individuals worldwide, with a global prevalence of approximately 14–15% [1]. It disproportionately affects women, with a female-to-male ratio of roughly 3:1, and is most common in individuals between 25 and 55 years of age, thereby contributing significantly to global disability-adjusted life years (DALYs) [2,3]. In India, the reported prevalence of migraine ranges between 11% and 18%, depending on the region and population studied [4]. Migraine is broadly classified into two major types: migraine without aura (MO), which accounts for nearly 70–75% of cases, and migraine with aura (MA), observed in about 25–30% of migraineurs [5].

The pathophysiology of migraine is complex and multifactorial, involving cortical spreading depression, trigeminovascular system activation, neurogenic inflammation, and vascular dysregulation [6]. Among these mechanisms, cortical spreading depression—particularly in patients with aura—has been implicated in transient hypoperfusion and microvascular dysfunction, raising concerns about structural brain alterations in chronic or severe forms of the disorder [5].

Neuroimaging, especially magnetic resonance imaging (MRI), has uncovered important structural correlates in migraine patients. One of the most consistent findings is the presence of white matter hyperintensities (WMHs), seen as punctate or confluent hyperintense lesions on T2-weighted or FLAIR sequences [5]. Several large-scale studies and meta-analyses have reported a higher prevalence of WMHs in migraineurs compared to healthy controls. For example, the CAMERA study (Cerebral Abnormalities in Migraine, an Epidemiological Risk Analysis) found that 38% of patients with migraine with aura and 19% of those without aura had deep white matter lesions, compared to 12% in controls [7]. Similarly, the GEM study (Genetic Epidemiology of Migraine) observed that the odds of having WMHs were nearly twofold higher in migraineurs, particularly in those with high attack frequency and longer disease duration [8].

The location of WMHs is also relevant; lesions are commonly found in the deep white matter, periventricular areas, and subcortical frontal lobes. Their presence in younger individuals (<50 years) without known vascular risk factors raises questions about whether migraine is independently associated with subclinical cerebral ischemia or microvascular damage. It has been proposed that repetitive neurovascular insults, especially in patients with frequent or prolonged attacks, may lead to cumulative structural changes in the brain [9].

However, the clinical significance of WMHs in migraine remains debated. While some studies have linked WMHs to subtle cognitive deficits, increased risk of stroke (especially in women with migraine with aura), and chronic migraine evolution, others have found no substantial functional consequences [10,11]. Importantly, the migraine with aura as a potential independent risk factor for ischemic stroke, especially when accompanied by smoking or use of estrogen-containing contraceptives [12].

Despite global research interest, there is a notable paucity of Indian studies examining the relationship between migraine subtypes (with vs. without aura), duration of illness, attack frequency, and chronicity with the prevalence, distribution, and burden of WMHs [10,11,12]. This study aimed to evaluate the association between different migraine patterns (aura status, episodic vs chronic migraine, frequency of attacks, and disease duration) and the presence and severity of WMHs on MRI brain in patients presenting to a tertiary care center. By identifying migraine subgroups at higher risk of structural brain changes, this study seeks to contribute to the evolving understanding of migraine as more than a benign episodic headache disorder.

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Material and methods

Study Design and Setting

This was a hospital-based cross-sectional observational study conducted in the Department of Radiodiagnosis in collaboration with Neurology Department at a tertiary care teaching hospitals. The study period spanned 24 months, from January 2023 to December 2024. Prior to data collection, ethical clearance was obtained from the Institutional Ethics Committee. The study aimed to evaluate the association between clinical migraine patterns and the occurrence and distribution of white matter hyperintensities (WMHs) observed on brain MRI.

Study Participants

Patients aged between 18 and 55 years who presented to the Neurology outpatient department with a clinical diagnosis of migraine were considered for inclusion. The diagnosis of migraine was established based on the International Classification of Headache Disorders, 3rd edition (ICHD-3) criteria [13]. Participants were enrolled consecutively after obtaining written informed consent. Migraine was further subtyped into migraine with aura and migraine without aura. Additionally, patients were classified as having episodic migraine (fewer than 15 headache days per month) or chronic migraine (15 or more headache days per month for more than three months). Patients were excluded if they had known vascular or neurological comorbidities including hypertension, diabetes mellitus, stroke, epilepsy, demyelinating diseases, psychiatric disorders, or a history of head trauma or neurosurgery. Individuals taking oral contraceptives or vasoactive medications, those with a history of smoking, or those with any abnormal neurological findings were also excluded to eliminate confounding factors that could influence MRI findings.

Sample Size and Sampling Method

A total of 93 participants who fulfilled the eligibility criteria were recruited using a consecutive non-probability sampling method. The sample size was calculated based on findings from previous studies such as the CAMERA and GEM studies, which reported WMH prevalence in 30–40% of migraineurs [7,8]. Assuming a confidence level of 95%, a margin of error of 10%, and a population proportion estimate of 0.35, the sample size required was approximately 89; however, a sample of 93 was included to account for dropouts and subgroup analyses.

Data Collection and Clinical Assessment

After enrollment, a detailed clinical history was obtained from each participant using a pre-structured proforma. Variables recorded included age, sex, age at onset of migraine, duration of illness, frequency of attacks per month, average duration of headache episodes, presence or absence of aura, family history of migraine, and associated symptoms such as photophobia, phonophobia, nausea, and vomiting. All patients underwent a detailed neurological examination conducted by a trained neurologist to confirm the absence of focal neurological deficits.

MRI Acquisition Protocol

All enrolled patients underwent a non-contrast MRI of the brain using a 1.5 Tesla scanner (Siemens Magnetom Avanto or equivalent). The standard imaging protocol included axial and sagittal T1-weighted sequences, axial T2-weighted sequences, fluid-attenuated inversion recovery (FLAIR), diffusion-weighted imaging (DWI), and susceptibility-weighted imaging (SWI). Particular attention was given to the FLAIR and T2 sequences for the detection of white matter lesions. MRI scans were performed within four weeks of clinical evaluation to ensure contemporaneity of clinical and radiological findings.

Radiological Evaluation of WMHs

All MRI scans were independently evaluated by a radiologist who was blinded to the clinical profile of the participants. WMHs were identified as hyperintense lesions on T2 and FLAIR sequences and were graded using the Fazekas scale. Periventricular WMHs were scored as grade 0 (absent), grade 1 (caps or pencil-thin lining), grade 2 (smooth halo), and grade 3 (irregular periventricular hyperintensities extending into the deep white matter). Deep white matter hyperintensities were graded as grade 0 (absent), grade 1 (punctate foci), grade 2 (beginning confluence), and grade 3 (large confluent areas). Lesions were also anatomically categorized by location (frontal, parietal, temporal, occipital) and laterality (unilateral or bilateral). The burden of WMHs was compared across subgroups of migraine defined by aura status, attack frequency, and duration of illness.

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Statistical Analysis

Data were entered into Microsoft Excel and analyzed using SPSS version 20.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were used to summarize baseline characteristics. Continuous variables such as age and illness duration were expressed as means and standard deviations (SD), and categorical variables such as presence of aura or WMHs were summarized as frequencies and percentages. Between-group comparisons for categorical variables were performed using the Chi-square test or Fisher's exact test, as appropriate. Continuous variables were compared using the independent sample t-test or Mann–Whitney U test depending on normality assumptions. Multivariate binary logistic regression was employed to determine independent predictors of WMH presence, adjusting for potential confounders. A two-tailed p-value of less than 0.05 was considered statistically significant for all tests.

Results

Out of the total 93 participants, 28 (30.1%) had migraine with aura (MA) and 65 (69.9%) had migraine without aura (MO). The mean age of the cohort was 34.5 ± 8.6 years, with no significant age difference between the MA and MO groups (p = 0.284). Females constituted a majority in both groups (77.4% overall; p = 0.924). The mean age at migraine onset was 26.8 ± 7.1 years, slightly earlier in the MA group (25.1 ± 6.8 years) compared to MO (27.6 ± 7.2 years), though the difference was not statistically significant (p = 0.186). Duration of illness, family history of migraine, and attack frequency ≥4 per month were also comparable between groups (p > 0.05). Chronic migraine was more frequent among MA patients (35.7%) than MO (26.2%), while episodic migraine predominated overall (71%), with no significant difference between the groups (p = 0.349) (Table 1).

Table 1: Baseline Demographic and Clinical Characteristics of Study Participants.

Variable	Total (n = 93)	MA (n = 28)	MO (n = 65)	p-value
	Frequency (%)/mean ± SD			
Age (years)	34.5 ± 8.6	35.8 ± 9.1	33.9 ± 8.3	0.284
Gender				
Male	21 (22.6%)	6 (21.4%)	15 (23.1%)	0.924
Female	72 (77.4%)	22 (78.6%)	50 (76.9%)	
Age at Onset (years)	26.8 ± 7.1	25.1 ± 6.8	27.6 ± 7.2	0.186
Duration of Illness (years)	7.5 ± 4.2	8.3 ± 4.7	7.2 ± 3.9	0.211
Family History	37 (39.8%)	14 (50%)	23 (35.4%)	0.167
Frequency ≥4 attacks/month	35 (37.6%)	13 (46.4%)	22 (33.8%)	0.238
Chronic Migraine	27 (29.0%)	10 (35.7%)	17 (26.2%)	0.349
Episodic Migraine	66 (71.0%)	18 (64.3%)	48 (73.8%)	0.349

MA: Migraine with Aura; MO: Migraine without Aura

White matter hyperintensities (WMHs) were observed in 38 out of 93 patients (40.9%), with a significantly higher prevalence in the migraine with aura (MA) group (57.1%) compared to the migraine without aura (MO) group (33.8%) (p = 0.031). Deep white matter hyperintensities were more common in MA (50.0%) than in MO (29.2%) (p = 0.045), while periventricular WMHs did not differ significantly between groups (p = 0.143). The mean Fazekas score, reflecting overall WMH burden, was

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significantly higher in the MA group (1.32 ± 0.89) than in the MO group (0.88 ± 0.74) (p = 0.014). Although higher proportions of bilateral WMH distribution, frontal, parietal, occipital, and temporal lobe WMHs were seen in MA patients, these differences did not reach statistical significance (p > 0.05). Similarly, the distribution across Fazekas grades showed a trend toward higher severity in MA but without statistical significance (Table 2).

Table 2: Distribution of White Matter Hyperintensities (WMHs) on MRI by Migraine Type.

WMH Characteristic	Total (n = 93)	MA (n = 28)	MO (n = 65)	p-value
	Frequency (%)/mean ± SD			
WMHs Present	38 (40.9%)	16 (57.1%)	22 (33.8%)	0.031
Periventricular WMHs	21 (22.6%)	9 (32.1%)	12 (18.5%)	0.143
Deep White Matter WMHs	33 (35.5%)	14 (50.0%)	19 (29.2%)	0.045
Fazekas Grade 1	18 (19.4%)	6 (21.4%)	12 (18.5%)	0.743
Fazekas Grade 2	15 (16.1%)	7 (25.0%)	8 (12.3%)	0.131
Fazekas Grade 3	5 (5.4%)	3 (10.7%)	2 (3.1%)	0.152
Fazekas Score	1.03 ± 0.82	1.32 ± 0.89	0.88 ± 0.74	0.014
Bilateral WMH Distribution	26 (28.0%)	11 (39.3%)	15 (23.1%)	0.117
Frontal WMH	29 (31.2%)	12 (42.9%)	17 (26.2%)	0.125
Parietal Lobe WMH	18 (19.4%)	8 (28.6%)	10 (15.4%)	0.147
Occipital Lobe WMH	11 (11.8%)	5 (17.9%)	6 (9.2%)	0.278
Temporal Lobe WMH	8 (8.6%)	3 (10.7%)	5 (7.7%)	0.673

MA: Migraine with Aura; MO: Migraine without Aura; WMH: White Matter Hyperintensity

Patients with WMHs (n = 38) had a significantly higher proportion of migraine duration ≥5 years (71.1% vs. 43.6%; p = 0.009), frequency of ≥4 attacks per month (55.3% vs. 25.5%; p = 0.003), and chronic migraine (47.4% vs. 16.4%; p = 0.002) compared to those without WMHs (n = 55). Migraine with aura was also more prevalent in the WMH group (42.1%) than in those without WMHs (21.8%) (p = 0.031). Additionally, the mean duration of headache episodes was significantly longer in patients with WMHs (9.8 ± 4.2 hours) compared to those without (6.2 ± 3.3 hours) (p = 0.001) (Table 3).

Table 3: Association between Duration, Frequency of Migraine and WMHs.

Clinical Variable	WMH Present (n = 38)	WMH Absent (n = 55)	p-value
	Frequency (%)/mean ± SD		

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Duration ≥5 years	**27 (71.1%)**	**24 (43.6%)**	**0.009**
Frequency ≥4 attacks/month	**21 (55.3%)**	**14 (25.5%)**	**0.003**
Chronic Migraine	**18 (47.4%)**	**9 (16.4%)**	**0.002**
Migraine with Aura	**16 (42.1%)**	**12 (21.8%)**	**0.031**
Duration of Headache (hours)	**9.8 ± 4.2**	**6.2 ± 3.3**	**0.001**

WMH: White Matter Hyperintensity

White matter hyperintensities (WMHs) were significantly more prevalent among chronic migraine patients (66.7%) compared to those with episodic migraine (30.3%) ($p = 0.001$). The mean Fazekas score, indicating WMH severity, was also significantly higher in the chronic group (1.48 ± 0.62) than in the episodic group (0.87 ± 0.49) ($p < 0.001$). Periventricular WMHs (40.7% vs. 15.2%; $p = 0.009$) and deep white matter WMHs (59.3% vs. 25.8%; $p = 0.003$) were notably more frequent in chronic migraineurs. Chronic migraine patients also showed a significantly greater proportion of bilateral WMH distribution (48.1% vs. 19.7%; $p = 0.005$), and involvement of the frontal (51.9% vs. 22.7%; $p = 0.006$) and parietal lobes (37.0% vs. 12.1%; $p = 0.007$). Differences in occipital and temporal lobe WMHs were not statistically significant (Table 4).

Table 4: Comparison of WMH Characteristics Between Chronic and Episodic Migraine Groups.

WMH Variable	**Chronic (n = 27)**	**Episodic (n = 66)**	**p-value**
	Frequency (%)/mean ± SD		
WMHs Present	**18 (66.7%)**	**20 (30.3%)**	**0.001**
Fazekas Score	**1.48 ± 0.62**	**0.87 ± 0.49**	**<0.001**
Periventricular WMH	**11 (40.7%)**	**10 (15.2%)**	**0.009**
Deep White Matter WMH	**16 (59.3%)**	**17 (25.8%)**	**0.003**
Bilateral WMH Distribution	**13 (48.1%)**	**13 (19.7%)**	**0.005**
Frontal Lobe WMH	**14 (51.9%)**	**15 (22.7%)**	**0.006**
Parietal Lobe WMH	**10 (37.0%)**	**8 (12.1%)**	**0.007**
Occipital Lobe WMH	**5 (18.5%)**	**6 (9.1%)**	**0.191**
Temporal Lobe WMH	**3 (11.1%)**	**5 (7.6%)**	**0.552**

WMH: White Matter Hyperintensity

Spearman's correlation analysis revealed that higher Fazekas scores, indicating greater white matter hyperintensity (WMH) burden, were significantly associated with longer duration of migraine ($\rho = 0.382$, $p = 0.001$), higher frequency of attacks per month ($\rho = 0.415$, $p < 0.001$), and longer duration of each migraine episode ($\rho = 0.336$, $p = 0.004$). A weak but statistically significant positive correlation was also observed with patient age ($\rho = 0.214$, $p = 0.042$) (Table 5).

Table 5:Correlation between Clinical Parameters and Fazekas Score (White Matter Hyperintensity Burden).

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Clinical Parameter	Spearman's rho (ρ)	p-value
Duration of Migraine (years)	**0.382**	**0.001**
Frequency of Attacks/month	**0.415**	**<0.001**
Duration of Each Episode (hrs)	**0.336**	**0.004**
Age of Patient (years)	**0.214**	**0.042**

Multivariate logistic regression identified several independent predictors of white matter hyperintensities (WMHs) in migraine patients. A migraine duration of ≥5 years (adjusted OR: 2.87; 95% CI: 1.17–7.04; p = 0.021), attack frequency ≥4 per month (OR: 3.14; 95% CI: 1.26–7.80; p = 0.014), and chronic migraine status (OR: 3.39; 95% CI: 1.30–8.83; p = 0.012) were significantly associated with increased odds of WMH presence. Migraine with aura showed a trend toward association (OR: 2.41; 95% CI: 0.92–6.29; p = 0.071), but was not statistically significant. Age and female gender were not significant predictors (Table 6).

Table 6:Multivariate Logistic Regression Identifying Predictors of WMHs.

Variable	Adjusted OR*	95% CI	p-value
Duration ≥5 years	**2.87**	**1.17–7.04**	**0.021**
Frequency ≥4 attacks/month	**3.14**	**1.26–7.80**	**0.014**
Chronic Migraine	**3.39**	**1.30–8.83**	**0.012**
Migraine with Aura	**2.41**	**0.92–6.29**	**0.071**
Age (per year increase)	**1.03**	**0.97–1.09**	**0.312**
Female Gender	**1.15**	**0.43–3.10**	**0.779**

*Only variables with p < 0.100 in univariate analysis are typically included in multivariate regression; OR: Odds Ratio; CI: Confidence Interval

Discussion

This cross-sectional study evaluated the association between migraine patterns and white matter hyperintensities (WMHs) on MRI brain in a cohort of 93 adult migraine patients. WMHs were observed in 40.9% of the study population, which is consistent with the prevalence range reported in previous neuroimaging studies of migraineurs. For instance, Zhang et al., reported WMHs in 44% of migraineurs, with a higher burden observed in those with migraine with aura (MA) [14]. Similarly, Negm et al., found WMHs in 43.1% of patients with MA, particularly localized in the deep white matter and posterior circulation territories [15]. In our study, WMHs were significantly more common in MA patients (57.1%) compared to migraine without aura (MO) patients (33.8%) (p = 0.031), supporting the hypothesis that cortical spreading depression (CSD)—a hallmark of aura—may contribute to transient blood–brain barrier dysfunction and white matter injury through repeated hypoperfusion or inflammatory cascades [16,17].

In addition to aura, we found that chronic migraine (defined as ≥15 headache days/month for >3 months) was strongly associated with WMHs. Nearly two-thirds (66.7%) of chronic migraine patients showed WMH presence, significantly higher than the 30.3% prevalence in episodic migraine patients (p = 0.001). Furthermore, the mean Fazekas score was markedly higher in the chronic group (1.48 ± 0.62 vs. 0.87 ± 0.49; p < 0.001), and these patients exhibited more extensive distribution of WMHs, particularly in the

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deep white matter, periventricular areas, and bilaterally. These findings are in concordance with reviews by Planchuelo-Gómez et al., and Chou et al., who reported greater WMH load and altered white matter microstructure in chronic migraineurs compared to episodic migraine and control groups [18,19]. This may reflect the cumulative effects of repetitive headache attacks, neurogenic inflammation, and sustained vascular dysregulation over time.

Spearman's correlation analysis reinforced the role of disease burden in WMH accumulation. Moderate, statistically significant correlations were found between Fazekas score and duration of migraine ($\rho = 0.382$, $p = 0.001$), attack frequency ($\rho = 0.415$, $p < 0.001$), and duration of headache episodes ($\rho = 0.336$, $p = 0.004$). These relationships suggest that the total exposure to migraine activity—rather than any single clinical characteristic—plays a critical role in WMH development. Our results are supported by the Trauninger et al., study, which identified attack frequency and disease duration as key predictors of WMH presence and progression in migraine patients [20].

Multivariate logistic regression further confirmed the independent predictive value of certain clinical variables. A migraine duration ≥5 years was associated with nearly a threefold increased risk of WMHs (adjusted OR: 2.87; 95% CI: 1.17–7.04; $p = 0.021$). Similarly, a frequency of ≥4 attacks per month (OR: 3.14; 95% CI: 1.26–7.80; $p = 0.014$) and chronic migraine status (OR: 3.39; 95% CI: 1.30–8.83; $p = 0.012$) were independently associated with higher odds of WMHs. Although migraine with aura approached significance (OR: 2.41; $p = 0.071$), it did not retain independent predictive value after adjusting for frequency and chronicity. These findings suggest that cumulative migraine burden may outweigh aura as a risk factor when evaluating long-term structural brain changes, a notion also highlighted by Erdélyi-Bótor et al., and Schramm et al., [21,22].

Regionally, WMHs in our cohort were most frequently observed in the frontal and parietal lobes, as well as bilaterally distributed, particularly in chronic migraine patients. These patterns mirror the findings from Su et al., who demonstrated predominant involvement of anterior and deep white matter tracts in migraineurs using T2-FLAIR and DTI imaging [23]. The frontal and parietal predilection could reflect vulnerability of watershed zones or areas with terminal arterial supply, which are more susceptible to ischemic or metabolic insults during repeated migraine episodes.

The mechanisms underlying WMH development in migraine are likely multifactorial. Recurrent episodes of CSD may lead to transient oligemia and hypoxia, particularly in vulnerable deep white matter tracts [24]. Inflammatory mediators such as calcitonin gene-related peptide (CGRP), which are elevated during migraine attacks, may also contribute to endothelial dysfunction and blood–brain barrier leakage [25]. Moreover, migraineurs have been shown to exhibit altered cerebral autoregulation, impaired vasomotor reactivity, and increased oxidative stress—all of which may promote small-vessel injury and WMH formation over time [26].

Nevertheless, the clinical implications of our study are significant. Identification of WMHs in migraine patients—particularly those with chronic symptoms or frequent attacks—may serve as a neuroimaging marker of cumulative disease burden. While the exact clinical consequences of WMHs in migraine remain under investigation, some studies have linked them to subtle cognitive dysfunction, increased stroke risk, and poor migraine prognosis [27,28]. Early recognition and aggressive management of high-burden patients may therefore be crucial to mitigating long-term neurological sequelae.

Limitations

Despite the consistency of our findings with existing literature, certain limitations must be acknowledged. First, this was a cross-sectional study, limiting our ability to establish causality or temporal progression of WMHs. Second, we did not include a healthy control group, which would have provided comparative baseline data for WMH burden. Third, although efforts were made to minimize interobserver variability in MRI interpretation, visual rating scales such as Fazekas may have limitations in sensitivity compared to volumetric or voxel-based analyses.

Conclusion

This study establishes a significant association between migraine patterns—particularly chronic migraine, high attack frequency, and presence of aura—and the occurrence and severity of white matter hyperintensities (WMHs) on MRI. Chronicity and cumulative migraine burden emerged as

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independent predictors of WMHs, highlighting the potential for long-term structural brain changes in affected individuals. These findings underscore the importance of early diagnosis and effective management strategies in high-risk migraine patients to potentially mitigate neurovascular alterations and reduce the risk of long-term neurological consequences.

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